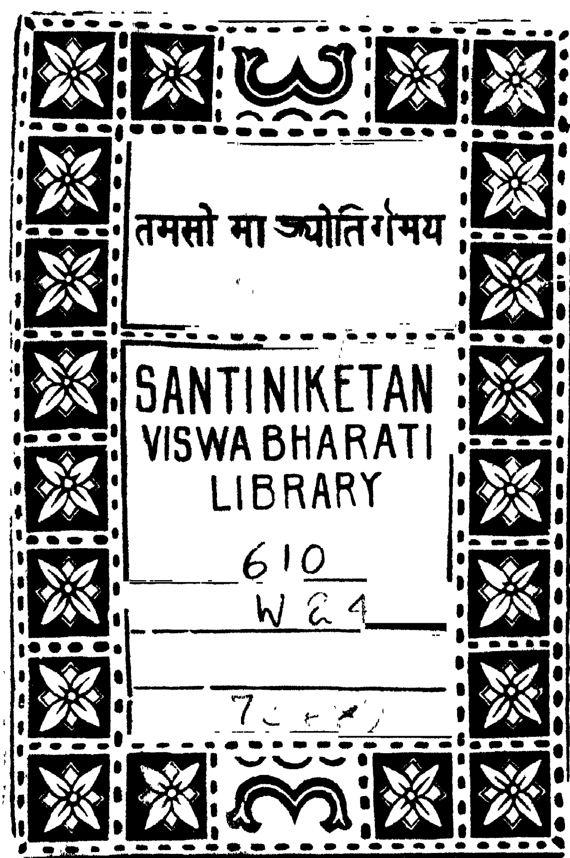

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SAVILL'S SYSTEM OF CLINICAL MEDICINE

DEALING WITH THE

DIAGNOSIS, PROGNOSIS, AND TREATMENT OF DISEASE

FOR

STUDENTS AND PRACTITIONERS

EDITED BY

E. C. WARNER, M.D., F.R.C.P.

THIRTEENTH EDITION



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PREFACE TO THE THIRTEENTH EDITION

MOST text-books of Medicine start by assuming the diagnosis of the various diseases, and then set out the symptoms which should be found. Over forty years ago, Dr. T. D. Savill realised that this is not the way any practising physician goes to work in his consulting-room or at the bedside : what he does is to listen to the patient's history, select the principal (or cardinal) symptoms, and by a process of integration with the other clinical features, arrives at a tentative diagnosis. He then proceeds to consider the probable cause of the condition and any alternative diagnosis, he weighs up the prognosis and then undertakes the treatment of his patient. Dr. Savill therefore constructed a text-book of medicine on these lines—and the success of this Savill System is demonstrated once more by the ever increasing popularity of this book which has now reached its thirteenth edition.

It is no small task to combine the features of a system of this kind with the ever-increasing advances of medical knowledge and medical science : particularly have I attempted to show that there is still an art as well as a science in Medicine : correct diagnosis, the essential preliminary of correct treatment, is not a matter of studying the results of X-ray and other investigations. Without in any way decrying the help that ancillary methods can give in the elucidation of disease, medical practice at its best will always demand a careful assessment of the patient, and of the physical and mental effects of his disease ; and for this a long period of training as a physician, combined with a sympathy for the human problems created by disease, and a knowledge of the pathological effects produced, are all necessary. This art of Medicine, developed by trained medical minds, is beyond the understanding of unskilled and untrained political planners.

Chapter I, which describes the rules for clinical investigation as written by Dr. T. D. Savill, has been little altered over the years : no great alteration has been made in this new edition, but a few salutary sentences have been added, such as the one which exhorts the new student still " to be complete in your examination of your patient ". In the chapters which follow, a careful revision has been undertaken, and no page has escaped alteration in some form or other. Among the major alterations will be found an almost complete rewriting of Chapter XV dealing with the pyrexial disorders and infective diseases: in this chapter, Dr. F. Murgatroyd has extensively revised the tropical section, and has added a new table on the typhus group of fevers : and I have rewritten much of the remainder of this chapter. Chapter XVI, which deals with the anæmias and the wasting diseases, has also received special attention. Dr. Britton has supplied the expert knowledge so that together we have rewritten the methods of examining the blood, and added tables which give the range of the normal values which may be expected in the red cells, white cells, etc.,

and in the sternal marrow. Dr. V. E. Lloyd has given generously of his time and knowledge to help me bring the section dealing with syphilis up to date: and Dr. Brewer has entirely rewritten and expanded the section on blood transfusion and the rhesus factor: this seemed vitally important in view of the expanding knowledge and increasing importance of transfusion in every branch of medical practice. It is impossible to catalogue all the other subjects which have been largely or entirely rewritten: illustrative examples include acute appendicitis, chronic gastric and duodenal ulceration, cancer of the stomach, gastric dilatation, acute infective hepatitis, rheumatic endocarditis, circus movement, periarteritis nodosa, hay fever, surgical shock, ringworm of the hair, tuberculous meningitis, cerebral tumour, electro-encephalography, and the vitamins. The variety of diseases which have been added is too numerous to be completely catalogued and we must content ourselves with some examples: pulmonary acariasis, sarcoidosis, bronchial adenoma, fœtal adenoma of the thyroid, the Waterhouse-Friderichsen syndrome, pheochromocytoma of the supra-renals, abacterial pyuria, artificial insemination, aspirin poisoning, cortical thrombo-phlebitis, choroidal tubercle, sterilisation of syringes, liver biopsy and aspiration, insect repellents, intramedullary blood transfusion, Reiter's disease. A number of new diagrams, tables, X-ray reproductions and graphic records have made their appearance. I make no apology for introducing new drugs which have established themselves in medical treatment, nor for giving considerable prominence to the antibiotics and the sulphonamides. Mention will be found for the use of D.D.T., stilbamidine, folic acid, vitamin B_{12}, intravenous iron, nitrogen mustard, the arsenoxides, tridione, paludrine, radio-active phosphorus, potassium thiocyanate, pyridoxine, calciferol, and thiouracil: and the uses of penicillin, streptomycin, aureomycin and chloromycetin have been described, and are amplified by special tables (Tables XXVIII, XXIX and XXX). To make it easier to turn up any particular table, a list is included in the front of the book.

I have indeed been fortunate in having the help of the team of experts who helped in previous editions. In a book written in a particular style, it is not easy for new contributors to develop the Savill System until after a good deal of practice. Dr. Murgatroyd has taken the place of Professor Hamilton Fairley, and has brought up to date the tropical diseases, in a way which does him great credit. We have missed the help of Dr. J. D. Rolleston, who during his lifetime had contributed to the last five editions. My very grateful thanks are due to the patience and help of Dr. Geoffrey Bourne who revised the Cardiological section, Dr. Geoffrey Evans, who is such a recognised authority on Arterial disease, and to Dr. Maurice Davidson who is such a well-known exponent of the diseases of the Lungs. Also to Mr. L. R. Broster who helped with the Abdominal diseases, Dr. S. W. Patterson who was responsible for the diseases of the Stomach and Intestines, and to Mr. F. S. Warner who has revised the diseases of the Mouth.

Others to whom I am indebted are: Mr. Norman Fleming, for the diseases of the Eye; Dr. Ethel Browning, for rewriting the Vitamins; Dame Louise McIlroy who for so long has been responsible for the Women's diseases; Mr. Arthur Gray who has rewritten the section on Sterility; Mr. W. A. Mill for diseases of the Nose, Throat, Ear, Larynx and Œsophagus; Dr. Thomas Tennent for revision of the Psychological disorders and who has paid particular attention to the new legal requirements of mental certification; and to Dr. Redvers Ironside whose extensive section on diseases of the Central Nervous System has long been one of the most popular sections of the book. It is with especial pleasure that I mention Dr. Agnes Savill, who again revised the diseases of the Skin, and who for so many years edited the book entirely on her own, until I joined her in the eighth and subsequent editions.

To these main contributors, I would like to add my thanks to Dr. T. Fane Tierney for supplying new radiographs; and to Dr. W. E. Clarke who has helped with various suggestions during the arduous tasks of proof-reading and preparation of the index. This task has been shouldered also by Mr. B. J. Newman and by my secretary Miss Valerie Myers whose help in preparation for the press has been invaluable. No textbook is ever perfect, and the earlier pages already seem to show occasional imperfections: but such as may be found are my entire responsibility, for no one could have had more generous help than I: particularly must I take responsibility for any defects in Chapters I, II, XIII, XV and XVII which were largely or entirely my particular responsibility. Lastly, I hope the index will be found helpful, for especial care has been taken with this—in a book of this description, where cross-references are so numerous, the index does assume a very especial importance.

E. C. WARNER.

LONDON, W.1.

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INTRODUCTION

Those who ponder on general principles and methods will have observed that a considerable change has gradually taken place during the last half-century in the methods of studying the science and art of medicine. Formerly, men were content to observe the symptoms or effects of disease at the bedside and in the dead-house, and to speculate on the etiological connection of these two series of phenomena. Wherever the association of such phenomena during life and after death was sufficiently constant, they were spoken of collectively as a "disease"; when a group of symptoms without anatomical lesion constantly recurred, it received a name and place among the list of "disorders." Then each disease or disorder was taken as a separate entity, its anatomy, symptoms, diagnosis, and treatment were described, and its various possible etiological factors discussed; and the result was known as "Descriptive" or "Systematic Medicine." The guiding principle of this descriptive process was the tracing from an assumed *cause* to a known *effect*.

EVOLUTION.

In later times great advances were achieved, almost synchronously, in two very different directions. On the one hand great improvements were made in the methods of observing and investigating the symptoms or effects of disease during life, and thus Clinical Medicine came into separate existence. This stage was marked by the appearance in this country of two very successful works—one by Dr. Samuel Fenwick, of London, on "Medical Diagnosis," first published in 1869, dealing with the symptoms and diagnosis of disease; another by Dr. James Finlayson, of Glasgow, entitled "A Clinical Manual," first published in 1878, dealing with the methods of observing and investigating the symptoms of disease. On the other hand, with the extremely rapid growth of chemical, biological, and bacteriological sciences, and the elaboration of experimental methods in the investigation of disease processes, a new school of pathology arose, whose methods were based upon experiment, and whose leading principle was the artificial production of a definite *cause* and the observing of its *effects*. The extraordinary advances made by these means, and the new light thus shed upon the science of medicine during the last twenty years, form at once the wonder and delight of the civilised world.

As the result of the movement to which I have referred, and the growth in the two directions named, treatises on Systematic Medicine, which attempt to deal at all fully with both the clinical and the pathological aspects of disease, have come to assume very considerable dimensions. In many of them there seems to be a tendency to become more and more pathological in their arrangement, and to treat diseases as separate entities, so that students of clinical medicine and busy practitioners, whose daily work consists of an endeavour to trace from *effect* to *cause*, have been heard to complain that they do not always find in them the clinical aid they seek.

Immediately after embarking on medical practice I realised, as probably many others have done, the importance for diagnostic purposes of reviewing the various diseases or pathological conditions which might give rise to a patient's leading symptom or symptoms, and being unable to find precisely the information desired in any of the current text-books, I proceeded to keep a brief record of all the cases I met with arranged under the heading of their leading symptom. This book is based upon those records, which extend over many years, combined with the valuable knowledge imparted to me at the bedside by my teachers—more especially Dr. Charles Murchison, Dr. J. S. Bristowe, Professor J. M. Charcot, and Sir William Broadbent. Hospital cliniques, at first of a general and later of a more special kind, have always been at my command; but it was at the Paddington Workhouse and Infirmary that the idea of this work was conceived, its foundations laid, and the chief part of its "skeleton" constructed. It would be hard to conceive circumstances better suited to the task, for our great poor-law infirmaries contain, as all the world now knows, a vast and almost unexplored field of every possible variety of disease, which can be studied from day to day from the beginning *to the end* of its course.

ORIGIN.

As regards the plan and arrangement of this work, the subject will be approached from the standpoint of symptomatology. The principle throughout will consist of tracing from effect (symptoms) to cause (the morbid cause in operation). The order of sequence will be that which should be adopted in the examination of a patient. Thus, the first chapter will give a general scheme for the examination of a case, and will deal with certain general principles underlying methods of observation, diagnosis, prognosis, and treatment. In the second chapter the physiognomy of disease will be discussed. The succeeding chapters will deal seriatim with the symptoms and signs referable to the several organs or anatomical regions of the body, and the disease which may cause those symptoms.

PLAN.

Each chapter will be divided into three unequal parts. Part A. will treat of the *symptoms* which may indicate disease of the organ or region under discussion, the fallacies incidental to their detection, and a brief differential account of the various causes which may give rise to those symptoms. Part B. will treat of the *physical signs* of disease in that region, and the various methods used to elicit them. Part C., which constitutes the major portion of each chapter, will be prefaced with a *clinical classification* of the various maladies affecting that region, and a summary of the routine procedure to be adopted; and this will be followed by a series of sections dealing with the several *diseases*, arranged according to their clinical relationships. For example, in Chapter III., on The Heart—Part A. describes and differentiates the various causes of breathlessness, dropsy, palpitation, precordial pain, and the other symptoms which may be indicative of heart disease; Part B. describes percussion, auscultation,

and the other methods of examining the heart; and Part C. deals seriatim with the various cardiac disorders, classified and arranged on a clinical basis.

Apart from the general plan and arrangement, there are two features special to this work. The first part of each chapter, dealing with symptoms and their causes, forms a feature on which great labour has been expended. To make each list of causes complete without redundance, and to check the various data again and again in the light of experience, has involved an expenditure of time quite out of proportion to the space occupied. These lists will, I trust, be as useful to others as they have been to me in obtaining a clue to diagnosis.

SPECIAL FEATURES.

Another feature consists of the italicised paragraphs in Part C. standing at the head of each section, which deal with a separate malady. These emphasise the salient features by which a disease may be recognised and differentiated from others belonging to the same clinical group. They are, in fact, brief clinical definitions, and form, metaphorically speaking, "sign-posts" or guides in the process of diagnosis. If, after carefully studying the lists of symptoms and their causes in Part A., and examining his patient (Part B.), the reader turns to these italicised paragraphs in Part C., the work will, it is hoped, serve as a "clinical index of diseases"; for by following the plan laid down he will shortly find himself reading a description of the diagnosis, prognosis, and treatment of the malady from which his patient is probably suffering; while adjacent to this are the disorders which clinically, and very often pathologically, resemble it, and for which in practice it is apt to be mistaken.

Such an arrangement as that proposed must inevitably lead to some repetition, but this difficulty has been obviated to a certain extent by cross-references. I would also ask the reader to remember that nothing fixes things so well in our minds, or aids us so much in tracing those analogies to which I shall shortly refer, as constantly looking at the same facts from a different point of view.

An attempt has been made to present the various diseases in some kind of perspective by placing them as far as possible in order of importance and using different sized types. The relative importance of different subjects in medicine is largely a matter of opinion, and I cannot expect to escape criticism in this respect.

It is a standing accusation against medical writers that they are careless in respect to literary style, and I fear that I shall not be found an exception. I have striven to be intelligible rather than academic; and in general I feel that I must plead guilty to having endeavoured to follow the Duchess's advice to Alice in Wonderland, to "take care of the sense and the sounds will take care of themselves." When so large an area has to be covered, a certain amount of abbreviation is indispensable, and in order to condense my material, it has been my practice to adopt a numerical

method of description. Some may take exception to this, though the student will find it to his advantage in the acquisition of knowledge.

I may perhaps be pardoned for adverting to certain advantages which appear to me to be associated with the method that I have adopted of approaching clinical medicine. And first let me remark that this method of diagnosis is not what has been called a "process of exclusion." It is a positive rather than a negative process, for by carefully considering the various causal diseases which may be in operation and balancing the evidence for and against each, the physician is guided, not to the least improbable, but to the most probable diagnosis.

ADVANTAGES.

The advantages of passing in rapid review all the possible diseases which may give rise to a patient's leading symptom, are very obvious to those actively engaged in clinical work. It was Dr. Charles Murchison's method in his bedside teaching; and another equally great clinician, Dr. Matthews Duncan, has aptly remarked: "If you do not know of a thing, you are quite sure not to suspect it; and in all cases of difficult diagnosis, if you do not suspect the disease, you are almost certain not to find it." But I am not aware that any work has yet been published which adopts precisely this plan of approaching clinical medicine.

This plan gives, I venture to think, a truer view of nature's facts than one which deals with diseases as so many separate entities. We see a case in all its clinical and practical bearings. We not only learn that the diagnosis of a patient's malady can at best be only a question of the greatest probability, but with almost mathematical precision we can also assess the probability or improbability of each of the other possible causes in operation. We learn further that all diagnoses can only be provisional, and that the degree of probability of each possible cause changes from day to day, like the coloured pattern of the kaleidoscope, as the course of the malady unfolds itself before us.

It is, moreover, in clinical work carried out on these lines—where diseases presenting analogous clinical phenomena are constantly being associated together from different points of view—that the rôle of the imagination, both in the investigation and in the treatment of disease, finds a legitimate place. The recognition of a clinical likeness between diseases has often led to the erection of a "working hypothesis" which by subsequent research has been found to be correct. Many of our greatest discoveries have been initiated in this way. It was, for instance, a process of this kind which led to the discovery that a large number of, perhaps all, pyrexial disorders are of microbic origin. There are still a number, notably measles, small-pox, and scarlatina, in which such a working hypothesis, based on clinical resemblances, forms at present the full extent of our knowledge; but so precise are these foundations that the microbic nature of these diseases is never doubted. Hypotheses framed in this way should always be tested and confirmed in the laboratory and dead-house, whenever the morbid conditions can be produced experimentally, or when they are

attended by fatal results. But unfortunately there are still a great many diseases, such, for instance, as the two great groups of clinical conditions we call hysteria and neurasthenia (conditions which form a not inconsiderable portion of the practitioner's daily work), which cannot, excepting in the most isolated instances, be observed in the dead-house, and which have not yet been produced in animals. In these cases the method of analogy or comparison to which I have just referred is not only a valuable means of investigation, it forms almost the only means we have.

It is given only to few to devote the necessary time to laboratory research; but all can study their cases at the bedside in the way indicated, and many a valuable and often unrecorded idea as to treatment will occur to the practitioner who thinks out and traces analogies between diseases.

There is yet another advantage which has always appeared to me to accrue, especially to the young observer, by this process of balancing evidence and comparing diseases. It not only impresses important facts upon his memory, but it constitutes one of the best possible means of training him to habits of accurate and complete observation, and of systematic and productive thought. The scope of his horizon is widened, his faculty of systematising his knowledge becomes by practice wonderfully increased, and his reasoning powers strengthened and corrected. He finds intuitively that without accuracy in respect to the most minute details he may be led astray in the more important ones, that without system in the arrangement of his facts he will never be able to attach the proper significance and importance to each; and finally, that without judgment in attaching due weight to each item of evidence, his conclusions may be erroneous although his premises and facts are correct.

RESPONSIBILITIES.

I have now described the scheme of this work, its purposes and scope—in a word, the ideal which I hoped to compass; and I believe no one could approach a task of this kind without realising the responsibilities and difficulties involved in its execution. Amidst the bewildering records of medicine there are many excellent treatises both on systematic medicine, the medicine taught in the schools, and on one or other of the several departments of clinical medicine. These deal with their respective subjects in a manner which I cannot hope to rival, and they have been to me an abundant source of instruction, but they have afforded me no exact precedent or guide along the path I wished to travel. The contemplation of the wide range of knowledge and experience required, of the immense advances which have recently been made both in the theory and practice of medicine, of the supreme importance of the subjects here dealt with, involving as they do questions of life and death, has filled my mind with a painful sense of the obligation imposed upon me to sift my facts, and to cull my knowledge, truly, from all sources, but, before all, to obtain my material as far as possible by careful observation and patient thought from the book of nature which lay open before

me from day to day at the bedside in infirmary, hospital, and private practice.

In these circumstances I have gladly availed myself of the help and advice of many friends, and there are some to whom special acknowledgment is due. In certain parts of the chapter on fevers, notably on scarlet fever, measles, diphtheria, and enteric fever, I have had much valuable advice and suggestion in the revision of the proofs from my old friend Dr. Foord Caiger. Similarly in the subject of aneurysm and in parts of the subject of pulmonary disease I am indebted to Dr. Robert Maguire, in parts of the chapter on diseases of the throat and nose to Dr. St. Clair Thompson and Dr. Scanes Spicer, in parts of the section dealing with serum-therapeutics to Dr. George Dean, in parts of the chapter on diseases of the heart to Dr. Alexander Morison, and in parts of the chapter on the urine to Dr. C. O. Hawthorne. The illustrations, with few exceptions, are taken from actual cases, and have been drawn specially for this book under my own supervision; my grateful thanks are due to the artist, Mrs. Stanley Berkeley, a Royal Academy medallist, who has lent her talent to enrich these pages with drawings which are not only accurate but, as far as scientific drawings can be, artistic. Finally, it is difficult for me to express in measured terms my indebtedness to my wife, who has assisted me in the elaboration of this work during the greater part of four years. Her skill and knowledge have largely helped to give it such completeness as it may possess; her patient industry has afforded me not only assistance, but example; and her companionship and encouragement have made many rough places smooth, and have often transformed what at times seemed to be a laborious and interminable task into a pastime.

T. D. SAVILL.

A SYSTEM OF CLINICAL MEDICINE

CHAPTER I

CLINICAL METHODS

Preliminary Definitions—Case-Taking—Methods of Diagnosis, Prognosis and Treatment—Rules for Clinical Investigation.

§ 1. **Definitions.**—Disease is a departure from health, and is manifested in an individual during life by symptoms. These are of two kinds—"*subjective symptoms,*" which are recognisable only by the patient, and present no external indication, such as pain, itching, or a feeling of chilliness; and "*objective symptoms,*" [1] which can be detected by the observer—*e.g.*, abdominal enlargement or dulness on percussion. The word "symptom" is used in two senses. Sometimes it is used in a general sense to indicate all the subjective and objective evidence of a disease; but more usually it is employed in a narrower sense, as synonymous with subjective symptoms. Objective symptoms are usually spoken of as *signs*; and those objective symptoms which are made out by physical examination are known as *physical signs.*

Just as the value and significance of physical signs depend on the skill and experience of the physician who observes them, so the significance of subjective symptoms has to be weighed and considered in relation to the character and constitution of the patient who complains of them. Thus a certain symptom may appear trivial and unimportant to a patient of strong character not addicted to introspection, although serious disease may be present; whereas in women with a susceptible nervous system every subjective symptom, however slight, may cause great anxiety, exaggeration, and even real suffering. Submammary pain, for instance, in the first might indicate aneurysm; in the second, hysteria.

General (or constitutional) symptoms are those which relate to the whole body, such as debility or pyrexia.

A *latent disease* is one which is unattended by any very obvious symptoms. Thus, we speak of latent pulmonary tuberculosis when a patient suffering from tuberculosis of the lung has none of the more usual and constant symptoms of that disorder. Physical signs are not necessarily absent in latent disease, but they are often difficult to detect. Some writers speak of a malady as being latent when the pain, which is usually a prominent feature of the disease, is absent. Thus, pericarditis is ordinarily attended by a good deal of pain, but pain is absent in the latent

[1] These words "subjective" and "objective" are borrowed from philosophy. Subjective reality is reality which exists in the mind only, whereas objective reality is that which can be demonstrated by means of tangible, visible, or outward signs.

form of pericarditis which frequently complicates rheumatic fever, and in the latent peritonitis which complicates typhoid fever.

A *paroxysmal disorder* is one which comes on in the form of attacks separated by intervals of comparative health. Each attack or paroxysm consists of a stage of invasion (usually more or less sudden), leading to an acme, and followed by a gradual decline in the severity of the symptoms. As instances of paroxysmal disorders may be mentioned Paroxysmal Tachycardia, Angina Pectoris, Epilepsy, Nervous Faints and Flush Storms, and Paroxysmal Hæmoglobinuria.

§ 2. **Case-Taking.**—In clinical investigation, or case-taking, our object is, *first*, to elicit all the data of the case; and, *secondly*, by reasoning based on those data to arrive at its Diagnosis, Prognosis, and Treatment. It will be found in actual practice that everything turns on the diagnosis; that is our first and principal object; the prognosis and treatment follow from this.

The investigation of a case consists of two parts: (A) The Interrogation of the Patient, and (B) the Physical Examination. Students should always accustom themselves to learn all that is possible by interrogation before proceeding to the physical examination.

A. By **Interrogation of the Patient** we learn—

(*a*) What is his *chief* or cardinal symptom;
(*b*) The facts concerning the *present illness*;
(*c*) The patient's *previous history*;
(*d*) The patient's *personal history*; and
(*e*) His *family history*.

Throughout the interrogation of the patient it is well to follow THREE GENERAL RULES:

(1) *Avoid putting what barristers call "leading questions"*—*i.e.*, questions which suggest their own answer—*e.g.*, "Have you had a pain in the back?" suggests an obvious answer to the patient. It might be put thus: "Have you had any pain, and if so, where?" The patient should be encouraged to tell his own story, without interruption. Moreover, the very words he uses should be recorded between inverted commas, and on no account should his words be translated into scientific terms. Some say that leading questions are permissible when the patient is very ignorant and stupid, but these are the very cases in which leading questions should be specially avoided. The only legitimate way of putting a leading question is in an alternative form—*e.g.*, "Have you suffered from diarrhœa or constipation?" Time, patience, and tact are necessary to elicit the true facts of the case, without irrelevant detail. Our object is to learn what the patient *feels* and knows, not what he *thinks* of his disease; and our patience is often sorely tried by a long story of his own or his previous doctors' views on his case. Our record should be comprehensive, including all important data, negative as well as positive, yet concise—*i.e.*, excluding irrelevant facts. Only experience and a knowledge of medicine can teach us what is or is not relevant. The beginner should strive after completeness rather than conciseness.

(2) A *chronological order* should always be adopted, both in eliciting and in recording the facts. Nothing is more wearisome than to wade through a mass of verbiage which mixes up dates. Dates should be recorded always in the same terms. It is very common, for instance, to read in students' reports that "breathlessness began in the year 1922," "palpitation started when the patient was aged forty," "dropsy came on two years ago."

(3) Always adopt a kindly and *sympathetic manner*. Not only is it our bounden duty to be considerate and patient with those who suffer, but by entering into the spirit of the patient's sufferings we can often get at more important facts, and a truer narration of them, than can one whose harsh or abrupt manner causes the patient to shrink up like an oyster into its shell. Put your questions in as simple and non-technical a form as possible, and be sure that the patient attaches the same meaning to the words as you do. Much will depend on the tact of the physician, and two very good rules may here be added—viz., Never put questions bearing on venereal disease before the husband or wife of the patient; never inquire concerning a family history of tuberculosis or cancer before a patient whose illness is likely to be of that nature.

(*a*) THE CHIEF OR CARDINAL SYMPTOM.—The first question to ask a patient should always be the same: "What do you complain of?" Special attention should be paid to the symptom for which the patient seeks advice or is admitted to hospital, because it is this symptom which guides most of our subsequent inquiries. It should always, as far as possible, be recorded in the patient's own words. *This book is based upon the patient's cardinal symptom;* and in the following chapters I shall, after each cardinal symptom, allude to the principal conditions for which it may be mistaken. The best way to avoid error is to verify your observations by repeating your examination.

(*b*) THE HISTORY OF THE PRESENT ILLNESS must be taken and recorded with care. It cannot too strongly be emphasised that in many diseases a full and accurate history of the illness may be the only method of arriving at a diagnosis, for physical signs may be absent or in abeyance (*e.g.*, in angina pectoris). Taking an average, it is fair to compute that of the information on which a diagnosis is ultimately founded, at least 50 per cent. comes from an accurate history, and rather less than 50 per cent. from the physical examination and subsequent special investigations. First ascertain when the illness started, by a question such as "When did you first notice or complain of this trouble?" and this being answered: "Did you ever have this symptom before?" Then the history should reveal (i.) the mode of onset, whether sudden or gradual, (ii.) what the patient was doing at the time, and whether he attributed the onset to any cause. In certain cases it is necessary to inquire into (iii.) the duration of the symptom, (iv.) whether it ended suddenly or gradually, (v.) its severity, (vi.) whether it has occurred since, and if so, how many times, and is it getting more or less severe; (vii.) what intervals of freedom have occurred, when the patient has been entirely free of the symptom;

(viii.) have other symptoms occurred in association with this chief symptom and if so, what they are; (ix.) what does the patient do during the time of the symptom to relieve it; (x.) has the patient found any measures of avail to ward off attacks, *e.g.*, drugs, diet, etc. In many cases, *e.g.*, in juvenile and unconscious persons, the history has to be elicited from near relatives or friends. It is useful also to know whether the patient has recently been, or is now, under medical care, not only because the symptoms may have been modified by treatment, but also because one of the most important ethical principles of the medical profession may be involved.[1] In all these inquiries the three general rules given above apply (p. 2).

(*c*) THE PREVIOUS HISTORY of the patient bears largely on the etiology, or *causation*, of his illness, and deals with any *illnesses* the patient may have had. Note in chronological order all ailments from which the patient has suffered prior to the present one, with the dates of their occurrence and their duration—*e.g.*, contagious diseases of childhood; and especially such ailments as venereal disease, rheumatism, and gout. If the attacks have been at all obscure, it is desirable to add a few of the leading symptoms to prove the nature of the alleged attacks, and in such instances inverted commas should be freely used. For instance "rheumatism" is a vague term which may mean any disease attended by pains in the limbs, such as alcoholism, syphilis, tabes dorsalis, or neurasthenia. The subject of syphilis should always be approached with delicacy in the case of women. Indirect information may often be gained by inquiring for prolonged sore throat, followed by loss of hair, by skin rashes, etc. In married women, a *series* of still-births, or children born with eruptions or snuffles, may have the same significance.

(*d*) THE PERSONAL HISTORY must be inquired into: such as (i.) present and previous occupations; (ii.) previous residence abroad; (iii.) the home conditions; (iv.) habits as to alcohol and tobacco and in what form alcohol is taken (*e.g.*, wine, beer or spirits) and whether between or with meals, because more harm is done by alcohol before meals (especially cocktails) than many times the same quantity taken with meals; (v.) the appetite; (vi.) the state of the digestion and the bowels; (vii.) the weight, and whether this is constant, being gained or lost; (viii.) the general state of the nervous system, *e.g.*, depression, excitability, nervousness; (ix.) the orientation of the patient to his (or her) work and to home life, and whether there are any special anxieties attached to these; (x.) the amount and quality of sleep. (xi.) In women, the previous state of the catamenia, and the number of pregnancies, miscarriages, or still-births, should be noted.

[1] By-law CLXIV of the Royal College of Physicians of London runs as follows: "No Fellow, Member, or Licentiate of the College shall officiously, or under colour of a benevolent purpose, offer medical aid to, or prescribe for, any patient whom he knows to be under the care of another legally qualified Medical Practitioner." This is perhaps the most important guiding principle in the ethics and etiquette of the medical profession. On the other hand, this law gives us no proprietary right in a patient because we have once prescribed for him or his family. He ceases to be our patient directly he ceases our treatment for that particular ailment.

(e) The Family History may, like the previous history, have a causal relationship to the patient's malady. The age and state of health if living, age and cause of death if dead, of near relations, should always be noted—*i.e.*, father and mother, brothers and sisters, sons and daughters, also of husband or wife. Inquiry should also be made as to whether any members of the family (parents, grandparents, brothers, sisters, uncles, aunts, or cousins) have suffered from tuberculosis, cancer, acute rheumatism, gout, nerve diseases, insanity, asthma, heart disease, apoplexy, and especially those diseases to which the patient himself seems liable.

B. The **Physical Examination** (*i.e.*, the State on Admission, or the Present Condition) may with advantage be prefaced by a few general remarks on how and what to observe.

(1) Here, again, having learned by interrogation our patient's chief complaint, we should ask ourselves, Is there any striking or predominant sign or appearance (Latin *facies*)? The importance of inspecting our patient cannot be overestimated. In these days of scientific instruments we are too apt to forget to use our faculties. By simply using our eyes many important data may be learned besides the colour of the skin, the condition of the teeth and gums, the general nutrition, the attitude or decubitus, and the facial expression. For instance, the manner in which a patient answers questions is often the first clue to hysteria, and a peculiar mode of speech is one of the pathognomonic signs of general paralysis of the insane, disseminated sclerosis, and other diseases. Moreover, with experience we can by this means form a conclusion as to the kind of patient we have to deal with. Again, never be in a hurry; only by taking time can we fully appreciate all the points presented to us. This habit of "observing" the patient is only developed by long practice; it will never be developed if the young physician allows himself to be infected by the hurry of modern times.

(2) It is important always to commence our examination with that organ to which the symptoms are mainly referable. Some teachers direct their pupils to examine and report on the physiological systems always in the same order (first the heart, then the lungs, then the digestive system, and so forth), whatever may be the malady. But such a course has, to my mind, three objections: (i.) The student goes about his work in a mechanical fashion; (ii.) if the patient suffers from some serious disorder, such as peritonitis, he may be seriously injured by a thorough investigation of the chest and other parts; and (iii.) in many cases it is a waste of time to examine all the organs with equal thoroughness. The same educational advantages and experience can be obtained by the other method, and in that way we come to the most important facts first. As a general rule, the most important data should be mentioned first.

(3) In all cases every organ in the body should be carefully examined; for although we may find in one physiological system sufficient mischief to account for the patient's symptoms, the other organs may reveal changes which considerably modify our treatment, our prog-

nosis, and even our diagnosis. Whatever order is adopted, the student should not wander from organ to organ, but examine each physiological system thoroughly before proceeding to the next. It is well to get into the habit of adopting some such order of physical examination as the following: *First*, note the general condition; *secondly*, examine the organ chiefly affected; *thirdly*, the other organs in the following order: Thorax (heart and lungs), Abdomen (alimentary canal, liver, spleen, and genito-urinary system), Head and Limbs (nervous and motor systems).

The examination should always be carried out *gently*, and *without undue exposure*. In serious cases, especially when the heart or lungs are involved, it is often well to postpone a thorough examination of some organs, so as not to risk harming the patient by exposing or fatiguing him. At the same time, the young physician should never allow modesty to prevent his making a thorough examination. This rule is especially necessary in patients of the better class, but a little firmness, tact, and a courteous demeanour will generally enable him to perform what is a duty both to his patient and to himself.

SCHEME OF CASE-TAKING

A. INTERROGATION OF PATIENT

(*a*) The patient's chief or **Cardinal Symptom.**
(*b*) Data concerning the **Present Illness.**
(*c*) The patient's **Previous History.**
(*d*) The **Personal History.**
(*e*) The **Family History.**

B. PHYSICAL EXAMINATION (i.e., Present Condition—Give Date) [1]

(*a*) **The general condition** may be summarised mainly under three headings: (i.) The Physiognomy or expression, especially in acute disease (Chapter II); (ii.) The Decubitus, Attitude, or Gait, especially in chronic disorders (Chapter II); (iii.) The Nutrition, General Conformation, and any Eruption on the skin (Chapter XVI). The temperature, pulse, respiration and weight should be noted.

(*b*) **Chest.**

I. Cardio-vascular System. (Chapters III to V.)

Symptoms.—Breathlessness, palpitation, cardiac pain.

Physical Signs.—Pulse: rate, rhythm, volume, tension, arterial wall. Heart: palpation, apex beat, percussion area, auscultation, dropsy.

[1] This scheme gives only the *chief points* which should be noted about the different physiological systems, with the object of excluding disease. For an exhaustive examination, such as must be made of the organ to which the patient's symptoms are mainly referable, the student should refer to the chapter dealing with the diseases of that organ.

II. Respiratory System. (Chapters VI and VII.)
Symptoms.—Cough, expectoration, dyspnœa, pain in chest.
Physical Signs.—Rate of respiration, inspection, palpation, percussion, auscultation.
Examine throat and nose.

(*c*) **Abdomen.**

III. Alimentary Canal. (Chapters VIII, IX, X, and XI.)
Symptoms.—Appetite, discomfort after food, nausea, pain, state of the bowels, colour of stools.
Physical Signs.—Examine mouth and tongue, gums, teeth and tonsils. Physical condition of abdomen as regards distension, and presence of fluid or tumour (inspection, palpation, and percussion). Rectal examination.

IV. Liver and Gall-Bladder. (Chapter XII.)
Symptoms.—Pain, jaundice.
Physical Signs.—Size (palpation and percussion), surface (if accessible), tenderness.

V. Spleen. (Chapter XII.)
Any enlargement (palpation and percussion) or local pain.

VI. Urinary System. (Chapter XIII.)
Symptoms.—Any undue frequency of, or difficulty in, micturition. Any dropsy or pain.
Physical Signs—(in catheter specimen when necessary).
(i.) *Urine:* quantity, colour, reaction, specific gravity, albumen, blood, sugar, bile, acetone, aceto-acetic acid, indican, deposit (microscopical examination).
(ii.) *Kidney.*—Any enlargement, mobility, or tenderness.

VII. Generative System. (Chapter XIV.)
Menstruation, frequency, duration, quantity, pain, intermenstrual discharge.

(*d*) **Head and Limbs.**

VIII. Nervous System. (Chapter XIX.)
Symptoms.—Headache, sleeplessness, neuralgia, etc.
Physical Signs.—Weakness or inco-ordination, muscular wasting, involuntary movements, gait. Reflexes, deep and superficial.
Sensation for touch, pain, temperature, joint and vibration sense.
Cranial Nerves.—Vision, fundi, pupils, movements of the eyes. Movements of the face, masseters, palate and tongue, sternomastoids. Hearing. Smell. Taste.
Sympathetic System.—Flush storms, trophic lesions, vasomotor system, perspiration.

(*e*) **Blood.**

In anæmic and some other cases examine the blood (Chapter XVI).

Progress of Case.—Notes (daily of acute or febrile cases, twice a week of subacute, and once a week of chronic cases) should be made of the progress of the case; and much care is required here to avoid redundancy on the one hand, and on the other to record completely all important changes, or any fresh symptoms, and the effect of the treatment adopted. In acute febrile cases there ought to be a daily note, and the pulse, respiration, and temperature should be charted every four hours. In chronic cases it will be sufficient to note, once a week, the persistence of the prominent symptoms or any change in the symptoms. In all cases any *sudden* change in the patient's symptoms or general condition should be recorded at once. Each note should have special reference to the previous one; and before taking a fresh note, the previous one should be read over. The treatment and its effects should always be incorporated; thus, if the patient has been ordered diaphoretics or purgatives, record should be made of the state of the skin or bowels.

History Sheets, Charts, Diagrams, etc.—A history sheet for recording the history of a patient should be ruled with one vertical line down the page one-third from the left-hand margin, so as to give space for information learned subsequently. It should have printed headings and spaces at the top, thus:

Diagnosis. (Space here for primary and secondary disease, filled in by physician afterwards.)

Name..........................**Age**................**Sex**............... **Occupation** ....

Address...Date of admission..

Date of discharge (or death) ....

Chief symptom on admission...

Temperature charts are of the greatest use to record the temperature and other features of diurnal variation.

Outline diagrams or rubber stamps of the various regions of the body can be purchased, and are very useful.

A kind of shorthand code for physical signs is advocated by some authors, and, once learned, may be useful in saving time and space.

§ 3. Examination of Children and Infants.—Here the same general rules apply as to interrogation and physical examination, and we should first endeavour to ascertain the child's leading symptom, either from the patient or the relatives. There are, however, certain additional rules upon the adoption of which much of our success with children will depend.

1. First endeavour to establish friendly relations with your little patient. This may be done sometimes by appearing not to notice the child at first; after a while it may make advances and investigate your watch-chain and be given a toy. A child dislikes being stared at. Time should always be given for the child to become accustomed to your presence, and anything like abruptness will defeat your aim.

2. The questions put to the child should always be of the simplest character—*e.g.*, "Where does it hurt you?" Interrogation of the mother is essential to learn the age up to when the child remained healthy, the symptoms of the present and previous illnesses. In the case of an infant, inquire whether it was a full-time child, if born with instrumental aid, whether it was born healthy, its weight at birth and subsequently, and details about the methods of feeding. If the child is past early infancy, the same questions may still be put, and in addition inquire when it began to walk and talk, and when dentition commenced. Carefully record its present and past

diet, as to its appetite, and the state of the bowels. Ask also how long it sleeps, bearing in mind that children require much more sleep than adults. Then ask for any recent illness in other members of the family.

PHYSICAL EXAMINATION.—Valuable as *attentive observation* may be with adults, it becomes quite indispensable with children, who cannot accurately describe their sensations. Much may be learned while you sit and allow the child to get accustomed to your presence. Notice its expression, the brightness of its eyes, its attitude, the colour of its skin, the state of nutrition, its size as compared with age, its movements, the condition of its lips (moist or dry), the character of the breathing, the sound of its voice. If it cries, inquiry should be made whether this is constant or only at times. Congenital syphilis may be plainly depicted on its face or skin. If the child be asleep when first you enter, do not wake it, but notice all the above before it is disturbed. When awake, the limbs of a healthy child should be constantly on the move; drowsiness, dulness, and listlessness are signs of pyrexia, and especially that of the contagious fevers. The hands are instinctively moved towards a seat of pain—*e.g.*, the head in meningitis. The state of the temper is altered in the prodromal stage of most diseases; but it is markedly peevish in the prodromal stage of meningitis. For other facial alterations, see **Facies** (§ 11). When the child is undressed for examination, the back of the chest should be examined first, while the child looks over the mother's shoulder at some one who attracts its attention with a bright object or a bunch of keys. Percussion should be delayed until the end of the examination.

§ 4. Methods of Diagnosis, Prognosis, and Treatment.

—Diagnosis, prognosis, and treatment are the objects we had in view in eliciting all the facts concerning the patient by the process of "Case-taking." Of these three, **Diagnosis**—which, as the Greek word (διαγνωσις) implies, means the distinguishing or discernment of the disease—is by far the most important. Everything necessarily hinges on this, for without recognition of the disease, rational prognosis and treatment are impossible. It is well, therefore, to consider how the data we have elicited may be utilised in order to arrive at a diagnosis. Several different methods are employed:—

The method usually adopted, which is the outcome of the student's studies in systematic medicine, is to erect a *hypothetical diagnosis*, and to see whether the patient's symptoms tally with the description of the disease. When a child, for instance, with disorderly movements comes before us, the diagnosis of chorea at once occurs to our minds. The age of the patient, character of the movements, and all the obvious features of the case appear to correspond with that disorder. It does not seem necessary to consider any other suggestion. This method answers well enough in straightforward, well-marked, typical cases; but in cases presenting anything unusual or atypical considerable difficulty may be experienced.

Another method of making a diagnosis is by a *process of exclusion*; that is, after studying the diseases which might possibly be in operation, we arrive at our diagnosis by excluding those which the disease least resembles. In such diseases as typhoid fever, where symptoms are few in number, this may be the only method possible. The patient, for instance, is suffering from a moderate degree of pyrexia, the illness came on gradually; that is all we may know about the case. There are many possible causes of such a condition, but we arrive at the conclusion that

it is probably typhoid fever, because all the other possible diseases are rendered improbable for one reason or another.

The third method consists of *noting the cardinal symptoms* and *balancing the evidence* for and against all the possible causes which might give rise to it. In this method, after having elicited all the facts of the case, we return to the patient's *cardinal symptom*, enumerate in our own minds the various causes which might give rise to that symptom, and balance the evidence adduced by the other facts of the case for and against each one in turn. It may strike some as being a little tedious, but it is not so when we have got into the habit of employing it. It is certainly the one best adapted for the elucidation of obscure or atypical cases; and under all circumstances it presents a truer picture to our mind, because diagnosis can never be a matter of absolute certainty. At most a diagnosis is only a strong probability, and this method enables us to ascertain the exact amount of probability in each disease. Even in the simplest and most typical cases it is a good mental exercise for us to keep in mind the other lesions which might produce the same symptoms, and then we are always on the look-out for possible errors, and ready at any moment to review the diagnosis—a correct mental attitude when in the presence of Nature's phenomena. The chapters which follow are based on this method.

Having aimed at a tentative diagnosis, it is advisable to confirm this, whenever possible, by X-ray examination or by special methods of pathological investigation. It should be remembered that these are by no means infallible, but are useful aids in diagnosis. Sometimes radiological evidence lags behind clinical findings. *X-ray and pathological examinations* should only be used in confirmation of a clinical diagnosis and *should never be used in place of clinical methods* of arriving at a diagnosis.

EXAMPLE.—Let us suppose, for instance, that the patient, a pale young woman, aged twenty-three, comes to us complaining of **vomiting blood** (*i.e.*, hæmatemesis).

First, we ascertain and verify this, the leading symptom, and find that she has really vomited a considerable quantity of blood.

Secondly, we INTERROGATE her as to the history of her present illness, her previous and family histories, and we find that she has suffered for several years from symptoms pointing to dyspepsia, and that latterly there has been severe pain in the epigastrium. There are always four features we have to investigate about every pain—its position, character, degree, and constancy; and we find that this epigastric pain is a sharp pain, not constant, but coming on shortly after taking food, and that it is followed *and relieved* by vomiting. The other details of the case we will omit for the sake of brevity.

Thirdly, we proceed to the PHYSICAL EXAMINATION, first of the abdominal organs, but this reveals nothing abnormal. Then we go through the other physiological systems in order, observing (*a*) her General Condition (noting, for example, how pale and thin she is, and how weak she seems); (*b*) examining the Chest (Cardio-vascular and Respiratory systems); (*c*) the Head and Limbs (Nervous system); (*d*) the Blood must also be examined, because anæmia may be inferred from the pallor of her skin and mucous membranes.

Having elicited all the data by interrogation and physical examination, we return to the *cardinal symptom*—hæmatemesis [1]—and consider its various causes (see the section on Hæmatemesis) *seriatim*, taking the most probable cause first.

[1] Here there was no difficulty about identifying or selecting which was the chief or most important symptom; but in another case the anæmia (or the vomiting or

(*a*) Simple Ulcer of the Stomach.

For: (i.) The profuseness of the hæmatemesis; (ii.) the character of the pain (brought on by food, relieved by vomiting); (iii.) the history of dyspepsia; (iv.) the age and sex of the patient.

Against: (i.) No tenderness in the epigastrium.

(*b*) Cancer of the Stomach.

For: (i.) The vomiting of blood; (ii.) pain in the stomach; (iii.) pallor and emaciation; and so on.

Against: (i.) The blood vomited was too profuse, and had not the character special to cancer (coffee grounds); (ii.) the pain was only produced by food, and entirely disappeared after vomiting; (iii.) age of patient much too young.

(*c*) Portal Obstruction.

For: The profuseness of the hæmatemesis.

Against: (i.) Absence of abnormal signs in the liver; (ii.) absence of ascites, piles, and other symptoms of portal obstruction.

(*d*) Other and less probable diagnoses can be discussed in like manner, though each of these may be more summarily dismissed thus: *Vicarious menstruation* would not account for the dyspepsia, acute epigastric pain, and other symptoms. *Leukæmia, Scurvy,* and *other blood conditions,* if present, would present the other symptoms of those maladies.

It follows, therefore, that the balance of evidence is in favour of (*a*) Simple Ulcer of the Stomach, partly because of the weighty arguments in its favour, and partly because the only argument against it is not vital, for tenderness may be absent when the ulcer is situated on the posterior wall of the stomach. Indeed, if a numerical value were given to each of the "reasons" for and against, it would be possible to express the precise degree of probability of each disease in the form of a mathematical ratio. This method may at first sight seem tedious, but after a little practice it becomes automatic and extremely simple; and it takes much less time than is here implied. Later, X-ray examination may confirm the diagnosis.

Prognosis (from the Greek word πϱογνωσις) is a "foreknowledge" of the events which will happen—*i.e.*, of the probable course the disease will run. Nothing but wide experience, combined with careful and minute observation, will enable a physician to prophesy with any approach to accuracy. It will, however, be useful to bear in mind that the prognosis of a case depends upon four circumstances—viz., (1) the *usual course,* duration, and event of the disease in operation (phthisis, for instance, runs a prolonged course, and until lately the event was almost invariably fatal); (2) the presence or absence of *untoward symptoms* (*e.g.*, profuse hæmoptysis in phthisis); (3) the presence or absence of *complications* (which are sometimes more fatal than the disease itself—*e.g.*, typhoid and many other fevers are fatal chiefly by their complications); and (4) the *causes* which are in operation, including among the predisposing causes

epigastric pain) might be the more serious or prominent symptom, the hæmatemesis consisting, perhaps, of a few streaks of blood. Then we should deal with the anæmia in the same way as hæmatemesis is here dealt with. Sometimes a good deal depends upon our choice of which is the "leading symptom," for it is not always the most prominent which is the most serious and important; and by an error in this respect we may be led far afield from the true disease. At times, however, it is useful to change the point of view we take of the case, by regarding it from another standpoint or leading symptom.

such data as age and sex (gastro-enteritis, for example, in middle life is not a serious affection, but in infancy and old age it is often fatal).

As practical hints to the young physician, I would advise him—(1) Never to commit himself to a prognosis unasked, or before awaiting the result of treatment. More reputations are wrecked on the rock "Prognosis" than on any other. (2) Avoid giving a prognosis before all the facts of the case are to hand (including the results of X-ray and other special examinations). (3) It is also well to impress upon the friends that a "physician" cannot hope to be also a "prophet"; and that prognosis may depend on many factors in the case which are not yet revealed. (4) When the physician considers that the prognosis is grave, he should not even hint this fact to the patient; he guards his own reputation if he informs a responsible relative.

Treatment is what the patient comes to us for; and it may be of three kinds: (1) In *Specific* (*or Radical*) treatment our object is to cure the patient of his disease by the removal of the cause. This is the only truly scientific treatment, and it is based mainly upon a knowledge of the pathology of the malady. (2) *Symptomatic* treatment is directed only to the relief of the symptoms. In some incurable maladies symptomatic treatment is the only kind possible, and all that we can do is to ease the passage to the grave. But in the practice of busy practitioners, the trouble and time needed for thorough investigation often lead to the adoption of the latter at times when a more radical treatment would be possible. We should constantly guard against an unfortunate tendency to fall into a routine of symptomatic treatment. Both Specific and Symptomatic treatment may be either internal or external on the one hand, and either medicinal or dietetic and hygienic on the other. (3) *Preventive* treatment has within the last quarter of a century developed almost into a separate science, the science of Hygiene or Social Medicine.

§ 5. General Rules in Clinical Investigation.—There are certain habits which the student should strive to cultivate when he comes to the practical aspect of his profession; and he should remember Thackeray's saying: "Sow an act and you reap a habit; sow a habit and you reap a character; sow a character and you reap a destiny." Clinical medicine depends more than anything else on accurate, complete, and well-directed observation, and there are six hints I would give to the student in this connection.

1. *Avoid superficiality* in your observations. Do not try to see many cases in one day, but rather one or two cases *continuously from day to day*, so that you may follow a given disease throughout its entire course. It is of more value to follow up one case in this way than to see a dozen on one occasion only. Practical knowledge must be acquired gradually. The thought will often occur to the student how slowly he progresses with his clinical knowledge. This is partly apparent, partly real. It is partly apparent because a student does not realise at the time the value he derives from listening, for example, to the same cardiac murmur over and over again. It is partly real because it is only by patiently devoting

the necessary time to the study of the same case from day to day that he will learn to make his observations adequate, thorough, and precise. That is why many a brilliant intellect falls behind, and many a plodder comes to the front in our profession. It is vain to attempt to substitute genius for patient industry in this arena. You must learn for yourself the effects of this or that line of treatment; learn to correct and control the observations you make one day by your observations of the morrow; and above all, try to learn what is the sequel or termination of the case, especially in such instances as may lead you to the post-mortem room. There, more than anywhere else, the most brilliant diagnosticians learn from their own errors more than from a multitude of successful cases.

2. *Do not strive after what is rare and curious.* It follows, as a matter of course, that, other things being equal, a fact is more important in proportion as it is more common. Moreover, by studying *only* the exceptions to a rule, our minds will have a distorted view of clinical phenomena. Do not therefore be led astray by those pedants who seek after the singular and uncommon. It is well to see rare cases when the opportunity offers, but be careful that you mentally register them as rare.

3. *Do not study only acute and severe cases.* It is true that in acute diseases there is often more to be done, more heroic and decisive effects to be produced, or apparently produced, and therefore more credit and renown to be obtained. But we shall find in actual practice not one-tenth, perhaps not one-hundredth of our patients will be suffering from these complaints. Our success, therefore, in practice, whether measured by that laudable satisfaction at having done one's duty, or by the pecuniary reward of which every earnest labourer is worthy, will depend very much on our experience of, and our ability to treat, chronic and what we are too apt to call trivial complaints. For one case of Graves' or Addison's disease, the student will, I venture to think, have a hundred cases of dyspepsia, chronic rheumatism, or chronic bronchitis. In the treatment of such complaints the greatest judgment and thoroughness are sometimes needed. No sudden or startling effects can be produced. Chronic diseases require chronic remedies, and it is only by experience that one can learn to produce those gradual effects which lead to a successful issue.

4. *Be accurate in your observations.* State facts precisely as you find them, no matter whether they accord with your hypothesis or not; and state only what you find and know to be the truth. The study of clinical medicine, like the study of any other of Nature's phenomena, should inculcate in the mind of the student a love of truth. It is impossible to have any dealings with Nature without learning that truth is the key to the discovery of her secrets. Accuracy is one form of truth, and it is only by repeatedly going over your observations, and sifting the patient's statements, that you can ensure accuracy.

5. *Be complete in your examination of your patient.* It may not be possible or advisable to make a complete examination when you see your

patient for the first time, or with a new illness. Many mistakes in diagnosis would be avoided if this rule were adhered to.

6. *Be systematic in the arrangement of your data,* for only by a systematic arrangement can you attach the proper importance to each observation, and get a firm grasp of the whole case. Nothing, for instance, is more liable to confuse and to prevent you from coming to a correct conclusion than wandering from one date to another without regard to the chronological sequence in the history of an illness. And again, in physical examination, nothing is so likely to lead you astray as wandering from organ to organ without first completing the examination of each.

§ 6. **Classification of Diseases—Method of Procedure.**—It has been customary, and the practice is convenient, to classify diseases into two great groups—Local and Constitutional. LOCAL diseases are those in which the principal, and perhaps the only, lesion is localised in one organ or situation, *e.g.*, facial neuralgia, ringworm. CONSTITUTIONAL diseases are those in which the disease has manifestations of general distribution, *e.g.*, acute rheumatism, typhoid fever, and pyæmia.

It is convenient for clinical purposes to preserve this division, but the rapid advance of pathology has gradually transferred disorders from the "local" to the "constitutional" group. A large number of diseases formerly believed to be lesions of local origin (such, for instance, as pneumonia, endocarditis, and peritonitis) are now known to be due to some general morbid process, toxic or microbic, which, on reaching the blood, is carried all over the body and causes a special local manifestation in one situation.

From a pathological standpoint diseases are sometimes divided into two groups—Organic, those in which some anatomical change is found after death; and Functional, those in which we can, *in the present state of our knowledge*, find no structural alteration by modern methods. The anatomical or structural change is spoken of as the "lesion." The word "functional" must not be regarded as synonymous with "hysterical."

Now it so happens that local disorders are very often met with as complications or effects of constitutional or general conditions; and since in clinical work we are engaged in **tracing from effect to cause,** we shall, in the following chapters, take the local diseases which are manifested by a lesion *localised* in some particular organ first, and the *constitutional* conditions afterwards.

When a patient comes to us, and if, as the result of our inquiries, we find he is suffering from a symptom localised to some organ (*e.g.*, pain in the liver), turn to the chapters relating to the diseases of that organ (one of the Chapters III to XIV).

If, on the other hand, he has no localised symptom, but complains of malaise, feverishness, or a sense of "bodily illness," turn to the chapters on constitutional diseases (Chapters XV to XX).

CHAPTER II

THE FACIES, OR EXTERNAL APPEARANCE OF DISEASE [1]

In our scheme of case-taking it will be remembered that the first step in physical examination was to observe the patient's general condition; and it will also be remembered how great was the importance of an adequate inspection of the patient while he was telling us the story of his illness.

Some diseases can be identified almost at a glance, before the patient opens his lips, such, for instance, as Chronic Alcoholism, some manifestations of Hereditary Syphilis, Graves' Disease, Cretinism, Myxœdema, Facial Paralysis, and Hydrocephalus, when these conditions have passed beyond the incipient stage. The existence of others can be very strongly suspected, such as Rickets, Post-nasal Adenoids (mouth-breathing children), and Chronic Bronchitis with Dilated Right Heart.

But, apart from these, much may be learned from the first glance at a patient—from his *decubitus* (the way he lies), from his *attitude* or *gait*, from the expression of his *face*, the colour of his *skin*, and from the *general conformation* of his body—without the employment of any special methods or apparatus for diagnosis. It is to be feared that as scientific methods become more and more perfect, these means, which constitute one of the most useful and important aids to diagnosis and prognosis to the experienced busy practitioner, are apt to be neglected. But, on the other hand, students and young practitioners had better not attempt "lightning diagnoses," or they will certainly fall into the most serious errors. Some men, it is true, seem to be especially gifted in this way; but it is only by long experience and the possession of special faculties that they can accomplish such feats.

It is a fundamental rule that your patient should face the light at all medical interviews. Similarly your own chair should be in the shade, lest the patient should read too readily what is passing through your mind. It is surprising what important clues can be obtained by an intelligent inspection of your patient, both as to his character and his disease.

The facies of disease may be summarised under three headings: (A) The Physiognomy in Disease. (B) The Decubitus, Attitude, or Gait. (C) Alterations in the General Conformation of the Body.

Hints to be derived from an inspection of the hands are given under Diseases of the Extremities (Chapter XVII). The various diseases will be only mentioned here. The description and differentiation of the several affections referred to will be entered into more fully in the chapters which follow.

[1] The Latin word *facies* signifies an appearance, form, or shape.

(A) The Physiognomy in Disease.

An observant physician can obtain important clues to diagnosis by the physiognomy—*i.e.*, the aspect and expression of the patient's face —even apart from the insight which can be gained by this means into his character.

§ 7. In **Acute Diseases** more can be learned from the position in which the patient lies (*i.e.*, his Decubitus, § 14) than from the physiognomy or expression of his face. But it is worth remembering that the face assumes an *anxious expression*, which is very characteristic in pericarditis, peritonitis, and severe pneumonia, also during attacks of angina pectoris. The supervention of *acute pericarditis* in the course of rheumatic fever is often unsuspected, as there may be no local symptoms; but it may be recognised by this anxious expression, the dilated nostrils, and the flush upon the cheeks, which were (probably) at our last visit so pale. Again, in acute *lobar pneumonia*, the appearance is very distinctive. The flushed face, hot dry skin, widely dilating nostrils, the eruption of herpes near the mouth, and the profound disturbance of the pulse-respiration ratio (2 : 1 instead of 4 : 1, which is the normal), form a picture which greatly aids the recognition of the disease. The *Facies Hippocratica*—a facies or appearance, of which the description has been handed down from Hippocrates—is the forerunner of death from exhaustion, such, for instance, as the final stage of cholera, and wasting disorders. The temples are hollow, the eyes sunken, the eyelids slightly parted, the eyes glazed, and the lower jaw droops. The *Risus Sardonicus* is a fixed grin, met typically in tetanus. The corners of the mouth, which twitch at intervals, are drawn upwards as in laughter, and the features assume a fixed sarcastic expression.

§ 8. A few **Chronic Diseases** may be enumerated in which the physiognomy is characteristic.

(i.) The aspect of a *phthisical* or *tuberculous* patient differs in the premonitory and advanced stages. (*a*) Before any evidences can be detected by physical examination of the chest, the patient has the appearance which is loosely described by the laity as "delicate." The skin is fine and soft, and the fresh, rosy colour of the cheeks is out of keeping with the dark rings around the sunken eyes. But it is the deficient measurements and movement of the chest and the local flattening which lead us to suspect the presence of tuberculosis. Sometimes such patients are plump and rosy; nevertheless, they have a deficient chest measurement. (*b*) When the disease is advanced, the phthisical patient often presents an appearance that enables the physician to hazard a diagnosis almost without further investigation. The pale, emaciated face, with sunken eyes, the circular crimson flush of hectic fever on the cheeks, the wasted body, bathed from time to time in sweat, the hoarse voice and easily-provoked dyspnœa, collectively form a picture which is very characteristic.

(ii.) *Chronic bronchitis with dilated right heart* is another condition of extremely common occurrence in the practitioner's daily practice, and the picture these patients present is very characteristic. The florid "healthy" looking cheeks, the distended jugulars, in a person over forty are very typical.

(iii.) In *chronic alcoholism* there is a puffiness of the face and a congested watery look about the eyes ("a blear-eyed look"). The eyelids are puffy, so that the person is described by sailors as having "an eye like a poached egg." The cheeks and nose are often red, and dotted with stellate venous capillaries. The belly is corpulent; and on holding out the hands and spreading the fingers, these are seen to be affected with fine small rhythmical tremors. The whole picture is unmistakable, though the eyes alone will tell the tale.

In various **diseases of the nervous system** the *face* presents a pathognomonic expression. Thus in Bell's or facial paralysis the face is *distorted,* and so also in that rare condition facial hemiatrophy. In tabes dorsalis the unequal pupils, drooping eyelids and wrinkled forehead are diagnostic (Fig. 184). The expression is *vacant* in idiocy and some hysterical subjects. A smooth, *expressionless* appearance (differing from the preceding in that there is a lack of mobility) is characteristic of paralysis agitans, and among rarer conditions, of double facial paralysis, the myopathies affecting the face muscles, and scleroderma. Very characteristic is the *spastic* smile, with open mouth, met with in lenticular degeneration. Bulbar paralysis gives a characteristic, *mournful,* or *sullen* appearance to the face; the orbicularis oris is paralysed, and allows the lower lip to pout; while the weakness of the zygomatici results in a drooping of the corners of the mouth, such as we usually associate with sorrow or sullenness of temper. In a more advanced stage the saliva dribbles out of the mouth. Certain *spasms* and *tremors* are recognised at a glance (§ 770 *et seq.*).

§ 9. **Swelling of the Face** and neck, if associated with œdema of the limbs and trunk, may be part of a generalised dropsy. In the dropsy of *renal disease,* the swelling is most obvious in the loose cellular tissues around the eyelids. The puffiness of the eyelids due to renal disease is, however, greater in the morning than in the evening, and in this way may be distinguished from a similar condition due to arsenical poisoning or whooping-cough. The dropsy of cardiac disease is more noticeable in the dependent parts of the body.

Swelling of the face, accompanied sometimes by flushing after meals, is a symptom for which dyspeptic patients often seek advice (acne rosacea). It is also seen in urticaria. In chlorosis and severe anæmia the pallor of the skin may be associated with some œdema. A *firm swelling* of the face occurs with scleroderma and scleredema. *Bilateral swelling* may be due to mumps and *unilateral swelling* to an infected tooth or antrum.

Chronic œdema around the eyelids must not be mistaken for myxœdema. It may be due to recurrent eczema, blepharitis, erysipelas or ethmoiditis. It is also met with in nervous or hysterical conditions, and in cases of vasomotor instability; transient exacerbations occur with fatigue and liver derangement. Œdema confined to the *head and neck* is found in those rare cases where there is pressure on the veins within the thorax, especially the superior vena cava, as in cases of mediastinal tumour.

Myxœdema is often recognised by a glance at the patient's face and

hands (Fig. 1). There is a solid œdema and puffiness of the face—the body and limbs being also affected—but it does not *pit on pressure.* The vacant, stolid look, flushed cheeks, yellowish skin, scanty, coarse, dry hair, supra-clavicular pads and slow speech are equally typical of this disorder.

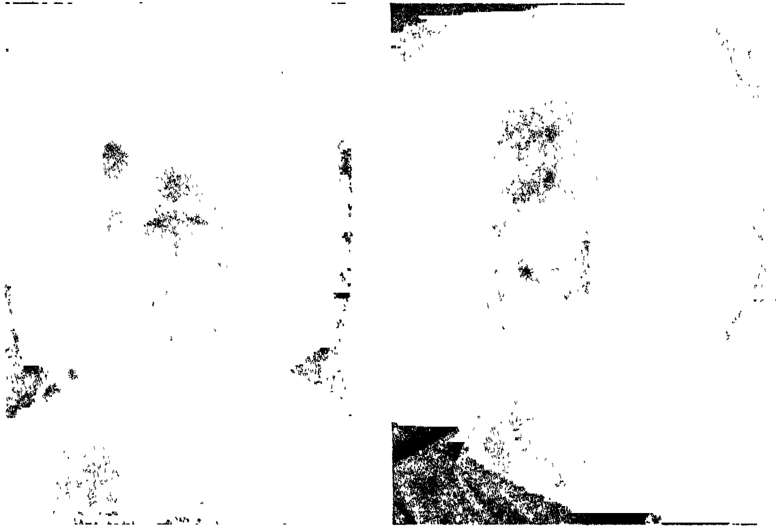


FIG. 1.—MYXŒDEMA. (*Left*) Before treatment: (*Right*) After treatment with thyroid by mouth.

The hands are flat, coarse, and swollen (see § 559). In *acromegaly* the lower jaw, supra-orbital ridges, lips, cheeks and end of the nose are thickened and enlarged (Fig. 6 and § 598).

Various forms of parotitis, acute and chronic, cause swelling. Acute forms may appear in connection with specific fevers, or disease of the pelvic organs. In *Mikulicz' syndrome* there is a chronic bilateral painless swelling of the lachrymal and salivary glands, or of the latter only. The cause is unknown. The glands are densely infiltrated with small round cells, and do not recur if removed. Swelling of the same glands may occur in leukæmia, syphilis, tuberculosis, lymphadenoma, lymphosarcoma and lead poisoning, conditions which therefore must be eliminated before undertaking any operation. In the *uveo-parotid syndrome*, after an initial period of malaise, fever develops and iridocyclitis, bilateral facial palsy (§ 859), bilateral parotid and lachrymal gland enlargements, occasionally polyneuritis and skin rashes. This is a variety of sarcoidosis (§§ 141*a* and 647).

§ **10.** The **Complexion** and colour of the face will repay careful inspection, for thereby the trained observer will acquire useful information. Thus, the *pallor* of anæmic and toxæmic conditions is often very striking. So also is the pallor, or rather *sallowness*, of aortic valvular disease; the dead white or *waxen puffy* appearance of parenchymatous nephritis; the *greyish pallor* of chronic interstitial nephritis; the *greenish* hue of chlorosis; the *lemon* colour of pernicious anæmia; the *deeper yellow* colour of regular mepacrine dosage;[1] the *deep yellow* to *greenish-yellow*

[1] It is only by long experience that one can distinguish these refinements of shade.

colour of jaundice; a *faint yellow* tinge with pallor occurs with old age, early catarrhal jaundice, cholæmia and severe anæmias. A *muddy* sallow complexion may be associated with dyspepsia, chronic constipation or other colonic disorders. The *dull earthy* look occurs with malarial cachexia, cancer, and chronic abdominal disease. The *purple* (or cyanotic) appearance of the cheeks and lips in congestive heart-failure and congenital heart disease, and the congested appearance of polycythæmia, chronic bronchitis and chronic alcoholism, are distinctive; so also are the *grey* or *violet* complexion of sulph-hæmoglobinæmia and methæmoglobinæmia. *Dark rings* around the eyelids appear in states of fatigue; they often indicate want of sleep, intestinal disorder, a septic focus or indigestion, and may be so pronounced in malarial conditions as to resemble the ecchymosis of a bruise. *Bronzing* is seen with Addison's disease, arsenical poisoning, hæmochromatosis, Gaucher's disease, and in half-castes.

Greasiness of the face occurs with acne vulgaris and seborrhœa oleosa.

§ **11.** The **Face in Detail** merits a little closer study, and, first, that most eloquent portion of it, the eyes.

(i.) The *eyes* may be *protuberant* as a whole (Proptosis) with myopia, in Graves' disease, intra-ocular tumour or hæmorrhage, mucocoele of the ethmoid sinuses, oxycephaly, advanced cerebral tumour, thrombosis of the cavernous sinus, or irritation of the cervical sympathetic. Enlargement of the thyroid gland is often associated with exophthalmos (Fig. 2). Some degree of exophthalmos may also be seen as a family trait, with chronic toxæmia such as occurs with cirrhosis of the liver and interstitial nephritis. The appearance of protrusion may be caused by loss of intra-orbital fat. The eyeballs may *recede* in paralysis of the cervical sympathetic, in wasting diseases, and those associated with dehydration. The pallor of anæmia is seen in the conjunctivæ; and in the sclerotic, or white of the eye, the tinge of *jaundice* can often be detected when the yellow colour of the skin is so slight as to escape notice. The sclerotic may also be yellow in severe anæmia and in old people; it may be bluish in congenital heart disease, in liver disease and in association with fragilitas ossium. The "*arcus senilis*" is a white ring of opacity in the cornea, just within its peripheral margin. It was once believed to indicate senile degeneration of the arteries and of other tissues, but this is erroneous. In adults who are subjects of hereditary syphilis, the corneæ may present *striæ* or the



FIG. 2.—EXOPHTHALMIC GOITRE.
(*Photograph lent by Sir Thomas Dunhill.*)

appearance of ground glass, due to interstitial keratitis, which may be confused with scars of corneal ulceration. Brownish-yellow, triangular pigmentation of the sclerotics, confined to the area uncovered by the lids, is seen in the Gaucher type of splenomegaly. Alterations of the *pupil* are dealt with in § 838.

(ii.) The *lips* may show the pallor of anæmia on the one hand or the congestion or cyanosis of cardiac disease on the other. The mouth is held open when nasal obstruction is present, in idiocy, cretinism, and certain paralyses. Fissures may be due to perlèche or, when indurated, to syphilis. Stellate cicatrices around the lips are a record of previous or hereditary syphilis. Dryness of the lips occurs with fever and gastric disturbance. The position and movements of the mouth are characteristic in facial and bulbar paralysis, in the Landouzy-Déjérine type of myopathy, and in the tremors of general paralysis of the insane.

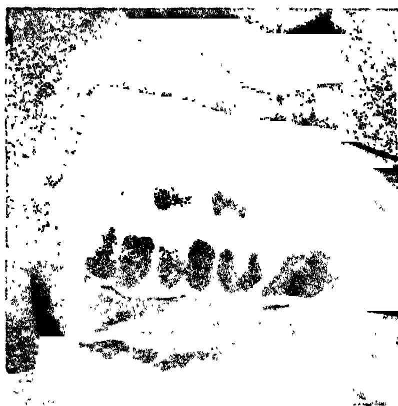


FIG. 3.—Hutchinson's Teeth.

(iii.) The *teeth* may present evidence of pyorrhœa or of hereditary syphilis, in which disease the permanent incisors (erupting at the age of 7–8 years) are characteristically "pegged"—*i.e.*, narrow at the cutting edge, and notched (see also Hutchinson's teeth, § 204). Ridged teeth usually denote stomatitis in infancy.

(iv.) Depression of the bridge of the *nose*, if marked, is due to chronic rhinitis in childhood (usually of syphilitic origin), or to hyperteleorism. In such cases the nose is characteristically broad and flat, or small and "snub," like a button, the opera-glass nose of Fournier. The end of the nose is enlarged in acromegaly, myxœdema and rhinophyma.

(v.) The *ears* may reveal diagnostic evidence of lupus erythematosus, circulatory disturbances, and the tophi of gout.

(vi.) Defective development may be recognised by "stigmata," such as epicanthic folds, hare-lip, cleft palate, accessory auricles, and dermoid cysts.

(vii.) The *tongue* is considered in § 212 *et seq.*

§ 12. The **Physiognomy of Childhood** requires considerable experience to appreciate it fully; then it lends us invaluable aid.

(i.) *Infantile atrophy* or *marasmus* gives to an infant a very characteristic pinched or "senile" face. The complexion is greyish white, the face is sunken and livid, and the skin hangs in folds. The eyes lie deeply in their sockets from which the fat has disappeared, and thus give to the infant the appearance of a little wizened old man.

(ii.) When an infant is experiencing *pain* the face will sometimes give

a clue to its situation. Thus, a wrinkling of the forehead or frown is indicative of pain in the head; a drawing-up of the mouth at the corners, producing marked naso-labial folds, points to severe abdominal pain; a dilatation of the nostrils and elevation of the eyebrows may suggest intrathoracic discomfort; and in tabes mesenterica and other chronic wasting diseases the face gradually assumes a fixed or contracted condition, in which the angles of the mouth are depressed.

(iii.) Nothing is more characteristic than the *listless* and apathetic facies of children suffering from the early stages of fever.

(iv.) *Mouth-breathing children* (often due to post-nasal adenoids) have a very characteristic expression. The broad bridge of the nose and open mouth give to them a vacant, stupid appearance, which sometimes belies their intelligence, though sometimes they are, in fact, mentally backward.

(v.) The *fontanelles* afford useful information. A *depressed* fontanelle is due to dehydration and is an untoward sign in all acute illnesses of infancy—*e.g.*, diarrhœa and vomiting. The fontanelles *bulge* in inflammation of the meninges, and this is a useful diagnostic feature between true meningitis on the one hand, and fevers, broncho-pneumonia, and other diseases with cerebral symptoms on the other. The fontanelles are bulging and tense in all diseases causing increased intracranial pressure—*e.g.*, cerebral tumour. Normally, the anterior fontanelle should close by the age of one and a half, and the posterior fontanelle at birth. In rickets and hydrocephalus the anterior fontanelle is late in closing.

§ 13. **Variations in the Form of the Skull** are met in several complaints, and chiefly in children, because cases of marked deformity of the head seldom reach adult life, except in an institution for the mentally defective. The following are noteworthy:—

(i.) *Asymmetry* may be congenital, due to a difficult labour, or acquired in early life from the continual nursing of the infant on one arm. The head is flattened on the side on which it rests. Nursing on the other arm will correct the deformity in the most surprising way.

(ii.) In *hydrocephalus* (§ 830) the head is large out of all proportion to the face, and the forehead overhangs the face.

(iii.) In *rickets* the skull is large and square, but the forehead rises straight up and does not overhang. There are often bosses in the frontal and parietal regions.

(iv.) In *hereditary syphilis* the bones around the anterior fontanelle are thickened, and there are irregular areas of thickening and thinning (cranio-tabes), especially behind the ears. The condition resembles that found in rickets, with which it may co-exist.

(v.) In *microcephaly* the forehead is receding and the cranium very small. The children are mentally defective. In *scaphocephaly* the head is elongated and its lateral diameter diminished. *Brachycephaly* indicates that the head is shortened from before backwards: an extreme variety occurs in *oxycephaly* in which the head is very tall ("steeple-shaped"), with exophthalmos and slanting eyes. Defective mental development may co-exist with other "stigmata of degeneration," such as high arched palate, accessory auricles, etc.

(vi.) In *adults* signs of infantile malformations may be found. Localised thickenings may also be seen in osteitis deformans, leontiasis ossea, and after injury.

(vii.) In *acromegaly* (§ 598) the lower jaw and often the nose are enlarged. The face is ovoid with the longer transverse diameter below. See Fig. 6.

(viii.) In *osteitis deformans* (Paget's disease) the face is ovoid but with the longer transverse diameter above.

(B) Decubitus (in Acute Conditions) and Attitude (in Chronic Diseases).[1]

§ 14. **Decubitus** signifies the position which a patient tends most constantly to assume, and it often gives a valuable clue to the disease, more especially in the diagnosis of **Acute Diseases,** and sometimes as to their probable issue as well. For example:



Fig. 4.—The attitude typical of Paralysis Agitans; from a plaster cast by M. Paul Richer.

(i.) *Sitting up in bed*, propped up with pillows, on account of inability to breathe in other positions (orthopnœa), is characteristic of the extreme breathlessness which occurs in advanced cardiac, pulmonary, or renal disease; and sometimes also in acute disease, such as pneumonia.

(ii.) *Lying on one side* is characteristic of considerable pleural effusion, pericarditis, or pneumonia on that side, as in this position free play is given to the healthy lung. When a patient with a chronic cough always lies on one side, suspect a cavity, bronchiectasis, or empyema of that side. A patient curls up on one side in colic and in certain forms of meningitis.

(iii.) The *dorsal decubitus*—*i.e.*, lying on the back—is seen in grave illnesses attended by marked prostration. (*a*) In the "typhoid state" the limbs are stretched out and completely relaxed. The typhoid state, so called from its occurrence in typhus and typhoid fevers, is a condition of profound prostration, attended by unconsciousness or muttering delirium, sordes on the teeth, and a dry, cracked tongue. (*b*) If the prostration be due to peritonitis, the legs are drawn up, so as to relax the abdominal muscles; and for the same reason the breathing is thoracic and the abdomen is quite still. The greater flexion of one leg may give a clue as to the side on which the trouble exists.

(iv.) *Opisthotonos* is an arching of the back which occurs in some

[1] The various characteristic gaits are described in § 705.

convulsive and spasmodic disorders. It may be so great that only the head and heels touch the bed. It is met with in tetanus, hystero-epilepsy, strychnine poisoning and in the cerebro-spinal meningitis of infants.

(v.) *Retraction of the head* is characteristic in cerebro-spinal and posterior basic meningitis. It is also met in the meningismus of infants with digestive disorders, otitis media, or febrile states (§ 731), in dyspnœadul to laryngeal obstruction and in rare cases of cervical caries.

(vi.) *Restlessness* occurs in many disorders, acute and chronic, and is generally a grave sign in the former—*e.g.*, in acute pericarditis. Sometimes, as in children, it is an indication of severe pain. *Carphology* (*καρφος*, the clothes; *λέγειν*, to pluck) is the picking at the bedclothes so characteristic of the "typhoid state." The hands seek after imaginary objects. *Subsultus tendinum* is the muscular twitching or tremor which occurs in the same state. Both of these imply extreme cerebral depression. They are met with in the malignant forms of the acute specific fevers, and are of the gravest possible import.

§ 15. The **Attitude** which is involuntarily assumed by a patient suffering from certain chronic diseases, if he be able to leave his bed, is very characteristic. Thus:

(i.) In *paralysis agitans* the head, neck, and thorax are bent forwards, the arms are flexed at the elbows, the body moves stiffly, as if a statue, and the patient has the characteristic "festination gait" (Fig. 4). The disease is recognisable at sight by the smooth, expressionless face, fixity of gaze (always looking forwards), the forward bending of the body, tremors of the hands, and the short steps which the patient takes as he shuffles along. (ii.) The attitude assumed by children suffering from *post-diphtheritic paralysis* is somewhat similar to the preceding, and is so characteristic that one can often detect the disease as the patient enters the room. The head hangs forward from weakness of the neck muscles, and the "flabbiness" of all the movements is peculiar. (iii.) The *rigidity*



FIG. 5.—PSEUDO-HYPERTROPHIC PARALYSIS. [From Taylor, *Practice of Medicine*, J. & A. Churchill.]

of the spine in rheumatoid arthritis, osteoarthritis, and spinal caries, gives a stiffness and awkwardness to all the movements which is very noticeable.

(iv.) Duchenne's *pseudo-hypertrophic paralysis* (Fig. 5) is a comparatively rare condition, but the arching forwards of the back, prominence of the buttocks, scapulæ, and calves, and inability to rise from a recumbent posture without the aid of the hands, are quite pathognomonic.

(C) THE GENERAL CONFORMATION.

§ **16.** Under this heading we note (*a*) whether the patient exhibits any loss of flesh (EMACIATION, *infra*); (*b*) whether he presents any increase in volume (GENERAL ENLARGEMENT, §§ 17 and 18); or (*c*) whether he presents any DEFORMITY or DWARFISM (§ 19).

Here we meet with several important diseases affecting the skeleton and general growth of the individual, including Hereditary Syphilis. The various causes of such alterations will only be mentioned here. They will be described and differentiated under the Diseases of Extremities, and elsewhere.

VARIATIONS IN HEALTH.—The terms "Emaciation" and "General Enlargement of the Body" are only relative. The healthy man should have an elastic skin, firm muscles, and a small amount of subcutaneous fat; but *individual variations* are so great that no definite standard can be set up as normal. Health in the wiry, nervous man is consistent with a spareness that would indicate disease in his stouter and more phlegmatic brother. The same holds true with regard to *age*. A child has an amount of fatty covering that would be abnormal in adolescence; an old man has atrophy of the soft parts and prominence of the bones which in the middle-aged man could only accompany serious disease. The question of build is very largely one of *heredity*: stout parents usually have children who tend to become stout, and *vice versa*.

Emaciation is necessarily attended by more or less weakness, and the subject is dealt with under General Debility (Chapter XVI).

The chief causes of debility with emaciation are as follows: Malignant disease, digestive disorders and privation, diabetes, tubercle, various nervous disorders, hyperthyroidism, pituitary cachexia (Simmonds' disease), sub-acute and chronic infections and toxæmias, chronic nephritis, syphilis, and pancreatic diseases; and in children, defective feeding, diarrhœa, and chronic infections including tuberculosis and hereditary syphilis.

In *advanced life* the first cause which occurs to our minds, if the patient has lost flesh, is cancer; in *middle age*, diabetes; and in *young adults*, tuberculosis. In tuberculosis of the lungs or elsewhere, emaciation may occur before any physical signs can be detected; indeed, loss of flesh which is accompanied by an intermitting pyrexia generally means latent tuberculosis. In *infancy* the two most common causes of acute or *rapid* wasting are defective feeding and acute gastro-enteritis. The most

common causes of slow, progressive, or *chronic* wasting in infants are defective feeding and environment, cœliac disease and tuberculosis.

Emaciation of the face and upper part of the body, with enlargement below the waist, is seen in *lipodystrophia progressiva*, a rare disease probably of endocrine origin.

§ 17. **General Enlargement** of the body is much less often met with than diminution. It occurs in *Obesity, Generalised Dropsy* (see §§ 9, 18 and 29), *Myxœdema* (Fig. 1, § 9, and § 559), *Acromegaly* and in *Eunuchs.* Hypersecretion of the anterior lobe of the pituitary gland, when in a child, leads to gigantism; when occurring in the adult, it causes acromegaly. Enlargement of the body, with sexual precocity,



Fig. 6.—Acromegaly.

may occur with tumours of the pineal gland. Obesity sometimes follows lesions of the nervous system, such as growths in the region of the hypothalamus.

§ 18. **Obesity,** the excessive accumulation of fat in the subcutaneous and deep tissues, is due to excessive intake as compared with output of calories. Two groups of cases are recognised: (*a*) exogenous, when the chief factors are overfeeding, or deficient exercise, or both combined, as in middle age and in sedentary workers; (*b*) endogenous, where endocrine factors are at fault, and obesity occurs in spite of small caloric intake. In some individuals, both causes may be in operation. The tendency to obesity is often hereditary.

(*a*) *Exogenous obesity.* Excessive intake of carbohydrate and fat is

deposited as fat; excess of protein less often acts thus, on account of its stimulant effect on metabolism (specific dynamic action). The obese individual is often below par and lethargic, is liable to develop diabetes, hyperpiesis, arterio-sclerosis, myocardial degeneration, arthritis of the knees, flat foot, sterility, liver and kidney troubles, and has been shown by the statistics of Insurance Companies to have a shorter expectation of life.

(*b*) *Endogenous* or *endocrine obesity* is due to a deficiency of one or several of the endocrine glands. Thyroid, pituitary and ovarian deficiency may cause obesity at any age, but especially at puberty and the menopause. Tumours of the pituitary, suprarenal, pineal and thymus glands are rare causes. In many cases the usual stimulant effect of food on the metabolism is deficient. (i.) Obesity due to thyroid deficiency shows fat deposits on the shoulders, back of the neck and the supra-clavicular regions, a dry skin, and falling hair or other signs of early myxœdema (Fig. 1). A minor degree of this form of obesity is common at the menopause. (ii.) Deficiency of pituitary secretion is a common cause of obesity in the young. The fat is deposited over the lower ribs, the hips, buttocks and abdomen; blueness of the extensor surfaces of the upper arms, thighs and buttocks may be present. The skin is usually clear, almost hairless on the body; there may be drowsiness, low temperature, decreased menstruation, and increased sugar tolerance. Pituitary obesity occurs with underdevelopment of the gland or when a tumour destroys it; this can be demonstrated radiologically by an abnormally small pituitary fossa, or by evidence of erosion of the bone of the fossa. Cushing's syndrome (p. 28) is a special variety. Or the gland may be injured by acute or chronic infection, as by encephalitis lethargica or by long-continued toxæmia from a septic focus. (iii.) In ovarian deficiency the fat tends to be deposited on the abdomen and thighs, and flushes are complained of.

Deficiency of all three glands may be present in varying proportions, especially at and after the menopause.

Treatment. The caloric intake must be reduced to a level lower than that of the output and continued until the requisite weight is lost. Forbid "snacks" and sweets between meals. Protein can be taken more freely than carbohydrate and fat, owing to its stimulant effect on metabolism. In healthy subjects one may order an initial period of fasting for two or three days; only fruit juice is allowed. Follow this with a diet with an intake limited to 1250 calories (§ 297 VIII). To reduce appetite, omit salt and condiments. Vegetables with 5% carbohydrate and raw fruit may be taken, as these satisfy the appetite, providing bulk without much caloric value. Banting gave protein 170 gm., carbohydrates 80 gm., fat 8 gm. (total calories, 1100); this is not permissible when the blood urea is raised. Oertel's method reduces the fluid intake also, and reinforces the dietetic regime by carefully supervised hill-climbing. Sometimes the weight increases with retention of water in the tissues; in these cases more protein but no salt is allowed: and

mercurial diuretics help. Physical methods of treatment include general massage and Bergonié treatment, in which temporary reduction of weight is brought about by muscular movements produced by means of a faradic current. In order to stimulate metabolism, appropriate endocrine preparations can be given by mouth or by injection; the most useful is thyroid which, especially in myxœdema, can be gradually increased up to 3 to 5 gr. t.d.s. Pituitary extract may be useful, given by mouth, when combined with thyroid; anterior pituitary injections are of decided value where indicated. Dinitrophenol was previously used on account of its power of stimulating metabolism, but is now considered too dangerous. Septic foci, when present, should be dealt with. Weight reduction must be carefully supervised in unhealthy persons; once weight has been reduced to a satisfactory level, it will rise again if former habits are resumed; one or two starvation days a week often prevent this. In endocrine cases, the appropriate extracts have to be continued indefinitely in order to prevent relapse.

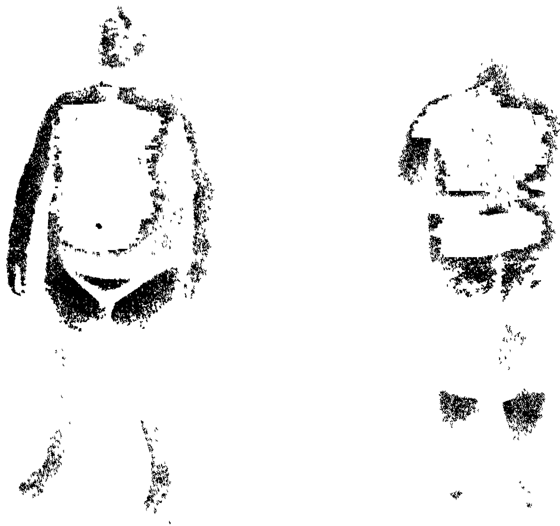


Fig. 7.

(Photographs taken by Mr. G. J. Potts.)

Fröhlich's syndrome. Boy aged 5; weight 5 st. 4 lbs.; has genital aplasia.

Adiposis Dolorosa is a rare disease described by Dercum, which occurs only in women, generally about the menopause. It is characterised by local subcutaneous and symmetrical deposition of a material probably mucoid in character, but the masses resemble lipomata. They are found chiefly over the deltoids, on the triceps, and the upper part of the body; and may occasionally spread downwards. In another form there is diffuse lipomatosis, only the hands and feet escaping. Pain is a constant symptom, due probably to pressure on the subcutaneous nerves. The administra-

tion of small doses of thyroid, massage, and plenty of open-air exercise have given good results, but treatment has often been disappointing. The disease is probably due to a defect in the pituitary. The condition described by Bowlby as *diffuse lipoma*, which occurs in alcoholic men, may be mistaken for Dercum's disease.

Fröhlich's syndrome is due to maldevelopment or to destruction of the posterior pituitary gland (Fig. 7). It is characterised by large deposits of fat in the subcutaneous tissue, chiefly the face and abdomen, and as it occurs in earlier life than adiposa dolorosa, the patients are asexual. It may be congenital or acquired. Pituitary and thyroid extracts do good if the case is seen in its early stage.

In *Cushing's syndrome* there is obesity of the face, neck and trunk, not the limbs, a considerable rise in blood pressure, hypertrichosis, polycythæmia with dusky skin; absence of sexual functions, glycosuria and other symptoms due to basophil adenoma of the anterior lobe of the pituitary may be present. A similar clinical condition has been found in association with *adrenal carcinoma* (§ 263).

The *Laurence-Moon-Biedl syndrome* shows obesity, retinitis pigmentosa, mental retardation and polydactylism. It is familial, but not hereditary, and due apparently to some defect in the mid-brain, involving the pituitary. Thyroid and pituitary extracts are helpful.

§ 19. **Dwarfism** means diminished stature only, and does not imply mental or sexual retardation. It may arise from any cause which affects the growth of the bones of the trunk or limbs, whether local or constitutional. The commonest causes of a stunted condition of the body, in order of frequency, are:

(i.) *Rickets*.—In this disease there is curving of the long bones, together with altered epiphyseal growth. This results in " bandy legs," " knock-knee," and other familiar deformities (§ 596). Varieties occur in the form of *renal* and *cœliac rickets*.

(ii.) *Hereditary Syphilis*, the means of recognising which are fully given in § 552.

(iii.) *Curvature of the Spine*, which may take three forms: (i.) *kyphosis* (*i.e.*, the convexity projecting backwards), usually due to postural defects, tuberculosis or other disease of the vertebræ, or to lax ligaments, as in rickets. The latter disappears when the child is held up by the shoulders. (ii.) *Lordosis* (*i.e.*, a forward projection), usually compensatory, or the result of muscular weakness; and (iii.) *scoliosis* (a lateral curve). All these may diminish the stature, but they differ considerably in importance. A certain amount of scoliosis is normal to nearly everyone, and the kyphosis of muscular weakness is common enough in old age, as a consequence of which our stature becomes slightly less in advancing years. Angular kyphosis is serious, as indicating organic disease of the bodies of the vertebræ.

(iv.) *Infantilism* is a condition of dwarfism, in which the usual changes, both sexual and physical, which normally occur at puberty, fail to take place, and the patient retains the stature, features, voice, and often the mental proclivities of a child. There are two main types: (*a*) those in which endocrine factors play a major part. Thyroid and pituitary deficiency are mainly responsible. Thyroid deficiency gives rise to cretinism. Deficient action of the anterior pituitary may follow an acute specific fever. *Ateliosis* may be of the same origin. It is hereditary; in the asexual form, there is delayed development of the whole body, but especially of the sexual organs; in another form improvement occurs at puberty, but the individual remains tiny. Under the name *progeria*, Hastings Gilford described a condition in which infantilism is associated with premature decay, the appearance, attitude and state of nutrition of the dwarf becoming senile, and degenerative changes occur in the vessels and viscera (§ 554). (*b*) Sometimes infantilism occurs with diabetes insipidus, polycystic kidneys, scleroderma, cirrhosis of the liver and spleen and with hydrocephalus. A toxæmic type of infantilism is described in which the development is arrested owing to chronic infection, such as tuberculosis, malaria, after scarlet fever, syphilis and cardiac disease, and recurrent diarrhœa in children (resembling cœliac disease), or it may be due to drugs, such as alcohol, tobacco, lead, mercury or morphia. In cases with pancreatic insufficiency, diarrhœa is often present and pancreatic extracts by mouth are of value. Thyroid medication is of major importance in the

endocrine group, and injection of gonadotrophic and other hormones of the anterior lobe of the pituitary, when injected, produce good results in experimental animals, and may benefit human beings. Even in the forms caused by toxæmia, thyroid should be given a trial.

(v.) *Achondroplasia.*—A rare condition somewhat resembling, and formerly confused with, Rickets (§ 598).

(vi.) *Osteomalacia,* when this disease involves the spine.

(vii.) *Cretinism* (§ 191) is a peculiar stunting of the growth which is either sporadic or is endemic among children in certain districts. The appearance is so distinctive that typical cases can be recognised at a distance (Fig. 8A). The face is broad and flat, and joined almost without a neck to the body. The skin and hair are coarse the hands broad and stumpy, the stature stunted, for even when twenty years of age a cretin may be only 3 feet high. It is due to a diminished action of the thyroid gland; recovery usually results and is maintained while thyroid extract is being given (Fig. 8B and C), but for the best results it must be started as early in life as possible.



FIG. 8.—Case of cretinism. A, aged 18 months. B and C after treatment, aged 38 and 66 months.

(viii.) Although *Mongolism* and *Microcephaly* (§§ 13, 907c) fall into this group on account of the lack of mental and physical growth and the sexual retardation, the changes produced make the subjects so unlike normal children that they are better described separately. *Mongolism* is a condition of defective development met chiefly in the children of older parents. It is differentiated from cretinism by the fine hair, clear complexion, liveliness of mannér, and broad head without an appreciable occipital

prominence, and absence of constipation. The name is derived from the resemblance to the Mongolian races. The eyes are oval and slant upwards at the outer angle, the little finger tends to curve inwards; there is often a squint, with various "stigmata of degeneration," and often a congenital heart.

(ix.) In addition to the foregoing there are certain rare conditions, of which the celebrated Tom Thumb and his wife, and the race of pygmies of Africa met with by Sir H. M. Stanley and others, are examples, in which the skeleton and the organs are diminished in size, but their proportions maintained. Such cases, however, seem to be functionally normal in every respect.

CHAPTER III

DISEASES OF THE HEART AND PERICARDIUM

§ 25. **Physiological Anatomy of the Heart.**—Before considering systematically cardio-vascular disease, it is advisable to review the more important points regarding the physiological anatomy of the heart. The heart is developed as a tube of muscle which becomes bent on itself and develops diverticula, forming the chambers, auricular and ventricular, of the adult heart. This tube in the course of development becomes modified, but remains can be distinguished in the fully formed heart, and are of importance as they form the specialised tissues whose functions are the initiation and conduction of the normal cardiac impulse. This specialised system is made up of: (1) Sino-auricular node (Pacemaker), which is situated between the superior and inferior venæ cavæ, and with which the extrinsic nerves are closely associated; (2) a series of intra-auricular paths which connect the sino-auricular node with a similar structure;—(3) auriculo-ventricular node. Passing down from the auriculo-ventricular node is a neuro-muscular strand—(4) The Bundle of His, which has a special nerve and blood supply, and forms the main connecting muscular link between auricles and ventricles. It lies just under the endocardium, under cover of the septal cusp of the tricuspid valve. The Bundle of His divides into—(5) A main right and left branch. The right branch, passing along the right side of the septum under the endocardium, runs along the moderator band and curves backwards to terminate chiefly in the base of the right ventricle and papillary muscles. The left branch passes along the left side of the septum, finally terminating in the wall of the left ventricle in close association with the cells of Purkinje. The different parts of this system are shown in the annexed diagram, Fig. 9.

The heart has an enormous reserve capacity; the maximum output during exertion is about ten times that produced by the resting heart. The increase in the work done is frequently so sudden that the organ is subjected to great strains as the result of sudden mechanical efforts or violent emotions. Injury is prevented by a series of protective mechanisms: the more important of which may be summarised as follows: (1) Nervous Mechanisms.—*The Vagus* has two main sets of fibres in relation to the heart: (*a*) efferent fibres with the power of slowing, weakening or even stopping the beat; (*b*) afferent fibres (depressor nerve), which run from the arch of the aorta to the vaso-motor centre in the medulla and convey stimuli which cause peripheral relaxation, lower the systemic blood pressure and so relieve the left heart. Overaction of the vagal mechanism may produce fainting attacks. The focal point of vagal cardiac inhibition is the *carotid sinus*. Direct pressure on it, over the right internal carotid, in some individuals slows the heart and lowers blood pressure to such an extent that it causes syncope. Such pressure may be digital, or from a stiff collar or other external object. More usually the stimulus is psychological, or the result of physical pain. Vaso-vagal attacks may be abrupt, or of more gradual onset. In the former case they must be distinguished from epilepsy, and in the latter from syncope caused by cerebral anæmia associated with vasomotor failure. Bradycardia and very low blood pressure are the signs of a vaso-vagal attack. *Sympathetic* stimulation produces tachycardia.

(2) Pain Mechanism.—In most organs of the body a pain mechanism exists, the primary object of which is protection. In the case of the normal heart, the pain mechanism is one of the last called into play. *Afferent impulses* conduct painful sensations from the heart through sympathetic fibres. These leave the heart, and pass to the cervical and upper dorsal sympathetic ganglia of the left side, whence the grey *rami communicantes* of the five upper dorsal segments pass the stimuli on to the spinal cord.

Heart sensations rarely become painful unless there is damage to the heart muscle or to the pericardium. Exercise cannot produce it in a healthy adult. This mechanism will be referred to again in connection with angina (§ 51).

(3) Myocardial Mechanisms.—(*a*) In the absence of anoxæmia or toxæmia the myocardium maintains its tone. Excessive exertion produces no dilatation when such exertion has finished. In the diseased heart, however, dilatation occurs. It is probable that under certain conditions the tricuspid and mitral rings may relax so that the valves may be rendered for the time being incompetent and the pressure in the ventricles relieved. (*b*) The Moderator Band is a special band



Fig. 9.—Diagrammatic section through the heart. S.A. = the sino-auricular node. A.V. = auriculo-ventricular node. The dotted lines joining S.A. and A.V. = intra-auricular paths, which convey the stimulus to the ventricles. Running down from the A.V. node is the main Bundle of His [Q] which divides into a main right branch [*r*] and a main left branch [*l*], the former terminates chiefly in the right papillary muscle and base of right ventricle, the latter in the wall of the left ventricle.

of muscle which crosses the cavity of the right ventricle and is reputed to prevent over-dilatation of this cavity; it contains the right branch of the Bundle of His.

(4) Pericardial Mechanisms.—The pericardium is a tough fibrous bag, the main function of which is to prevent over-distension of the auricles.

PART A. SYMPTOMATOLOGY.

The general symptoms of cardiac disease, as distinct from the local signs referable to the heart, should be studied very carefully, inasmuch as the gravity of any given case depends not so much on the local signs present as on the general condition of the patient.

In the investigation of a case of cardiac disease the various methods have roughly the following relative values: history 50%, physical examination 25%, electrocardiography 10%, radiography 10%, and pathological

and other special methods of diagnosis 5%. It is thus clear that the importance of an accurate evaluation of symptoms is very great.

The CARDINAL SYMPTOM of cardiac dysfunction is **Breathlessness.** When there is marked failure, **Dropsy, Venous Engorgement** and **Cyanosis** are also present. **Pain, Palpitation** and **Cough** are found in certain cases. **Fainting Attacks,** although in the lay mind invariably attributed to the heart, have in practice only rarely a cardiac cause. **Sleeplessness** and **delirium** occur in cases of failure. **Fever** and its concomitant symptoms occur in acute affections. **Unexpected** or **Sudden Death** may terminate cardiac disease.

§ **26. Breathlessness,** or Dyspnœa. Breathlessness may be present without cardiac disease; but it may be affirmed that no serious affection of the CARDIAC MUSCLE can exist without breathlessness. Dyspnœa is a physiological result of muscular exertion. It becomes pathological when evoked in excessive degree by an amount of exercise which previously had no such result. Two points to be elucidated are: (1) the amount of exercise now noticed to cause dyspnœa, and (2) the rapidity of development of the symptom. The severity of the myocardial failure and the progress of myocardial damage can in this way be estimated. The slightest degree of dyspnœa may be detected by observing that the scaleni and lower edges of the sterno-mastoids are brought into play at the end of inspiration.

Severe disease of the VALVES of the heart may exist for many years —provided the disability so caused is adequately compensated by muscular hypertrophy—without the patient having any noteworthy symptoms, or even being aware of its existence. When the heart muscle fails to compensate for the valvular defect, breathlessness appears. When the patient is unable to breathe on lying down, and the night is passed sitting upright in a chair, or propped up with pillows in bed, *orthopnœa* is present. It indicates pulmonary congestion and left-sided failure. It is absent in pure right-sided failure. This upright position relieves the embarrassed lung from the weight of the engorged liver, lowers the pressure in the venæ cavæ, assists the accessory muscles of respiration, and so reduces pulmonary congestion as far as possible. Towards the end in many cases of heart failure *Cheyne-Stokes' respiration* may be observed.

Sighing is frequently regarded as being due to organic heart disease, but this is not the case. It is suggestive of a cardio-vascular neurosis. It is frequently associated with vasomotor instability, with a labile heart rate and blood pressure, or may result from great nervous or bodily fatigue. The sighing is often long-drawn and occurs at frequent intervals.

OTHER CAUSES OF BREATHLESSNESS (DYSPNŒA).—Difficult breathing may arise in five different groups of disorders.

1. **Cardiac Disease.**—The dyspnœa of heart disease has no intrinsic features which distinguish it from that due to other causes, but it is often associated with cyanosis. There is also usually a history, or evidence, of some of the other symptoms of cardiac disorder. In cardiac

disease the amount of breathlessness present or the amount of exertion which can be taken without producing breathlessness are, of all symptoms, the most valuable indications as to the amount of inadequacy of the cardiac muscle (cardiac failure) present in any particular case (cf. § 43). If, in a dyspnœic patient, there is no cardiac enlargement, some cause other than heart disease must be sought.

2. **Embarrassment of the Heart by Neighbouring Structures,** such as mediastinal tumours, a large pleural effusion, ascites, a dilated stomach. Obesity may be a subsidiary cause of dyspnœa, but there is generally some associated myocardial change present.

3. **Laryngeal or Tracheal Obstruction.**

4. **Pulmonary Disease,** of which emphysema is the most common. In ACUTE PNEUMOTHORAX, dyspnœa of sudden onset is characteristic; pain in the side is usually present (§ 126).

5. **Blood Conditions.** Patients with a severe degree of ANÆMIA are often markedly dyspnœic; in addition they often have palpitation, hæmic murmurs, and œdema of the feet. Anæmic patients with dyspnœa prefer to lie flat; cardiac patients with dyspnœa prefer to be propped up. ACIDOSIS, due to diabetic ketosis or to uræmia, may cause dyspnœa, but this is present both at rest and on exercise.

Causes of Breathlessness which are apt to be overlooked.—The differentiation of the various forms of cardiac disease is given in the following pages; but, supposing *a patient over thirty-five or forty,* who complains of breathlessness, presents no definite signs of cardiac or pulmonary disease, nor any evidences of dyspepsia or anæmia, then there are certain conditions which should be *suspected:*

1. **Myocardial Degeneration,** often secondary to disease of the coronary arteries. The sounds and impulse may be feeble, and the other signs mentioned in § 57 may be present.

2. **Syphilitic Aortitis** with or without disease of the aortic valves.

3. Deep-seated **Aneurysm of the Aorta** and other **Intrathoracic Tumours** may give rise to the breathlessness and general symptoms of heart disease without the physical signs. In such cases the dyspnœa may be paroxysmal, and in neoplasm is often very severe.

In *a patient under thirty-five or forty* the three following causes of UNEXPLAINED BREATHLESSNESS may be suspected:

4. **Latent Pulmonary Disease,** and especially latent pulmonary tuberculosis, should always be suspected in cases of breathlessness without obvious cause.

5. **Pericardial Effusion,** also, is often attended by relatively few physical signs (§ 56).

6. When severe dyspnœa sets in suddenly in the course of cardiac or **Acute Renal Disease,** the chest should always be carefully examined, because hydrothorax may set in rapidly without any general dropsy or other warning symptom.

§ 27. Paroxysmal Dyspnœa is that form of dyspnœa which occurs in attacks from time to time. It is apt, as above mentioned, to occur in some cases of cardiac disease, especially in the last stages of myocardial degeneration, and in any given case attention should first be directed to the heart. But there are several other conditions which one would suspect in a patient in whom the chief or only symptom consists of paroxysms of breathlessness. If the paroxysms are cardiac in origin the heart is always enlarged; in the other conditions there is no cardiac enlargement, provided that there has been no hypertension.

1. Paroxysms of dyspnœa causing the patient to wake up at night are often one of the first symptoms of CHRONIC NEPHRITIS, and are spoken of by the patient as asthma; they are typical of cardio-renal failure (§ 372).

2. In ASTHMA, laryngismus stridulus, and whooping-cough, the attacks of breathlessness are typically paroxysmal.

3. NEUROTIC DYSPNŒA.—Some neurotic patients are liable to attacks of rapid respiration. These usually cease when the patient converses or thinks that he is not being observed, or during sleep. If prolonged, tingling in the fingers and other evidences of tetany are present (§ 778). The heart rate may be rapid in these attacks.

4. ANEURYSM and other INTRATHORACIC TUMOURS may give rise to paroxysmal dyspnœa before other signs can be made out.

5. ACUTE PULMONARY ŒDEMA (§ 118).

6. Enlargement of the THYMUS GLAND, whether due to neoplasm or to the condition known as LYMPHATISM, or status lymphaticus, in which there is general hyperplasia of lymphatic structure, associated with a persistent thymus, may cause paroxysmal dyspnœa, to which the name "thymic asthma" has been given (§ 37).

7. FOREIGN BODIES in the trachea and retropharyngeal abscess in children, and polypi or papillomata of the larynx, give rise to paroxysms of dyspnœa.

8. Sudden dyspnœa, coming on during vomiting, is the main indication of that rare accident, RUPTURE OF THE ŒSOPHAGUS. This dyspnœa is due to pneumothorax.

9. The LARYNGEAL CRISES of tabes dorsalis may take the form of paroxysmal dyspnœa.

§ 28. **Cheyne-Stokes' Respiration** (so called after its first observers) consists, in its typical form, of a series of eight or ten rapid inspirations gradually increasing in depth and rapidity, and then dying gradually away, each series being separated by a pause of some seconds (the stage of apnœa), in which there is little or no respiratory movement. It is due to lack of CO_2 in the blood and can be abolished by giving the patient inhalations of O_2 with 5 per cent. of CO_2. The hyperpnœic stage may produce such exaggerated movements as to wake the patient and cause a sensation of acute discomfort.

In a modified form, without the apnœic pause, Cheyne-Stokes' breathing is not infrequent. It is usually a serious symptom, and appears in cardiac patients *towards the end of life*. It has less significance at the extremes of life, for it may be observed during sleep in normal infants, and is occasionally compatible with a hale old age.

Its principal *causes* are as follows:

1. CEREBRAL ARTERIO-SCLEROSIS; 2. CARDIAC DISEASE due to coronary atheroma; 3. URÆMIA; 4. Rapidly increased INTRACRANIAL PRESSURE such as occurs with apoplexy, tuberculous meningitis, and in some cases of cerebral tumour; 5. SUNSTROKE.

Prognosis.—In the presence of organic heart disease, especially if there is also hyperpiesis, the expectation of life is reduced to two or three years.

Treatment.—The best methods of treatment are continuous oxygen administration, or in some cases the use of morphia, gr. $\frac{1}{4}$ by injection. The writer has found a course of daily intravenous injections of cardophylin (euphyllin) useful, in doses of 0·48 G. in 20 c.c., for a period of 7–10 days.

§ 29. **Dropsy** is a chronic effusion of fluid into the skin and subcutaneous tissues (when it is known as anasarca or œdema) or into a serous cavity (as in hydrothorax, hydropericardium, ascites). The former, **Anasarca,** is the variety of dropsy we are now concerned with, for it is a very constant feature of some forms of cardiac disease. General anasarca has to be differentiated from myxœdema, in which the swelling is harder, and does not pit on pressure. It is best to apply the pressure over a bone, such as the lower end of the tibia, on its inner aspect.

Causes.—The causes of localised dropsies are given in Diseases of the Extremities (§ 570). There are *three varieties of general anasarca,* which differ from each other both pathologically in their origin, and clinically in the course they pursue.

1. **Cardiac Dropsy** is partly due to the raised intracapillary pressure resulting from the venous engorgement of right-sided heart failure, and partly to the malnutrition of the capillary endothelium resulting from the slowed peripheral circulation. (1) It *starts,* and throughout the case predominates, in the *most dependent parts,* that is to say, in the legs if the patient has been walking about, or in the lower part of the back if he has been lying in bed. On inquiry, the patient may complain that the ankles swell towards evening around the top of the boot. (2) Other signs and symptoms of cardiac enfeeblement or dilatation are present. (3) In the history of the case dyspnœa will have *preceded* the dropsy. Dropsy does not occur with equal frequency in all forms of cardiac disease. The œdema which complicates pulmonary disease has the same features as cardiac œdema, because it is the resulting right ventricular failure which produces it: but here pulmonary congestion and orthopnœa are absent. Dropsy, in the absence of dyspnœa or of cardiac enlargement, is not due to heart disease; some other cause, such as phlebitis or obstruction to the vena cava, must be sought. Dropsy is often present in renal disease without dyspnœa; it is then usually a general œdema.

2. **Hepatic Dropsy** (1) usually begins and predominates *in the abdomen* (ascites), although the legs may swell subsequently by reason of the pressure of the fluid on the veins within the abdominal cavity. (2) There may be also enlargement or other signs of the liver affection which has given rise to the condition; and if these be absent some other cause of obstruction to the portal vein should be sought (§ 260). (3) The dyspnœa will have *followed* the abdominal enlargement.

3. **Renal Dropsy** is (1) *general in its distribution* from the beginning, occurring in the legs and eyelids at the same time; though it is probable that the œdema around the eyes on rising in the morning first attracts the attention of the patient or his friends. (2) Examination of the urine reveals the features of renal disease, but it should be remembered that some degree of albuminuria is common in heart failure. The presence of many casts is strong evidence of a renal origin. (3) The patient presents a characteristic pale or waxy appearance. In some cases of general anasarca associated with albuminuria the question arises whether the dropsy is renal or cardiac. This may sometimes be answered by finding the liver enlarged, for this is a natural sequence of right-sided heart failure, though not of renal disease. The frequent association of chronic renal disease and cirrhosis of the liver must not be forgotten.

Prognosis.—Dropsy usually indicates a severe degree and a late stage of heart disease. The outlook varies greatly, according to the cause.

Treatment.—The principles are as follows: Absolute rest in bed; raise the limbs and keep the patient warm. Reduce the fluid intake to

30 to 40 oz. daily; give small, dry, palatable, non-fermenting diet with a reduced sodium chloride content; also give digitalis and some diuretic—such as mersalyl ½ to 2 c.c. intramuscularly or intravenously, preceded for one day and accompanied by the administration of ammonium chloride gr. 15 t.d.s., p.c. (in capsules). Other diuretics to be tried are theobromine and sodium salicylate (diuretin) gr. 15 t.d.s., p.c.; theophyllin and sodium acetate gr. 2 to 5 t.d.s., p.c., and urea gr. 120 t.d.s., in lemon juice and water. If the dropsy in the limbs is extensive, wrap them in cyanide gauze or some other dressing, as they are liable to eczema, erythema, cellulitis or exfoliative dermatitis. Should the above methods fail, multiple small punctures with needles or with small incisions through penicillin cream, or the insertion of Southey's tubes under strictly aseptic methods may be practised. The patient's legs should have been dependent for several days to allow the fluid to accumulate. The abdomen or pleural cavity may require tapping. It is never wise in cardiac œdema to allow fluid to accumulate in the pleural cavities.

Obscure Causes of General Anasarca.—If, in a patient who complains of dropsy, no marked evidences of cardiac, renal, or hepatic disease are discoverable, the following causes may be *suspected*:

1. In women with **poor muscular tone**, but otherwise normal, œdema of the legs and feet is found. It is common in multiparæ and the left leg is often the more severely affected. It is especially marked after a preceding phlebitis. These patients generally have the symptom for many years, especially in hot weather; no marked dyspnœa and no cardiac enlargement are found.

2. **Anæmia** is not infrequently attended by some swelling of the ankles at the end of the day. Swelling of the feet and ankles may be present in the last stages of many exhausting diseases, such as phthisis, in septic and anæmic states, and in cases of insufficient nutrition and old age. 3. Among the causes of dropsy rare in this country are **Beri-Beri** (§ 795) and **Epidemic Dropsy.** Epidemic dropsy occurs in sporadic outbreaks amongst rice-eaters and those suffering from Vitamin B deficiency. This "nutritional" œdema has several causative factors, all secondary to the avitaminosis; they are myocardial failure, lowered serum albumen, and possibly an excessive (for the patient) intake of fluids and sodium. 4. Milroy first described a hereditary œdema in which a solid œdema of the legs existed in many members of a family (§ 570). 5. **Congenital general œdema** (hydrops fœtalis, § 551 V) is usually fatal.

Venous Engorgement. In an advanced stage of heart failure the veins are continuously distended. This is visible chiefly in the veins of the neck and is increased during systole. The level below which the neck veins remain distended in a normal individual is the lower border of the manubrium sterni. In heart failure the veins remain swollen to a higher level according to the amount of increase in the pressure. If the neck veins are in a state of distension and there is no dyspnœa nor cardiac enlargement, the cause is to be sought in some lesion producing intrathoracic venous obstruction, such as mediastinal tumour, aneurysm or constrictive pericarditis.

§ **30. Cyanosis,** or bluish discoloration of the body surface, is due to an abnormal amount of reduced hæmoglobin in the peripheral capillary blood. This may be the result of (1) slowing of the peripheral circulation,

(2) slowing of the general circulation, (3) insufficient aeration of the blood in the lungs, (4) admixture of venous with arterial blood in congenital heart disease, (5) abnormal blood conditions. When the blood stream is slowed, more oxygen is taken from the blood by the tissues. *General* cyanosis affects also the mucous surfaces. *Local* cyanosis, as in Raynaud's disease, may be differentiated from general cyanosis, as in heart failure, by immersing the patient's hand in hot water for 10 minutes; in the former case the skin colour becomes pink, in the latter it remains blue.

Cardiac cyanosis is most pronounced in heart failure secondary to chronic pulmonary disease, or to mitral stenosis. It is found in congenital heart disease with pulmonary stenosis, or with gross auricular or ventricular septal defect. **Respiratory** causes of cyanosis are anatomically diffuse, such as emphysema, bronchitis, asthma, pulmonary œdema, pneumonia, miliary tuberculosis and pleurisy with effusion.

When there is polycythæmia, as in Vaquez' disease, the increased viscosity of the blood produces a slowing of the peripheral circulation and therefore some cyanosis.

Local cyanosis may be unilateral, as for example when an intrathoracic tumour is pressing upon the venous return of one arm. A venous thrombosis produces a similar effect. And see § 576.

Treatment of cyanosis.—The treatment of cyanosis of pulmonary origin depends on the cause. Oxygen, given by a B.L.B. or other type of mask, intra-nasal catheter, or oxygen tent or chamber, is most effective in emphysema, bronchitis, asthma, pulmonary œdema and pneumonia. Five per cent. CO_2 should be used with the oxygen, either when the breathing is shallow or when there is pulmonary collapse. Oxygen given by funnel is useless in every type of disease. Paracentesis of a pleural effusion always helps cardiac anoxæmia and cyanosis. In the absence of pulmonary œdema or bronchitis, oxygen is not of much use in heart failure, for the slowed circulation rate allows more, not less, time for the pulmonary capillary blood to be fully oxygenated. Heart failure with cyanosis is often benefited by rapid venesection and removal of 10 to 20 oz. of blood, the particular indication for this being distension of the veins in the neck.

§ 31. **Polycythæmia Vera** (Synonyms: Vaquez' disease, Erythræmia, Splenomegalic polycythæmia).—This is a disease in which there is an overgrowth of the red cell forming tissue in the bone marrow. (i.) The patients are usually middle-aged and complain of headache, vertigo and other nervous symptoms, pains in the limbs and dyspnœa. (ii.) They are easily recognised by the redness of their complexion, which often deepens to cyanosis, especially in cold weather. All the superficial vessels are dilated. (iii.) The spleen is enlarged to a variable extent. (iv.) The blood shows a marked increase in the red cells up to 13,000,000 per c.mm. with 120–160% hæmoglobin and a colour index of 0·7–0·9. Polychromasia and a few normoblasts are usually present and the platelets and white cells are increased: myelocytes may be present. Owing to the relative increase in the number of red cells, the viscosity of the blood is raised and also the blood volume. (v.) Hæmorrhage may occur from the distended vessels at any site. A variety, **Gaisbock's disease** or *polycythæmia hypertonica*, is described without enlarged spleen, but with high blood pressure and arterio-sclerosis. It follows a chronic course, with death, sometimes after many years, from heart failure, cerebral hæmorrhage or thrombosis. In **Ayerza's**

disease the polycythæmia is secondary to an obliterative endarteritis of the pulmonary artery. It is associated with extreme dyspnœa, a normal or low blood pressure, no splenic enlargement, but a dilated conus of the pulmonary artery seen radiographically.

Treatment may be (*a*) by removing excess blood with repeated venesection—this is the safest method; (*b*) blood may be destroyed by a hæmolytic poison, *e.g.*, acetylphenylhydrazine gr. ½ t.d.s. for 7–10 days, followed by a rest, as it is a cumulative poison; (*c*) erythropoiesis may be depressed by radiation to the bone marrow or by internal radiation using intravenous radiophosphorus—give 3–8 millicuries initially, followed by 1–5 millicuries each three to six months so long as the red cell count remains above six million. Treatment must be controlled by blood counts. It is essential to establish a correct diagnosis, for if the polycythæmia is secondary to pulmonary or cardiac disease, treatment with drugs is harmful.

§ 32. Rare causes of cyanosis are: **Sulph-hæmoglobinæmia** and **Methæmoglobinæmia.** The most prominent symptoms are (1) cyanosis of a peculiar greyish leaden colour; (2) marked weakness, vague pains and collapse; (3) constipation, sometimes alternating with offensive diarrhœa and most marked in sulph-hæmoglobinæmia; (4) periods of relative freedom followed by exacerbations. Two factors appear to be necessary for the formation of these compounds: (i.) some activating substance in the blood, and (ii.) absorption of sulphur or reducing substances from the bowel. It has been shown that sulphonamide compounds, metadinitrobenzene. trional, sulphonal, pamaquin, potassium chlorate, acetanilide, phenacetin and related compounds and certain aniline dyes, can act as such sensitising agents, and a history of taking these drugs can usually be obtained. Certain nitroso-bacilli, which have the power of reducing nitrogen compounds, have been isolated from the saliva and bowel, and are probably causal in those cases without a drug history (*enterogenous cyanosis*). Magnesium sulphate and other saline cathartics predispose by increasing the fluid content of the bowel, and hence bacterial fermentation. The *diagnosis* is based on the history and cyanosis without a cardiac or respiratory cause; it can be verified by spectroscopic examination of the blood (Plate IV). *Prognosis.*—This condition is not fatal, but may prove very resistant. In *treatment*, any possible sensitising drug must be excluded, and it is advisable to restrict sulphur in the diet; the constipation must be relieved by liquid paraffin or enemata. In severe cases, inhalations 2–3 times a day of 5% carbon dioxide in oxygen may be given. In cases due to nitroso-bacilli a vaccine of these organisms may be of value. The cyanosis of methæmoglobin usually disappears within 48 hours of ceasing to take the drug, whilst that of sulph-hæmoglobin may persist for very much longer. Methylene blue helps to relieve cyanosis: it is given in doses of gr. 1–2 t.d.s. by mouth, or 1–2 mgm. per kilo intravenously. Ascorbic acid is effective in cases of familial methæmoglobinæmia.

A **Sallow Hue** of the skin is common in infective endocarditis. This sallowness is distinguished from jaundice by the absence of the yellow colour from the eyeballs and the absence of bile in the urine. True jaundice, however, does arise in cardiac disease, as a result of the hepatic congestion of severe cardiac failure.

Clubbing of the fingers is found in heart disease in the following conditions. (1) Congenital pulmonary stenosis is nearly always accompanied by clubbing. (2) Clubbing is found in malignant endocarditis. (3) Rarely, clubbing is found in cases of chronic rheumatic carditis. (4) An aneurysm pressing on or affecting the flow in one subclavian artery may produce clubbing of the affected side (and see § 568).

§ 33. **Pain in the Chest** is absent in most forms of heart disease. It is present in coronary disease, less frequently in aortic insufficiency, and

occasionally in mitral stenosis. Pain due to heart disease is usually proportional to the amount of exercise the patient is taking: it is generally substernal, sometimes præcordial in position and affects the upper rather than the lower part of the chest. It may radiate to either arm or to both, or to the neck or jaw (§ 51). Many cases of neuro-circulatory asthenia (effort syndrome) suffer from pain which is often submammary; hyperæsthesia of the præcordium suggests a functional rather than an organic lesion.

The causes of præcordial or cardiac pain are:

(*a*) Arising from **Organic affections outside the heart and pericardium.** Intercostal fibrositis or "rheumatism," often known as intercostal neuralgia, especially that which precedes or follows herpes zoster; pleurodynia; neoplasms; pleurisy and pneumothorax (§ 103); spinal caries and carcinoma of the vertebræ, and tumours eroding the bones; the crises of tabes dorsalis; aneurysm. Muscular thoracic pain is increased by coughing or other muscular effort, and abolished by injection of a local anæsthetic; pleural pain is worse on breathing; herpetic pain is constant, and the pain of neoplasm is often worse at night.

(*b*) The pain may be of **Cardiac origin,** in which case it is usually called Angina (§ 51). This is a loose term and refers to three separate clinical conditions: (1) CORONARY THROMBOSIS (§ 52) produces an anginal pain, often of great severity. The onset is rapid, and the pain persists, with or without remission, for a period of three to six days, during which time its intensity slowly subsides. (2) ANGINA OF EFFORT is characterised by an anginal pain which comes on during exertion, disappearing again when exertion ceases; its intensity is directly proportional to the amount of exercise taken. (3) SPASMODIC ANGINA leads to paroxysmal attacks of very severe anginal pain, induced by exercise, emotion or cold, and relieved by nitrites; the pain is not proportional to exercise, and does not begin to subside directly exercise is stopped; angina of effort nearly always co-exists. These forms of pain are probably due to an interference of blood flow through the coronary circulation. The interference in coronary thrombosis is permanent; that in angina of effort is partial, but is relatively increased when the heart activity is increased by exercise. The mechanism of an attack of spasmodic angina is not yet understood (see § 51).

(*c*) Pain of a cardiac type is also found in **neuro-circulatory asthenia,** effort syndrome, or disordered action of the heart (§§ 34, 53). The pain may be little more than a dull ache, when the term left submammary pain is often used, but it is sometimes acute and radiates to the left arm, thus simulating severe angina of effort or spasmodic angina. In such cases a careful history will reveal that it is not quantitative to exertion, is left-sided rather than central, and is closely related to fatigue or to emotion. The term angina innocens (§ 53) is a useful stimulus to correct diagnosis. In some cases intercostal or subscapular fibrositis seems to be a causative factor, but heart consciousness or fear are often the reasons for localisation of the pain to the præcordium. Sharp sudden stabs of

pain sometimes occur in these cases, and accentuate the dull left-sided ache: syncopal attacks often follow these sharp stabs of pain. Organic heart disease is never indicated by pain of this type.

In cases of unexplained pain in the chest, and in the absence of cardiac signs, *mediastinal tumour* or *aneurysm of the aorta*, either of the arch or of the descending aorta, should always be suspected, and an X-ray examination made (§§ 80, 81).

Disease of the heart may also be an indirect cause of pain elsewhere than in the præcordium. For instance, with the engorged tender liver which is commonly associated with failure of the right auricle and ventricle, the muscles and skin of the abdominal wall are often also tender. In coronary thrombosis and in acute pericarditis the pain may be referred entirely to the upper abdomen. A simple cause of epigastric tenderness which must never be forgotten in cases of heart and lung disease is muscular strain of the upper rectus muscle and diaphragm from the exertion of constant cough. Pain in the shoulder, arm, neck and jaw is common in disease of the first part of the aorta; it is usually accompanied by superficial tenderness.

In the *treatment* of præcordial pain an endeavour should be made to ascertain and relieve the cause.

§ 34. **Palpitation** is consciousness of the heart's action. It arises under two sets of conditions, non-cardiac and cardiac. The essential symptomatic difference between the two types of palpitation is that in the *cardiac* group the onset is felt to be absolutely abrupt, as also in many cases is the termination of the paroxysm. The *non-cardiac* group is the larger and less serious; it includes:—

1. In **Anæmia** the palpitation is a frequent and often distressing feature.

2. **Dyspepsia** is a more common cause of palpitation than is cardiac disease. In such cases it often comes on at night, especially after a heavy meal. It may, in these circumstances, be accompanied by morbid dreads —*e.g.*, of impending death—by breathlessness, "night starts," cardiac pain, and other cardiac symptoms.

3. Certain **Local Conditions**, such as thoracic or abdominal tumour, or dilated stomach, which hamper the heart's action, may produce palpitation, although the heart be healthy.

4. In **Graves' Disease** (exophthalmic goitre) violent palpitation and increased rate of the heart are prominent features. In quite a number of cases this and the other nervous symptoms of the disorder exist for months before the two diagnostic features—thyroid enlargement and exophthalmos—become obvious. Graves' disease should always be suspected in cases of persistent palpitation (§ 186). In the type of thyrotoxicosis known as toxic adenoma, cardiac disease with congestive failure and auricular fibrillation may exist without exophthalmos, and the true cause may thus escape notice (§ 190).

5. Early stages of **pulmonary tuberculosis** (§ 131).

6. **Nervous** conditions, such as fright, fear, or other emotion, especially after an exhausting illness. It also occurs in hysteria and anxiety neurosis.

7. **Effort syndrome,** neurocirculatory asthenia, Da Costa's syndrome, cardiac neurosis, are all terms descriptive of a condition seen frequently in civilian life, but rising into prominence in times of war. The symptoms

are largely cardio-vascular—extreme lassitude and fatigue, dyspnœa on slight exertion, palpitation, præcordial discomfort, submammary ache and angina innocens, are the commonest cardiac manifestations. In addition, vasomotor and psychological abnormalities are often present. Undue sweating, especially of axillæ and hands, tachycardia and lowered blood-pressure on standing upright, fainting attacks and postural dizziness, are common. Psychologically there may be anxiety or hysteria, but far more frequently there is an idiosyncrasy of character rather than a neurosis, the individual being of the shy, hypersensitive, introspective type, who has avoided as far as possible situations in childhood or in adult life involving friction and physical or mental exposure and stress.

Treatment consists first in the thorough exclusion of all physical abnormalities. This is followed by explanation to the patient of the causes of his condition, and then by progressive exercises and interesting occupations, often while resident in a special treatment centre.

8. The excessive use of certain **Drugs** or **Articles of Diet**, notably tobacco, tea, coffee, and alcohol.

Cardiac causes include: (1) gross cardiac lesions; (2) auricular flutter; (3) auricular fibrillation; and (4) paroxysmal tachycardia. These conditions are dealt with in Section C (§§ 63 *et seq.*).

Cough is a symptom which belongs chiefly to diseases of the lungs (§ 101), but it is met with in diseases of the cardio-vascular system in two circumstances. (*a*) Firstly, the lungs are very often involved in left-sided failure; mitral stenosis produces a chronic pulmonary congestion, and coronary disease with left ventricular failure causes an acute or a chronic pulmonary congestion or œdema. The acute form is known as acute pulmonary œdema; this produces a sudden attack of severe cough, with copious, frothy, albuminous sputum, which may be pink (§ 118). (*b*) Secondly, from pressure. When an aortic aneurysm presses on the recurrent laryngeal nerve, a peculiar dry, brassy cough is present. Pericarditis or an enlarged left auricle in mitral stenosis may produce cough. Cough after effort may indicate heart failure.

§ 35. In **Syncope** there is transient loss of consciousness, due to anæmia of the brain. It is often preceded by giddiness, nausea, and a feeling of faintness. The face is ashy pale and the pulse and respiration feeble. Its advent is usually sudden, but recovery, after the attack has lasted some minutes, is gradual. Syncope is rarely caused by organic heart disease. With rare exceptions *patients with heart disease do not faint.*

Diagnosis.—Syncope has to be distinguished from *epilepsy* (§ 718*a*). (1) Epilepsy is sometimes preceded by an aura, though this is evident to the patient only. Its advent is more sudden than syncope, the duration of the attack is shorter, and the return to consciousness equally sudden and complete. (2) Syncope is rare without some definite determining cause, although it may be of a trivial nature—such as a heated room, or the sight of blood. *Aural vertigo* may be mistaken for syncope; for differential features, see § 692.

Causes.—(1) Deficiency of blood, *e.g.*, hæmorrhage. (2) Vasomotor instability is seen in the common form of faint in which the abdominal vessels suddenly lose their "tone," dilate, and retain blood which is needed elsewhere. (3) Fainting is often due to vasovagal attacks (Lewis), in which as a result of disturbance of the carotid sinus or of the depressor nerve, vagal slowing of the heart is produced, which must not be confused with the type described by Gowers (§ 720). The onset is usually gradual, sometimes sudden. The patient sweats, loses consciousness, possibly as a result of some fright or other emotional disturbance; the heart rate is slowed and the blood pressure falls. The heart is not diseased, and the prognosis is good. (4) Senile syncope gives rise to attacks, preceded by giddiness, in old people who are the subjects of arterial degeneration (see § 719).

The **Vasomotor** group is the largest. The "faints" occur chiefly in the upright position and in young, anæmic, and nervous females and in boys at puberty; who, when exposed to grief, bereavement, or any sudden emotion, or too hot rooms full of vitiated air, develop the familiar "fainting attack." (See also postural hypotension, § 88.)

Predisposing causes are:—(1) Anæmia, debility, hunger, or starvation; (2) diminished resistance in the peripheral and splanchnic arteries, such as occurs with excessive heat, as in hot rooms or Turkish baths; (3) sudden assumption of the erect posture, as in jumping from bed, may produce syncope; (4) sometimes, in addition to the preceding, the splanchnic veins are suddenly dilated when the intra-abdominal pressure is rapidly lowered, as by emptying the bladder or by rapid paracentesis, and this leads to anæmia of the brain and syncope.

Cardiac causes.—Certain cases of aortic incompetence or stenosis, Stokes Adams' attacks in heart block, rare cases of auricular flutter, or paroxysmal auricular fibrillation. In aortic stenosis there may be a direct relationship between exercise and fainting.

Prognosis.—Syncope in the young is usually not organic in origin, whereas in the aged it is generally a proof of cardio-vascular degeneration. In the former, therefore, it is usually as trivial as in the latter it is serious —the gravity depending upon the nature of the lesion.

Treatment.—Place the patient immediately in a horizontal position with the head low. This may be most readily done on the floor, but if there is little space, instruct the patient to bend forward and lower the head between the knees. Apply ammonia to the nostrils, throw cold water on the face. If recovery does not promptly take place, and the pulse be feeble, a hypodermic injection of leptazol or nikethamide B.P. (coramine) may be resorted to. For further treatment, see Collapse (§ 239). The underlying cause must be carefully sought and treated when the patient has recovered from the urgent syncopal condition.

Sleeplessness is a distressing symptom of severe heart failure, and is due to slowing of the cerebral circulation. Morphia has no deleterious effect upon the heart, but it should only be used for short periods of time owing to the danger of habit formation; it is contra-indicated where there is much bronchitis. Paraldehyde in full doses, soluble barbitone

B.P. (medinal), hexobarbitone B.P. (evipan) and phenobarbitone are useful. Oxygen intranasally is helpful when pulmonary œdema co-exists.

Delirium, generally worse at night, is a more severe result of the same cerebral anoxæmia. Should the remedies above mentioned fail, hyoscine hydrobromide gr. 1/100 by subcutaneous injection may be necessary, and can be repeated after 4 hours.

Pyrexia and its concomitant symptoms (see Chapter XV) are present in many *acute disorders* of the heart and pericardium. The temperature in malignant endocarditis is usually of an intermittent or remittent type, with an irregular range, as in other forms of septicæmia.

§ **36. Sudden** or **Unexpected Death** is not a common occurrence in patients with heart disease.

1. In *aortic valve disease*, stenosis more often than regurgitation, death may suddenly supervene during apparently good health.

2. In *acute coronary infarction* or after it has healed, in *myocardial degeneration*, in *syphilitic aortitis* involving the coronary orifices, unexpected death may happen, the mechanism here being the onset of ventricular fibrillation, which condition is incompatible with life.

3. *Pulmonary embolism* from previous phlebitis, *fat embolism* from skeletal trauma and *air embolism* from thoracic paracentesis, may cause sudden death.

4. Nerve diseases which in their progress involve the *medulla* terminate suddenly; and thus, among the rarer causes, atlanto-axoid disease and syringomyelia may be mentioned.

5. Emotional shock, injuries to the head, and other conditions acting on the *nervous system* by shock (§ 239).

6. *Hæmorrhage* into a previously silent *cerebral* or *pontine tumour* may also cause this.

7. *Poisons.*—Prussic acid acts very rapidly; others acting less quickly are cocaine, carbolic, volatile and non-volatile narcotics and anæsthetics.

8. Sudden rupture of a large cyst, an internal organ, acute disease of the suprarenals, or other cause of *Surgical shock* (§ 239).

9. Foreign bodies in the trachea, or other causes suddenly stopping the respiration (*asphyxia*)—*e.g.*, reflex apnœa from irritation of the pleura (pleural shock).

10. Status lymphaticus (§ 37).

§ **37. Status Lymphaticus** (*Lymphatism*) is a rare condition frequently unrecognised during life, but it is a cause of sudden death in children and young adults. There is overgrowth of the thymus gland and of the lymphatic tissues throughout the body. There may be no symptoms, the first evidence of the existence of the condition being death after a trivial shock, such as a plunge into a cold bath, a hypodermic injection, or the first touch of the knife in a minor surgical operation. The patient is flabby and pale and the physical signs are indefinite, consisting only of hypertrophied tonsils and adenoids. In other cases the enlarged thymus causes dulness beneath the upper part of the sternum, the spleen is palpable, and there may be overgrowth of adenoid tissue at the base of the tongue. Subjects of this diathesis must be guarded against anæsthetics, sudden shocks, or exertion. X-ray application to the thymus is the treatment of choice and appears to be successful.

PART B. PHYSICAL EXAMINATION.

§ **38. Landmarks of the Chest.**—There is a *ridge* on the sternum between the manubrium and the gladiolus; it can always be felt opposite the second costal cartilage; and the other ribs can be counted from the second one. The *nipple* is usually situated just external to the fourth costal cartilage, near its junction with the rib. At the back, the *lower angle* of the scapula just covers the seventh rib; and the *scapular line* is a vertical line drawn through the inferior angle of the scapula. The position and relations of the heart can be studied in Fig. 11, which is a sketch taken from the cadaver.

Inspection.—First inspect the patient from the foot of the bed. In a cardiac case the bed-ridden patient is almost invariably propped up. The appearance is often characteristic: The throbbing neck vessels of aortic regurgitation; the malar flush of mitral stenosis; the undergrown body and reddish-blue appearance of congenital pulmonary stenosis; the pinched patchy face, with the tortuous temporal arteries, and the often wasted body typical of cardio-vascular degeneration; the sallow toxic, anxious face of infective endocarditis; the large white face of renal disease; the blue face seen in congenital heart or failing mitral disease; the apprehensive look of the patient with angina, or the pale, puffy face of pericarditis, are all characteristic.

Other points to look for are the respiratory rate, depth and rhythm: cyanosis: engorgement, pulsation or otherwise of the jugulars: presence or absence of carotid pulsation: epigastric pulsation: thyroid enlargement: clubbing of the fingers and, if present, whether the fingers are blue (congenital heart disease) or white (infective endocarditis). Œdema around the ankles should be noted.

The abdomen should be examined for distension, and the importance of this sign, with its serious cardiac embarrassments, should not be underestimated. The presence of engorged veins, diminished respiratory movement, and the presence of ascites should all be observed.

Should the patient be confined to bed, attention should be directed to the position in which he lies or which he assumes. When the chest has been exposed, its shape and movements, any bulging of the præcordium (cardiac disease in early life before the chest has ceased growing) should be noted; also the cardiac impulse, its position and character, special attention being directed towards whether it is heaving (the true sign of cardiac hypertrophy) or slapping and diffuse in character. Systolic recession, indicative of adherent pericardium, should be looked for, not only in the region round the apex, but in the region of the epigastrium and also in the back (Broadbent's sign).

§ **39. Palpation and the Localisation of the Apex** (see Figs. 11 and 12).—The apex beat is the point farthest downwards and to the left at which the cardiac impulse is distinctly felt. After inspection it should be first palpated by the flat of the hand, and then localised with the finger tips.

In an adult male it is normally situated in the fifth interspace ½ inch to the inner side of the mid-clavicular line, at a distance of about 3 inches from the mid-sternal line. *These and other cardiac measurements vary with the age*[1] *and proportions of the patient*—a fact which is apt to be forgotten. The *most external* portion of the apex beat should be marked by a dot with an aniline pencil. At the level of the apex, measure and note the distance from the mid-line to the apex; measure also the distance from the middle of the neck to the middle of the left clavicle. In health these measurements should be the same, or at least the apex should not lie to the left of the mid-clavicular line. Thus at the first examination the position of the apex can be accurately defined, in terms of the mid-clavicular line. Further measurements need only be made from the mid-line, provided the patient does not grow. The principal features to observe about the apex are—its POSITION, CHARACTER and FREQUENCY. The beat of the left ventricle is felt as a forward thrust, if the myocardium is healthy; that of the right is less well defined. It is important to bear in mind that the apex beat is considerably modified if the apex happens (as is not infrequent) to pulsate precisely behind a rib. Only when the apex beats in an intercostal space can the three above features be satisfactorily noted. The apex can sometimes be felt more distinctly when the patient leans forward. In dextrocardia the apex is on the right side of the chest.

Roughly speaking, two abnormal types of apex beat can be recognised: (1) heaving; (2) slapping. A *heaving* apex beat can be recognised by the forcible lift which the fingers experience at each systole when pressed over the apex. It is *the* sign of cardiac hypertrophy and is typically met in cases of aortic regurgitation, hypertension, and, with modifications, in adherent pericardium. The *slapping* apex beat means a poorly contracting left ventricle, and this occurs in three conditions: (1) When the ventricle is badly filled, badly stretched and therefore badly stimulated, and consequently contracts badly; *e.g.*, mitral stenosis. (2) When the muscle has degenerated from coronary arteriosclerosis. (3) When the muscle is poisoned as in diphtheria.

In *hypertrophy* of the left ventricle the apex beat is displaced chiefly downwards, and the cardiac impulse is forcible and heaving. In hypertrophy of the right ventricle there is pulsation in the epigastrium and in the lower interspaces, but the apex is in its normal site. With *dilatation* the impulse is diffuse and weak and the apex beat is moved to the left. The apex is *displaced* in cases of empyema or pleurisy with effusion; if the latter be on the left side, the apex may even be displaced beyond the

[1] **In the child the heart normally differs a good deal from that of the adult. The apex is outside the nipple line until 6 years of age; it is, moreover, often in the fourth space. The right cardiac dulness extends slightly beyond the right margin of the sternum, while on auscultation the first sound at the apex is short (not long, dull and booming); at the base the pulmonary second sound is louder than the aortic second; finally, the rhythm is irregular owing to the heart speeding up during inspiration—sinus arrhythmia (§ 65).**

right border of the sternum (see Fig. 49). The apex is displaced *upwards* in pericardial effusion, collapsed lung, abdominal tympanites, or with any abdominal tumour pushing up the diaphragm. The apex beat is *obscured* by very muscular or adipose chest walls or by emphysema. It is *feeble* with myocardial and pericardial disease, and with ventricular dilatation. With pericardial adhesions there is a *systolic retraction* of one or more interspaces; with hypertrophy of the heart a similar condition may be seen near the apex.

The apex rate should be counted and compared with the pulse. Where the beats are regular, apex and pulse rates coincide, but where the rhythm is irregular, as in auricular fibrillation or premature beats, apex and pulse rates are different. The difference between the two is known as the pulse deficit.

THRILLS.—A thrill is a palpable "purring" sensation corresponding to the murmur of an organic lesion. If present, they should be timed with the carotid and their exact position noted; observe also whether they are constant or intermittent.

Two types of thrill are found in mitral stenosis, both being due to the flow of blood from left auricle to left ventricle. (*a*) The first, the *presystolic* or as it is sometimes called the auriculo-systolic thrill, is due to the blood flow produced by auricular systole. When the auricles fibrillate and no longer contract effectively, this thrill disappears. (*b*) The second is the *diastolic* thrill; it is due to the flow of blood from the left auricle to the ventricle through the stenosed valve during diastole, and is best felt early in diastole when the difference in the pressure in the two chambers is greatest. It may be found together with the presystolic thrill when the auricles are contracting, or alone when they are fibrillating; it generally indicates a severe degree of stenosis. A *systolic* thrill may be present at the apex in mitral regurgitation; at the pulmonary base in pulmonary stenosis; at the aortic base in aortic stenosis and aneurysm. Pericardial friction may produce a thrill. Systolic thrills are also common in congenital heart disease, especially in pulmonary stenosis and interventricular patency.

Any abnormal pulsation should be noted and investigated. Special attention should be directed towards the liver, and the spleen should also be palpated. The condition of the brachial arteries is of importance. Note whether they are visible, tortuous, thickened; if the latter, note whether the thickening is uniform or otherwise, bearing in mind that if patchy in character, this usually indicates involvement of the muscle coat as well as of the intima. The locomotor artery generally signifies two conditions, viz., a rigid vessel and a hypertrophied heart.[1]

§ 40. Percussion.—By percussion one is able to make out the position and the approximate size of the heart. Cardiac dulness is elicited by

[1] In any routine examination of the cardio-vascular system, ophthalmoscopic inspection of the retinæ and of the retinal arteries furnishes valuable information as to the condition of the smaller arteries (§§ 91, 848).

percussion and gives more or less accurately the actual size of the heart. In a normal heart the right margin extends slightly beyond the right margin of the sternum; the left margin is slightly external to the apex, and just internal to the nipple line, while the upper border is approximately level with the third intercostal space. The upper limit of the cardiac dulness is extended when there is dilatation of the pulmonary conus.

Method.—The student should lose no opportunity of PERCUSSING THE NORMAL HEART and of attending to the following points: (i.) *Having first localised the apex-beat,* begin outside the cardiac area in a perfectly resonant area. The middle finger of the left hand should be held vertically and placed flat and *firmly* upon the chest wall in an interspace; then moved ¼ inch at a time towards the centre of the heart. (ii.) Use only one finger—the second of the right hand—as a hammer, making a short sharp tap with the finger *tip*. The percussing finger should rebound immediately—"staccato," as pianists say. The movement should be made from the *wrist*, or from the knuckle (metacarpo-phalangeal joint), as in playing the piano, and the tap should be a light one. (iii.) By listening attentively to the sound elicited, it will be noticed that it is dull and flat over the heart, like that produced by striking any solid object; but louder and more resonant outside the area, like the sound produced by striking an empty barrel. It is only possible to define in this way the right, the upper, and the left limits of the dull area, because at the lower limit the cardiac dulness is continuous with that of the liver. Mark with a blue aniline pencil the right or sternal border in two places. The curved upper and left border of the dulness should also be marked by a pencil in two positions—viz., close to the left side of the sternum, and in another place near the nipple; these can then be joined and continued to the apex beat.

FALLACIES.—Cardiac enlargement may be *obscured* by the hyper-resonance of emphysematous lungs, and in these circumstances enlargement of the heart or pericardium is very difficult to make out. We have then to rely upon other means than percussion. On the other hand, cardiac enlargement may be *simulated* by a fibrous retraction of the left lung, the heart, nevertheless, remaining of normal size; or, thirdly, the heart may be *displaced* by an aneurysm or other mediastinal tumour pushing forward, and making the præcordial area appear larger. One or other border of the area of dulness may be *obscured* by pleural effusion. Ascites, pleural effusion, or abdominal distension may actually *displace* the heart.

§ 41. **The Pulse.**—At this stage, one may well investigate the arterial pulse. The radial is the one commonly selected. The usual method of palpating the pulse is to place three fingers of the right hand on it, when the following points can be systematically investigated: (*a*) *Rate.* Whether abnormally fast or abnormally slow. (*b*) *Rhythm.* Whether regular or irregular; if the latter, the nature of the irregularity should be investigated. Irregular pulses can be classified in two groups: (1) regularly irregular or (2) irregularly irregular. (*c*) The *Force* (estimated by the impact against the finger) depends upon the rapidity of the filling and emptying of the artery, *e.g.*, in aortic regurgitation, hyperthyroidism, anæmia and in certain febrile conditions, the force is considerable. (*d*) The *Volume*, estimated by the lift and duration of the wave, gives one the output of the heart, *e.g.*, in athletes and hyperpiesis. (*e*) The

Tension is estimated by the obliterative force and indicates systolic blood pressure. (*f*) The condition of the *vessel wall.*

The most common regular irregularities are: (1) Sinus arrhythmia (where the pulse speeds up during inspiration, and slows down during expiration), the slowing is vagal, and the irregularity is physiological (§ 65); (2) pulsus bigeminus or pulsus trigeminus (where the pulse goes in twos or threes followed by a pause), the result of regularly occurring premature beats; and (3) pulsus alternans (where big beats and little beats alternate at regular intervals), indicative of left ventricular failure (§ 71): it is of grave prognostic significance. It is difficult to determine by palpating the pulse, but can invariably be detected by the sphygmomanometer, the alternate beats coming through at a slightly lower systolic pressure.

The commonest irregular irregularities are: (1) The perpetually irregular pulse due to auricular fibrillation. Here the beats not only follow one another at irregular intervals, but are of unequal strength and volume. In addition the pulse rate may differ from the apex rate. The great clinical test of the presence of auricular fibrillation is that the irregularity is increased by exercise (§ 68). (2) The irregularity due to irregularly occurring premature beats or extrasystoles (§ 64). This indicates myocardial hyper-irritability, resulting from fatigue, inflammation or degeneration. This irregularity is in most cases abolished when the rate is increased by exercise.

§ 42. **Auscultation.**—For auscultation much practice is required, and the student should never miss an opportunity of listening to the sounds of the heart, *particularly the normal heart.*

The normal heart sounds are three in number—the *First,* or systolic sound, is long, dull and booming in character, and of lower pitch than the second sound. It is best heard over the region of the apex beat, *i.e.,* left fifth intercostal space just internal to the mid-clavicular line. It is due to two factors: (1) the contraction of the ventricular muscle, (2) the vibrations caused by the closure of the auriculo-ventricular valves. The *Second* or diastolic sound is short, sharp, slapping, and higher pitched, and is heard at the apex and at the base on a level with the second costal cartilage. It has two components, being produced by the closure of the aortic and pulmonary semilunar valves. The *Third* sound is also diastolic in time, is occasionally audible by the ordinary stethoscope, and can be easily detected by means of the cardio-phonograph. Its origin is doubtful. In diseased or damaged conditions of the heart not only are the normal heart sounds modified in various ways, to be described below, but adventitious sounds, murmurs or bruits, are liable to be produced either at the valve orifices or on the surface of the heart. When ausculating the heart, therefore, one should pay attention to (*a*) the characters of the normal heart sounds, and (*b*) the presence and character of any abnormal sounds (murmurs).

ALTERATIONS OF THE HEART SOUNDS AND THEIR SIGNIFICANCE.—AT THE APEX. From what has been said about the origin of the First sound,

it is clear that its character will be modified by any condition which interferes with the contractility of the muscle or the closure of the auriculo-ventricular valves. This modification may be: (1) change in pitch, when the First sound becomes somewhat similar to the Second sound, *i.e.*, short and sharp in nature. This indicates a poorly contracting ventricle, due to inflammation, degeneration, toxæmia or non-stretching, *e.g.*, mitral stenosis. It is also found in shock, or after severe hæmorrhage

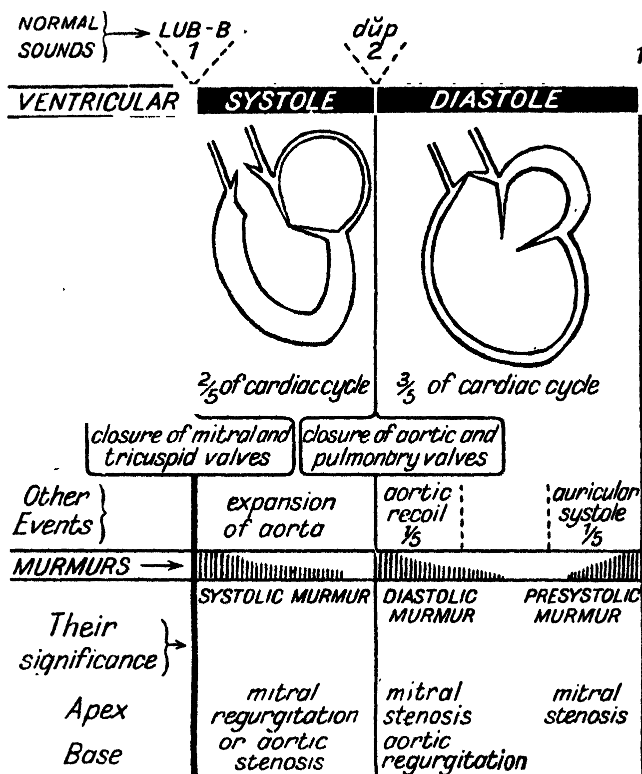


FIG. 10.—Diagram of a Cardiac Cycle, showing various events and their duration, how the different murmurs are produced, and their clinical significance. The student should study this and Fig. 11 very closely.

when the blood-pressure is low. (2) Reduplication due to a non-synchronising closure of the auriculo-ventricular valves. (3) Weakening or suppression due to pericardial effusion, emphysema, myocardial degeneration, myocardial infarction, etc.; or (4) partial or complete replacement by a murmur or adventitious sound.

The second sound at the apex, due to the closure of the aortic and pulmonary semilunar valves, may be (1) distinct; (2) modified by the presence of a murmur as in mitral stenosis, or (3) accentuated when the systemic or pulmonary tension is abnormally high.

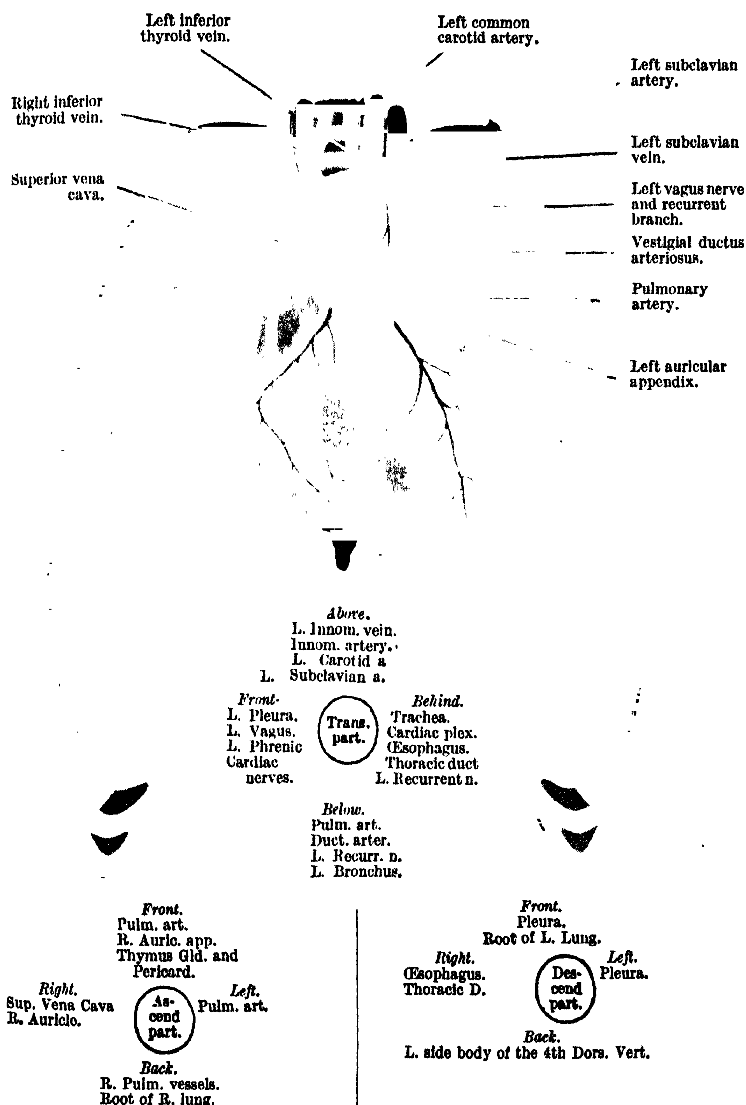


FIG. 11.—**The Heart and Great Vessels in Situ,** with lungs turned back, sketched from the cadaver. Right ventricle forms the greater part of the anterior surface of the heart. Above and to right of this is the right auricle, into which the superior vena cava opens, which collects the blood from the two innominate veins. Passing out from and above the right ventricle is the pulmonary artery, above which again is the remains of the ductus arteriosus, connecting it with the arch of the aorta. Just to the left of the pulmonary artery the left auricular appendix peeps round the corner. The arch of the aorta is seen coming forward from the left ventricle (which is at the back, and therefore only seen at the left margin of the heart), and from its upper convexity arise in order the innominate, left carotid, and left subclavian arteries. The trachea is seen behind the vessels, and the phrenic and vagi nerves are seen at the sides, those on the left passing down in front of the aorta behind the root of the left lung. The relations of the ascending, descending, and transverse portions of the aorta are given diagrammatically above.

A *canter, gallop,* or *triple rhythm* is a condition in which there are three distinct sounds at or internal to the apex. The canter is slower than the gallop rhythm. These sounds are due to reduplication of one of the heart sounds and are indicative of ventricular failure. A canter or gallop rhythm is common when conduction is defective in one or other

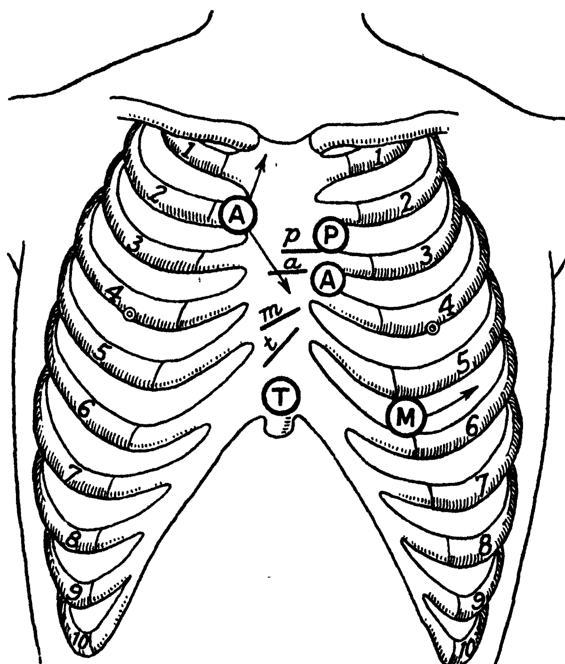


FIG. 12.—DIAGRAM SHOWING THE situation OF THE Cardiac Valves AND THE position IN WHICH THE SEVERAL murmurs ARE HEARD LOUDEST.

p = Pulmonary orifice, at level of upper border of third left costal cartilage.
a = Aortic orifice at level of lower border of third left costal cartilage.
m = Mitral orifice at level of lower border of fourth left costal cartilage.
t = Tricuspid orifice at level of fourth interspace, lying obliquely behind the sternum.
The positions where the sounds produced at the various orifices are best heard are indicated by the letters enclosed in circles. The arrows mark the direction in which murmurs produced at the corresponding orifices are conducted.
M, Mitral murmurs are best heard at the mitral area—*i.e.*, the apex.
A, Aortic murmurs are best heard at the aortic area—*i.e.*, second right costal cartilage; or along the left sternal border.
P, Pulmonary murmurs are best heard at the pulmonary area—*i.e.*, second left intercostal space.
T, Tricuspid murmurs are best heard at the tricuspid area—*i.e.*, at lower end of sternum.

of the branches of the Bundle of His (and see § 55). It is also frequent in large hearts with hypertension, when coronary disease has caused failure with much dilatation.

AT THE BASE, the *aortic second* sound, normally short, sharp and slapping, due to the closure of the aortic semilunar valves, may be (1) accentuated—indicative of a high peripheral resistance and a high blood pressure; (2) ringing, indicative of atheroma and often dilatation of the aorta and rigidity of the valves. This condition, most characteristic

to those who are familiar with it, differs from an accentuation and has another significance. It may, or may not be, associated with a high blood pressure. The aortic second sound is often very accentuated in cases of aortic aneurysm. (3) Absence of the aortic second sound means either that the aortic valves do not close owing to injury, destruction or absence, or that they close so quietly that they do not produce an audible sound. (4) The aortic second sound may be modified by the presence of a murmur which replaces it partially or entirely.

The Pulmonary second sound, due to closure of the pulmonary valves, is also short, sharp and sudden, and in adults less distinct than the aortic second sound. In young children the reverse is the case—the pulmonary second sound being louder than the aortic. It, in turn, may be accentuated (high pulmonary tension), as occurs in mitral stenosis and acute lung conditions; reduplication may be physiological, especially in children, and also occurs in mitral stenosis; or it may be modified by a murmur.

MURMURS.—Murmurs may be either systolic, presystolic or diastolic in time. The latter, moreover, are frequently divided into early, mid, and late diastolic according to the time at which they occur in the diastole. Further, murmurs may be produced either at the valve orifices when they are spoken of as endocardial, or outside the heart, *e.g.*, of pericardial origin. Endocardial murmurs are of two kinds: (*a*) those indicative of structural damage to the valves (organic), and (*b*) those indicative of softening or loss of tone in the auriculo-ventricular rings (atonic or functional murmurs). Endocardial murmurs may generally be differentiated from exocardial murmurs by the following points: (1) *Endocardial* murmurs are best heard in defined areas corresponding to the normal valve sounds; (2) are conducted or propagated in a definite direction; (3) are harsh or blowing in character. *Pericardial* murmurs are: (1) superficial and appear to be heard just under the stethoscope, (2) are usually not heard only over the valve area, (3) are not propagated in the same definite directions, (4) are not necessarily truly systolic or diastolic in time, (5) are often modified by pressure by the stethoscope, and are accentuated, diminished or removed by full inspiration or expiration. A single murmur of presystolic or diastolic time is usually an indication of organic disease at one of the cardiac orifices, but may be exocardial—*e.g.*, pericardial.

Functional Murmurs.—These murmurs, which may be heard over either apex or base, are usually soft and blowing in character. When present at the apex, a functional murmur may be local or conducted to the axilla. They are characterised by their variability under different conditions. Thus they are often present when the patient is lying and disappear when he stands; they may be heard during inspiration and not during expiration; they may appear only after exercise and disappear during rest—or they may only be audible when these conditions are reversed.

Hæmic Murmurs are frequently heard in anæmia and in some other

blood conditions (see Chapter XVI).[1] They are also common in thin-chested adolescents, and in patients with Graves' disease. They are usually systolic in time, are rarely double, are usually heard loudest in the pulmonary area, and are heard best when the patient is lying down.

Atonicity murmurs are found only when the heart has lost its tone from myocarditis or anæmia and are due to stretching of a valvular ring.

§ 43. **Estimation of Myocardial Efficiency.**—The measure of a heart's efficiency is its capacity for work; this is true of all hearts, whether healthy or diseased. Furthermore, it must be clearly borne in mind that many hearts work perfectly, exhibiting no defects at all, when the patients are at rest, but show serious derangements and definite evidence of myocardial impairment when called upon to do extra work. The great symptom of myocardial insufficiency is dyspnœa. If undue dyspnœa is absent, the heart muscle is not failing. The amount of dyspnœa is proportional to the degree of myocardial failure. The dyspnœa is complained of by the patient; it can also be observed objectively by the physician. When taking the history an exact idea should be formed as to how far and how fast a patient can walk, and whether hills or stairs cause shortness of breath. A definite idea as to the amount of work or exercise a patient can take in the course of daily life is the best test of cardiac function. A patient who is made short of breath by the exertion of undressing is unsafe for an exercise test and has a severe degree of cardiac failure.

Effects of Exercise upon the Heart.—(*a*) *The Rate.*—The normal heart responds to exercise by a gradual increase in rate. The increase is more or less uniform, the rate climbing up as exercise is increased. The normal heart rarely speeds up to over 150 for any length of time. It rapidly returns to normal on ceasing the exercise. A soft atonic or poisoned heart responds to exercise by undue acceleration, and very slowly settles down to its normal rate; while in certain diseased conditions of the heart one gets impaired acceleration, the rate scarcely altering at all. This may occur in very fast hearts (*e.g.*, auricular flutter), or in very slow hearts (*e.g.*, heart-block). Lastly, in a well-trained physiological heart, such as one meets with in young highly-trained athletes, the rate does not climb on exercise, but suddenly doubles (*e.g.*, at the commencement of the exercise, the rate may be 42, and on exercise suddenly becomes 84)—the so-called athletes' reaction.

(*b*) *Rhythm.*—The rhythm of the heart may be profoundly modified by exercise. (1) An irregularity may be produced, and any heart that becomes irregular on exercise is likely to be diseased. The most common irregularities revealed by exercise are auricular fibrillation, alternation (indicative of left ventricular failure), a sign of grave significance, and, in some cases, premature beats. (2) An existing irregularity may be abolished. Practically speaking, the only irregularities abolished by exercise are those caused by sinus arrhythmia and premature beats. (3) An existing irregularity may be increased. This is true of auricular fibrillation or extrasystoles due to disease.

(*c*) *Sounds.*—Under the influence of exercise, normal heart sounds may be reduplicated or modified by the production of adventitious sounds or murmurs. In early mitral stenosis, exercise brings out the signs of the lesion.

(*d*) *Thrills.*—Thrills may be actually made evident. This occurs in early mitral stenosis, when increased filling of the auricle results in increased stretching and increased contraction, and so produces a thrill, presystolic in time. An existing thrill is increased by exercise or abolished by exhaustion or by tachycardia.

[1] These so-called hæmic murmurs are not due to anæmia *per se*.

§ 44. **Special Methods of Investigation.**—1. **The Polygraph** is an elaboration of the older sphygmograph. Historically, it is of great interest, for by its invention and use Sir James Mackenzie was the first to analyse and classify the cardiac irregularities. His findings were confirmed by the electrocardiograph. Clinically, its chief value is to record auricular movement in cases of obscure irregularity. When the auricular and ventricular complexes overlap in the electrocardiographic record, simultaneous polygraphic and electrocardiographic tracings will often clear up the difficulty by defining the position of the hidden " P " wave. In the normal polygraph tracing the " A " wave coincides with auricular, and the " C " wave with ventricular systole. The " V " wave coincides with the opening of the tricuspid and mitral valves at the start of diastole.

2. **The Electrocardiograph** is an instrument for recording the minute electrical currents which are formed by the contraction of heart muscle. The two principles usually employed in the instrument are those of the string galvanometer, and the cathode-ray oscillograph. The electrical changes are photographed on a moving photographic film or plate, and the record is called an *Electrocardiogram.* The patient is placed in circuit with the machine by the following four sets of leads: With lead 1 the two electrodes are applied respectively to the right arm and the left arm ; lead 2 to the right arm and the left leg ; lead 3 to the left arm and the left leg. In the standard lead 4 the left arm electrode is placed at the apex and the right arm electrode either remains *in situ* (lead CR4) or is connected with the left leg (lead CF4) : these give normally an upright " T " wave, and leads CR4 and CF4 give almost identical curves. Other leads may be used in certain conditions : the right pectoral lead—right lower sternum to right arm—is valuable in the diagnosis of pulmonary embolism. Fig. 13 represents a normal set of curves : 1, 2, 3, CR4 refer to the corresponding leads.

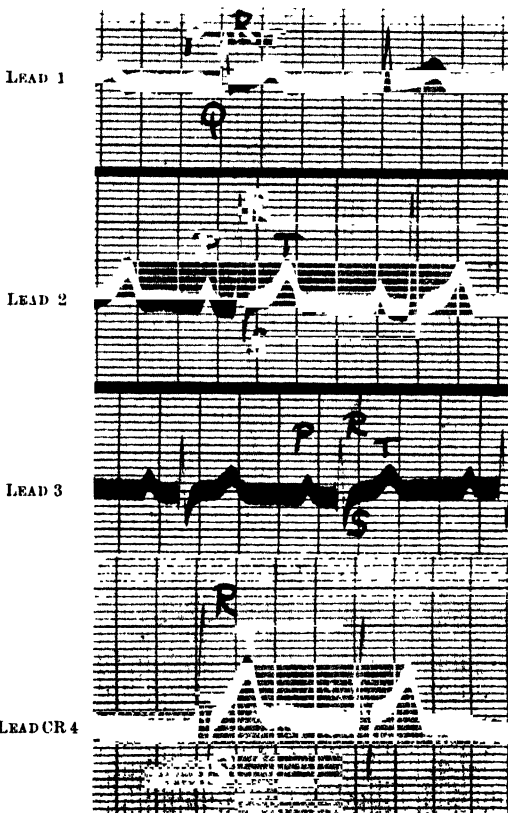


FIG. 13.—A normal tracing showing leads 1, 2, 3 and CR4. (Thick lines indicate 0·2 of a second and thin lines 0·04 of a second.)

When reading an electrocardiogram the following routine is advisable. First observe if the whole of the tracing is slightly blurred by very fine oscillations : these are due to fine muscular tremors with failure of the patient to relax, and are common in nervous subjects and in Graves' disease. Next examine the " P " waves in all

leads. These are produced by the auricles and for each lead should be uniform in shape and upright: occasionally in lead 3 they are diphasic or inverted in adipose patients with an abnormally elevated diaphragm. The amplitude of the "P" wave is increased if the auricles are hypertrophied, as in early mitral stenosis: while if the two auricles do not quite synchronise the wave is notched (*e.g.*, mitral stenosis). The "P" wave may be absent, and in its place there may or may not be irregularly occurring fine fibrillary waves (the ventricular waves being totally irregular): then auricular fibrillation is present (Fig. 14). Isolated "P" waves may be inverted, indicating that the auricles are here contracting from some ectopic pacemaker and not from the sino-auricular stimulus: these auricular premature beats may arise in any part of the auricular muscle, but the nearer they are to the sino-auricular node, the more closely does their shape approximate to normal (Fig. 15). If a series of these inverted or abnormal "P" waves occurs regularly at a rate higher than normal (between 120–200 per minute) and the ventricle contracts with each auricular beat, paroxysmal tachycardia (Fig. 34) is present. If no normal "P" waves are present, but instead there is visible a series of regular coarse undulations at a rate of between 200–300 per minute and best seen in leads 2 and 3, auricular flutter (Fig. 35) is present. Here the "QRST" follows either each second, third or fourth auricular undulation, giving an auriculo-ventricular ratio of 2 : 1, 3 : 1, or 4 : 1.

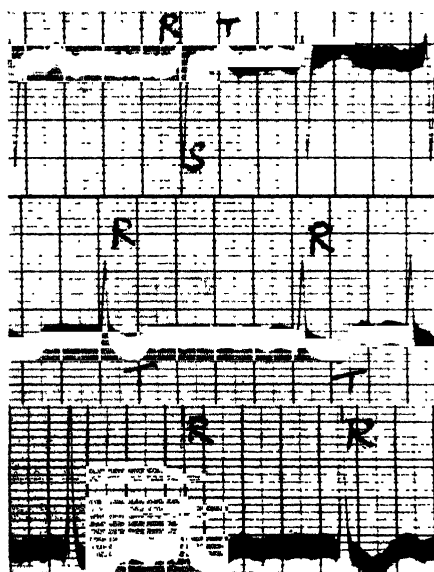


FIG. 14.—Auricular fibrillation. There is no "P" wave. Between the ventricular beats are seen fine fibrillary movements of the auricle. The ventricular rhythm is completely irregular. Note also the right axis deviation due to the fact that the tracing is from a case of advanced mitral stenosis.

Next measure the P–R interval. This represents the interval between the commencement of auricular and ventricular contractions, and is chiefly occupied by the time taken for the impulse to traverse the bundle of His. It should measure ·12–·20 sec. Heart-block is present in a minor degree if the P–R interval is prolonged beyond 0·20 sec. (Fig. 16), and in a greater degree if a ventricular beat drops out (Fig. 17): if a "P" wave is not followed by a QRS complex, but two, three or four "P" waves intervene between each QRS complex, then 2 : 1, 3 : 1 or 4 : 1 heart-block is present. If the interval between QRS and the nearest "P" wave varies continually, complete heart-block is present (Fig. 18). Occasionally the P–R interval is shortened. This is due to nodal rhythm with an abnormal pacemaker situated between the sino-auricular and auriculo-ventricular nodes (Fig. 19).

Next study the QRST portions representing ventricular action. The QRS portion should not exceed ·10 sec.; this is increased when conduction is impaired in one branch of the bundle of His. Normally, the "R" wave is tallest in lead 2: left axis deviation (Fig. 20) is shown when the "R" wave is tallest in lead 1, and the "S" wave deepest in lead 3. Conversely, with right axis deviation (Fig. 14) the "S" wave is deepest in lead 1 and the "R" wave tallest in lead 3. A ventricular complex of abnormal shape and size, placed between others which are normal, indicates a premature ventricular beat



FIG. 15.—Two auricular premature beats (P') are shown, each arising from a different focus. The first ectopic " P " wave is inverted, showing its abnormal position of origin, and is also premature.



FIG. 16.—First stage of heart-block. " P-R " interval measures 0·3 of a second.

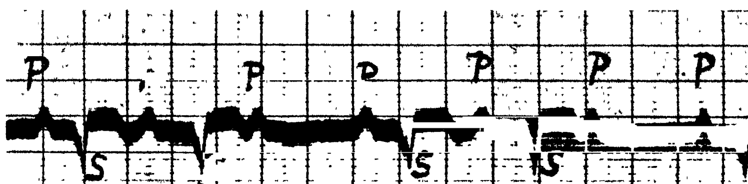


FIG. 17.—Heart-block. Stage of dropped beats. The " P-R " interval at first measures 0·20 of a second, then 0·25 of a second, and the third " P " wave fails to excite a ventricular contraction.

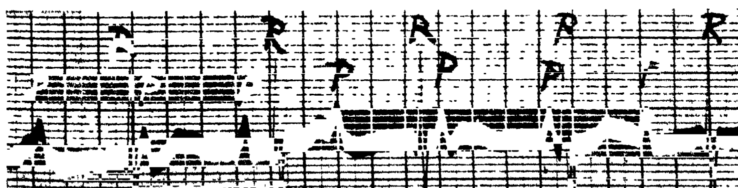


FIG. 18.—Complete heart-block. There is no relationship between the auricular and ventricular contractions.

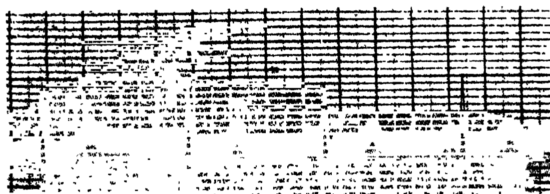


FIG. 19.—Tracing showing shortening of the " P-R " interval. This is due to nodal rhythm, in which the pacemaker is situated between the sino-auricular and auriculo-ventricular nodes. The impulse reaches the auricles slightly before it reaches the ventricles.

(Fig. 32). The start of the ST interval should be isoelectric and the "T" wave well formed and upright in all three leads, although if the "T" wave in lead 3 (T_3) is inverted this has no special significance. This wave, especially in lead 2, is well developed in proportion to the physiological state of the ventricular muscle. It is inverted in leads 1 and 2 by full doses of digitalis, in myocardial disease, in acute and chronic pericarditis, and in some cases of aortic regurgitation, and is decreased in amplitude in myocardial toxæmia or degeneration. Suddenly occurring inversion of the "T" wave is a feature of coronary thrombosis (Figs. 25, 27), in which condition almost any variation may occur in the shape of the "QRST" waves, such variation occurring rapidly and often disappearing within a few weeks. In coronary infarction that part of the curve immediately following R or S fails to return to the resting base-line before continuing on to the "T" wave. This is known as R–T or S–T deviation and is characteristic of coronary disease (Pardee's sign). With infarction of the anterior coronary artery the R–T deviation is above the base-line in lead 1 and below it in lead 3. In posterior infarction the reverse is the case in both leads.

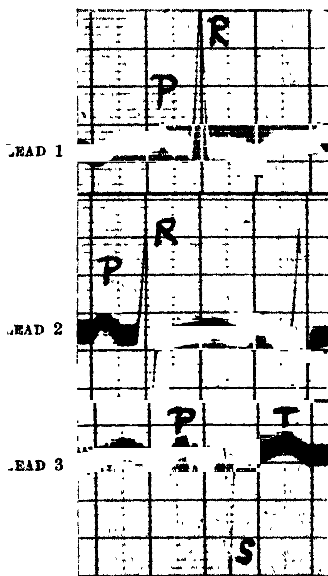


FIG. 20.—Left axis deviation, from a case of aortic regurgitation: "R" is tallest in lead 1, and "S" is deepest in lead 3.

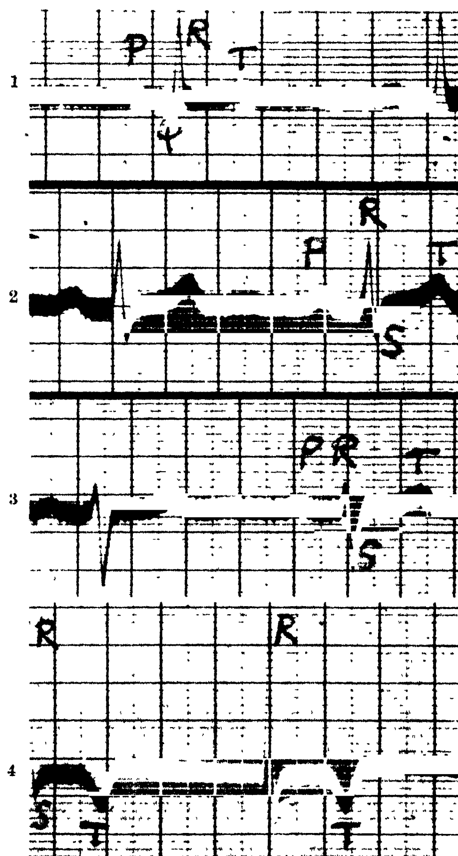


FIG. 21.—A tracing showing left axis deviation, but no other abnormality in leads 1, 2, 3. In lead CR4 the "T" wave is inverted, indicating disease of the ventricular muscle.

Lead CR4 should show a vertical—or upright—"T" wave. The "T" wave here is also inverted in coronary thrombosis and in chronic myocardial disease. The advantages of lead CR4 are that the changes are often more definite than in leads 1,

2 or 3, and in some cases may be present when they are absent in leads 1, 2 and 3 (Fig. 21).

3. No examination of the cardio-vascular system can be regarded as complete unless it includes an *X-Ray* investigation (Figs. 22, 23A and 23B). The technical

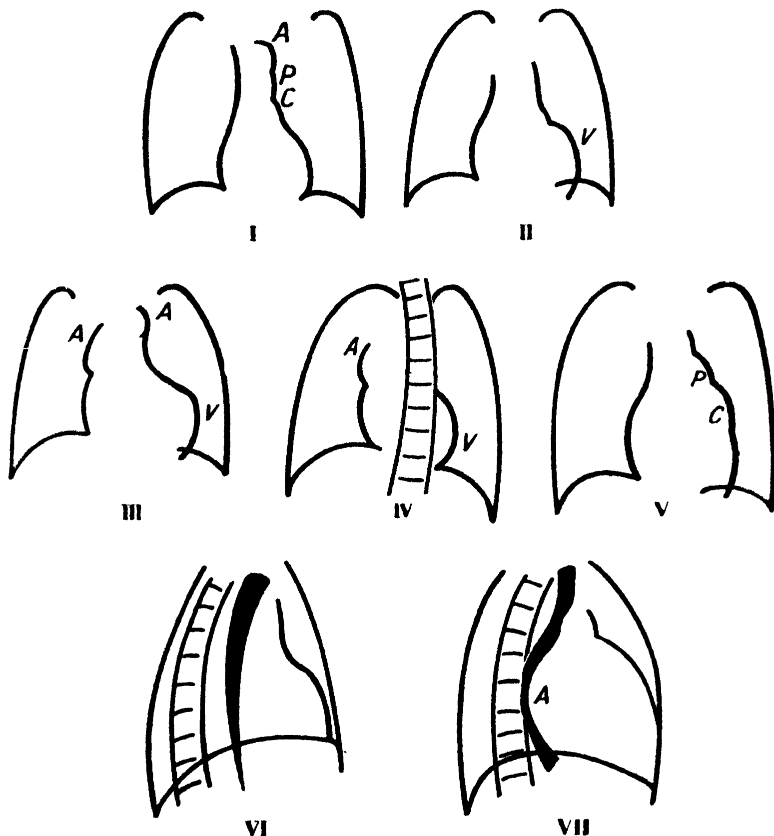


FIG. 22.

I. The normal anteroposterior outline of the heart.
II. Left ventricular hypertrophy (V), in aortic stenosis, without enlargement of the aorta.
III. Left ventricular hypertrophy and dilatation (V), with enlargement of the ascending and transverse aorta (A, A).
IV. Left anterior oblique view, showing left ventricular enlargement (V), and enlargement of the aorta (A).
V. Anteroposterior view in mitral stenosis, showing enlargement of pulmonary conus (C), and prominent pulmonary artery (P).
VI. Right oblique view, showing normal œsophageal shadow.
VII. Right oblique view in mitral stenosis showing displacement backwards of œsophagus filled with barium, outline of left auricle shown (A).

details must be obtained from a larger textbook; the main points to which attention should be directed may be thus summarised:—

A. THE HEART:—

(1) *Its position* in the mediastinum—its relation to the lungs and diaphragm.

(2) *Outline and form*: This is often characteristic, *e.g.*, in young healthy hearts

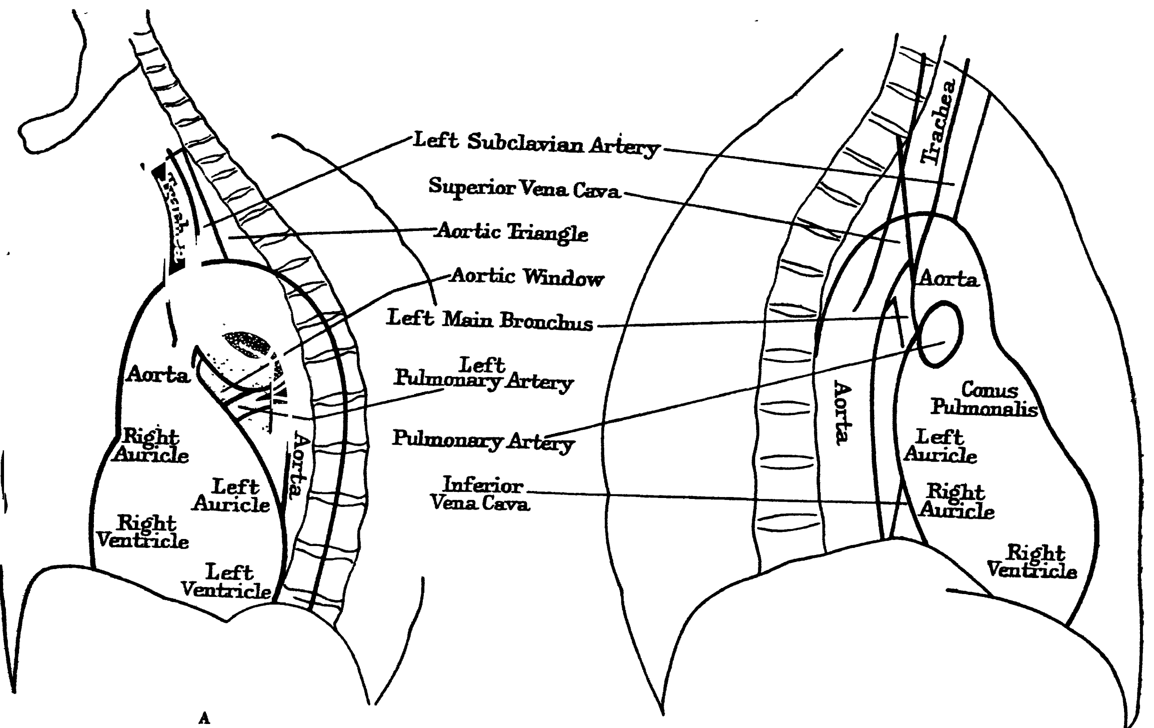


FIG. 23.—Outline of Normal Cardio-vascular Shadow.
A—Left oblique view (left shoulder forward).
B—Right oblique view (right shoulder forward)

it is vertical, in congenital heart disease round, in old valvular disease globular, but varies with the lesion. In enlargement of the left ventricle it is boot-shaped with the long axis horizontal, while in pulmonary tuberculosis it is elongated and tubular. Hypertrophy or dilatation of the left and right ventricles, enlargement of the left auricle, as in mitral stenosis, are some of the chief points to be demonstrated by radiography.

(3) *Size:* The maximum transverse diameter of the heart is less than half the greatest diameter of the thorax. Thus a cardio-thoracic ratio of more than 0·5 is evidence of enlargement.

B. THE GREAT VESSELS: Examine especially in oblique positions; note cardio-phrenic angle; ? dilatation or aneurysm; ? mediastinitis or growth or foreign body.

C. THE PERICARDIUM: ? adhesions; ? fluid.

4. *The Orthodiagraph* is an instrument whereby outline diagrams can be made of the actual size of the heart itself, so that accurate measurements can be obtained not only of the heart but also of the great vessels, etc. An outline of the cardiac shadow, resulting from parallel rays, is traced on an X-ray screen. A six foot X-ray film (*Teleradiogram*) is often substituted for this.

Circulation Times.—It may be helpful to determine local or general slowing of the speed of the circulating blood. The blood may travel more slowly as a whole, or it may be held back in some local area such as the venæ cavæ or the pulmonary veins and capillaries. This slowing can be measured. If decholin, which tastes bitter (10 c.c. of 20 per cent. solution), or calcium gluconate, which causes a hot sensation in the mouth (4 c.c. of 20 per cent. solution), be injected rapidly into a vein, the interval between the time of injection and the sensation of bitterness or sweetness in the patient's mouth, gives the time taken for the blood to traverse most of the venous and the whole pulmonary system, together with a second or so of arterial transit. In normal resting adults this time averages 12 sec., the upper limit being 16 sec. If ether is injected (5 ♏ with 5 ♏ of normal saline) the time interval taken is from the injection to the first notice by the patient of ether in the breath. This measures the arm to lung time or the amount of delay in the systemic venous system. (Average normal figure, 5 sec.; upper normal limit, 9 sec.)

PART C. DISEASES OF THE HEART AND PERICARDIUM: THEIR DIAGNOSIS, PROGNOSIS, AND TREATMENT

§ 45. Classification.—For practical purposes diseases of the heart and pericardium may be classified under five prominent differential features: Disorders WITH PYREXIA; Disorders in which PAIN is a characteristic symptom; Disorders which are attended by an ENLARGEMENT of the AREA of CARDIAC DULNESS; Disorders in which an ALTERATION of the CARDIAC SOUNDS, or a MURMUR forms the diagnostic feature; and Cardiac conditions which are recognised by an ALTERATION of the RHYTHM or RATE of the PULSE.

A. PYREXIA . . . . {
I. Pericarditis.
II. Acute Endocarditis.
}

B. PAIN . . . . {
I. Angina of Effort.
II. Spasmodic Angina.
III. Coronary Thrombosis.
IV. Angina Innocens (Pseudo-Angina).
V. Pericarditis.
}

C. ENLARGEMENT OF THE AREA OF CARDIAC DULNESS .	I. Cardiac Hypertrophy. II. Cardiac Dilatation. III. Chronic Pericardial Effusion IV. Adherent Pericardium.
D. ALTERED HEART SOUNDS AND MURMURS . .	I. Myocardial Degeneration. II. Endocarditis. III. Congenital Heart Disease. IV. Pericarditis.
E. ALTERATION OF RHYTHM OR RATE OF PULSE .	I. Sinus Arrhythmia. II. Premature Beats (Extrasystoles). III. Tachycardia. IV. Auricular Flutter. V. Auricular Fibrillation. VI. Bradycardia. VII. Heart-Block.

The **Routine procedure** in the investigation of a cardio-vascular problem may be considered under the following headings—(1) The origin of the present symptoms, *e.g.*, whether they supervened on any definite illness, acute or chronic, or followed on some definite action, emotion, etc.

(2) The personal history, especially as regards (*a*) previous diseases such as rheumatic fever, growing pains, chorea, scarlet fever, tonsillitis, influenza, diphtheria, syphilis, etc.; (*b*) habits of life, especially as regards exercise, alcohol and tobacco.

(3) Family history. Certain diseases, *e.g.*, rheumatic fever, arterio-sclerosis, etc., tend to run in families, and predispose to heart disease.

(4) Symptoms. The commonest symptoms associated with heart disease are dyspnœa, palpitation, pain, vertigo, faintness, and a sense of exhaustion. These are dealt with in Part A.

(5) Physical examination of the patient (Part B). Inasmuch as many hearts, when only slightly damaged, function normally when the patient is at rest and their "load" is light, but develop obvious defects of action under "load," it is essential to examine the patient three times—standing, lying, and after exercise. Further, it is convenient to divide the examination into: (*a*) Ordinary routine clinical examination, under which heading one would include the results of inspection (§ 38), palpation (§ 39), percussion (§ 40), the pulse (§ 41) and auscultation (§ 42). (*b*) Cardiac efficiency tests (§ 43) used for ascertaining the reserve energy of the heart. (*c*) Special instrumental methods of examination (§ 44).

GROUP A. If the symptoms of which the patient complains are unattended by Pyrexia, turn to § 51. If the disease is **attended by Pyrexia**, it is probably ACUTE PERICARDITIS or ACUTE ENDOCARDITIS, either rheumatic or infective in origin, or CORONARY THROMBOSIS. It must be remembered that cardiac patients are often subject to other febrile illnesses.

I. THE TEMPERATURE IS ELEVATED, *the patient is in evident distress, and the præcordial area of* DULNESS IS INCREASED, *the shape of the dulness*

being PYRAMIDAL, *with the point upwards.* A PERICARDIAL FRICTION SOUND *is audible. The disease is probably* ACUTE PERICARDITIS.

§ 46. **Acute Pericarditis** is an acute inflammation of the pericardial sac. It is not infrequently met as a primary affection. It supervenes during the course of many different diseases, and the symptoms of these may mask its onset. Rheumatic fever is certainly its most common cause, and it should be remembered that it may be the first manifestation of this affection. We should always examine the heart daily in patients with rheumatic fever or nephritis, because in these acute pericarditis may come on insidiously, without pain or tenderness. Its advent in rheumatic fever is marked by high fever, tachycardia, pallor and vomiting (especially in children) or by the occurrence of delirium.

Symptoms.—(1) The patient wears an anxious, troubled look, and the cheeks are generally pallid ; a distinct puffiness of the face, not amounting to obvious œdema, is often present ; there are fever and a rapid pulse ; the breathing is rapid, and he may complain of severe pain in the left chest (occasionally referred to the abdomen), increased by pressure, movement, or respiration ; a short irritative unproductive cough is common. Abdominal rigidity may occur. (2) *Physical Signs.*—The præcordial dulness is increased in all directions, and the cardio-hepatic angle of resonance, to the right of the heart, becomes obtuse instead of acute. A friction sound is heard on auscultation. It is harsh, somewhat creaking in character, generally double, frequently triple, and occasionally systolic only. This may be distinguished from a murmur produced *within* the heart by (i.) usually being double, *i.e.*, accompanying the movements of the heart, and rarely exactly synchronous with the first and second sounds ; (ii.) the second part of the rub is occasionally continuous with the first, without any diastolic pause ; (iii.) it is often loudest at the root of the great vessels, over the third left costal cartilage ; (iv.) it varies in its character from time to time, and is increased by firm pressure with the stethoscope ; (v.) pressure will also elicit another character—viz., that the disease is usually accompanied by tenderness, as well as pain. The differentiation between peri- and endocardial murmurs is so important that it is also given in a tabular form below (Table I, p. 67). To distinguish pericardial from pleural friction is very easy, because the latter ceases if the patient holds his breath. Note that as the effusion occurs the murmur may become less distinct, but it rarely disappears entirely. It is again intensified as the effusion clears up. In most cases of acute pericarditis, with or without effusion, physical signs are to be found over the left lower lobe behind. These signs are those of collapse of the lung, and first occur, and are last to disappear, at the apex of the lobe ; they may involve the whole lower lobe.

Second Stage, or stage of pericardial effusion. The inflammation may subside, but occasionally, in the course of a day or two, effusion of fluid occurs, and the pain and tenderness diminish. Effusion of a quantity of fluid or more than half a pint is very rare in rheumatic pericarditis.

When a large effusion does occur, the rub becomes less audible, though it may still be heard at the base of the heart. The breathlessness and other symptoms continue; the cough becomes more troublesome; dysphagia and vomiting rarely occur. Pulsus paradoxus may be present. *The increased area of dulness*, due to pericardial effusion, may be greater than the enlargement from any other cause. (i.) It is of *triangular shape*, with apex upwards, reaching to the third, or even second, costal cartilage. (ii.) There is often actual or apparent *raising of the position of the apex beat.* (iii.) The *dulness extends to the left* of the apex beat. There is progressive weakening of the heart sounds at this time, from the associated myocarditis.

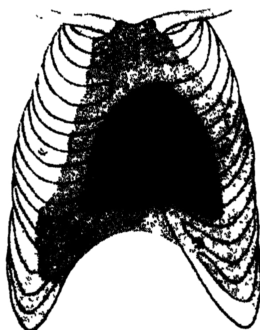


FIG. 24.—DIAGRAM FROM A CASE OF RHEUMATIC PERICARDITIS WITH EFFUSION.—Eliza P., aged twenty-seven. Heavy shading corresponds to position of maximum intensity of friction. Medium shading corresponds to the area of deep cardiac dulness. Light shading corresponds to the area over which the pericardial friction is audible. It is often taught that one of the features distinguishing peri- from endocardial murmurs is the limitation of the former to the præcordial region; but I have many times satisfied myself that this is not so, and this case is one of several examples I have met with verified by autopsy.

Etiology.—The causes may be classified under five headings: (1) acute infections, *e.g.*, rheumatism, scarlet fever, pneumonia, pyæmia, etc.; (2) extension from adjacent structures, *e.g.*, malignant disease and tuberculosis; (3) chronic nephritis; (4) coronary disease or infarction; (5) injury.

Course and Prognosis.—The duration of acute pericarditis varies widely, according to the cause, but it averages about fifteen to twenty-five days. It may undergo resolution with or without the formation of adhesions (Adherent Pericardium, § 56); or result in chronic pericardial effusion (§ 56); or become purulent (Pyopericardium, § 47 below). Pericarditis with effusion is always a serious malady; but the prognosis depends much on the underlying cause, the amount of distension of the pericardial sac, and the evidences of interference with the cardiac action—dyspnœa and cyanosis with feebleness, rapidity, and irregularity of the pulse. Pericarditis complicating rheumatism, like the other complications of that disease, tends to recover, but it frequently leaves a weakened heart (damaged myocardium), and leads to cardiac dilatation (enlargement). In renal disease it is a serious though often latent affection; and in pyæmia, when it is generally purulent, is nearly always fatal. In infancy and in debilitated patients it is also grave.

Diagnosis.—The diagnosis from acute endocarditis has been considered above, and in Table I, § 49. It is distinguished from enlargement of the heart by the following points: the left border of the dulness in pericardial effusion extends beyond the apex beat, and the apex beat may be displaced upwards; the right border of dulness has a convex outline and the cardio-hepatic angle at the right fifth intercostal space is obtuse; lack of movement of the epigastrium with respiration is another valuable

sign. Both conditions may be present at the same time. X-Ray examination will usually enable one to verify the diagnosis. It should be remembered that inflammatory conditions of the *left lung* and *pleura* not infrequently give rise to a to-and-fro friction sound along the left border of the heart; this is produced by pleural and not by pericardial inflammation (pleuro-pericardial friction). The intensity of this friction often varies with respiration. The signs of consolidation of the left lower lobe described above may occasionally suggest a diagnosis of *lobar pneumonia.*

Treatment.—In the inflammatory stage the patient must be kept lying comfortably in bed *and absolutely forbidden to move.* A fluid diet is advisable. A poultice, or warm fomentation applied to the præcordium usually gives more relief than the ice-bag, though this undoubtedly relieves the symptoms, controls the restlessness of a young patient, and possibly also reduces the heart rate. If the pain is great, relief is often obtained from the application of four or five leeches over the præcordium. Blisters are occasionally used. If cyanosis, orthopnœa, and venous distension are present, indicating considerable cardiac embarrassment, bleeding (15 to 20 ounces) is a prompt and efficacious measure. Opium by mouth, or morphia hypodermically, is of great value for the pain and distress, much smaller doses being given to children. The bowels should be regulated. Stimulants, such as nikethamide B.P. (coramine), can be given when the blood pressure falls. Mersalyl is often useful in the more chronic forms of pericardial effusion. For hyperpyrexia and delirium tepid or cool sponging is a useful means of lowering the temperature, and will often induce sleep.

Treatment of the cause of the pericarditis should be combined with the foregoing—*e.g.*, sodium salicylate combined with alkalies for acute rheumatism; diuretics and hot-air baths for renal disease. In the stage of effusion free blistering promotes absorption, but it must be remembered that renal disease is a contra-indication to blistering. If the effusion becomes chronic, give diuretics; mersalyl is very useful but must be used with great care in nephritis. Iodine paint and other local counter-irritants can be tried.

Paracentesis Pericardii.—When the amount of effusion becomes considerable, as shown by marked dyspnœa, cyanosis, distention of the neck veins, tachycardia and lowered blood pressure, paracentesis pericardii should be performed. The site chosen for this procedure may be in the fifth left intercostal space just inside the mid-clavicular line, or one inch from the margin of the sternum in the same space; or in the angle between the ensiform cartilage and the left costal margin, near the lower end of the body of the sternum and passing upwards and inwards behind it into the pericardial sac. A trocar and cannula or an aspirating needle or a Potain aspirator may be employed. Eight or twelve or even forty ounces (in a chronic case) of fluid may be slowly removed. This operation is rarely required; it is never necessary in the rheumatic pericarditis of childhood.

§ 47. **Pyopericarditis.**—Sometimes in debilitated children and in the course of pyæmia, in phthisis and empyema, and in some other conditions, the fluid in the pericardium takes on a purulent or sero-purulent character. This is sometimes revealed (as is a collection of pus in other parts of the body) by (1) shivering attacks,

(2) profuse perspirations, and (3) a temperature with wide variations in the course of a few hours, in addition to the clinical features of acute pericarditis above described. But it is difficult to diagnose, because the *friction sound is usually transient.* It is often fatal.

Pyopericarditis is the form which pericarditis frequently assumes in infancy, and is often not diagnosed. Progressive weakness, fever, anæmia, leucocytosis and X-ray examination may suggest the presence of pus.

Treatment.—A large sterilised (No. 10) needle should be very carefully introduced whenever the existence of pyopericardium is suspected. If the fluid withdrawn be of a purulent nature, paracentesis, or free drainage, should be effected. Penicillin is injected locally once a day, and is administered systemically by three-hourly injection if the infecting organism is sensitive.

Pneumopericardium is a very rare condition in which air reaches the pericardial sac from the lungs or stomach.

Hæmopericardium is rare. Aneurysm of the first part of the aorta or of the cardiac wall, rupture or wounds of the heart, scurvy and other blood diseases, may lead to sudden death owing to the sudden influx of blood into the pericardium. A small amount of bleeding may be seen in the pericarditis due to nephritis, malignant growths, acute rheumatism and tubercle.

§ 48. **Latent Pericarditis**—*i.e.*, pericarditis without *symptoms* (though not necessarily without physical signs). In most patients in whom we find a pericardial effusion a history of acute pericarditis is obtainable; but it is not sufficiently recognised that pericarditis may come on insidiously, without acute symptoms. The effusion may be discovered during routine examination of the heart, or perhaps not until autopsy. Moreover, at the post-mortem a totally adherent pericardium is sometimes found in a patient in whom careful inquiry has failed to reveal any symptoms pointing to the heart during life. In ACUTE RHEUMATISM *its advent may be indicated only by delirium or vomiting.*

Pericarditis frequently results in Adherent Pericardium (§ 56).

We now pass to the other acute disorder with pyrexia.—II. ACUTE ENDOCARDITIS.

II. THE TEMPERATURE IS ELEVATED. *The præcordial area of dulness is not necessarily increased, and on auscultating the chest there is a* MURMUR *added to the heart sounds—the disease is probably* ACUTE ENDOCARDITIS. *It is not always easy to distinguish an endocardial from a pericardial murmur.*

§ 49. **Acute Endocarditis** is acute inflammation of the valves or of the mural endocardium of the heart. It is usually attended by enlargement of the præcordial dulness, because some degree of myocarditis and dilatation is associated with it. In a large proportion of cases it complicates some other disease; and, like pericarditis, it is most frequently associated with acute rheumatism; it may even be the first evidence of that disease.

There are two varieties of endocarditis with fever: RHEUMATIC Endocarditis, and INFECTIVE or MALIGNANT Endocarditis.

In RHEUMATIC ENDOCARDITIS: (1) *a murmur develops* at the apex or base of the heart, corresponding to the mitral or aortic valves (see Fig. 12). The mitral valve is most frequently involved in acute rheumatism, but the mitral and aortic valves may be affected together, or rarely the aortic valve alone. The murmur is usually soft and heard over a limited area, and only occasionally is it harsh soon after its appearance. (2) There is often some myocarditis sometimes causing cardiac

TABLE I.—DIAGNOSIS OF ENDOCARDIAL FROM PERICARDIAL MURMURS.

Endocardial Murmurs.	Pericardial Murmurs.
1. May accompany first or second sound only, or both.	Usually double—always superficial and are as loud in diastole as in systole; not quite synchronous with the heart sounds.
2. Often loudest at one of the valvular areas.	Usually loudest over third left costal cartilage (root of big vessels).
3. May be conducted into the axilla, or along the aorta and carotids.	Mostly confined to the præcordium.[1]
4. Usually no pain or tenderness.	Often accompanied by pain.

dilatation, with a weakened diffuse apical impulse and weak cardiac sounds. (3) *Constitutional symptoms* may be marked when there are the general symptoms and signs of rheumatic fever (§ 582); but in children these may be slight or even absent. The onset of endocarditis may then be suspected when there is sudden pallor, with loss of physical vigour, accompanied by an evening rise in temperature and increase in pulse rate. Præcordial pain and distress are rarely found—a point worth remembering. The erythrocyte sedimentation rate is always raised when active endocarditis is present. The presence of rheumatic nodules (§ 573) around the elbows, wrists, knees and ankles usually indicates a severe form of active carditis.

Causes of Rheumatic Endocarditis.—It must be remembered that acute rheumatic endocarditis may complicate, not only rheumatic fever proper, but rheumatic tonsillitis, chorea and scarlet fever. The patient is generally young, usually a child: it tends to run in families.

The *Diagnosis* of acute rheumatic endocarditis is sometimes difficult. The murmur will be found over the mitral or aortic valve, will immediately follow a valve sound, will be localised and if conducted will follow the direction of the murmur of cardiac valvular disease (Fig. 12); it will not be materially affected by change of posture or by deep breathing. Care must be taken to avoid confusion with exocardial murmurs (§ 59). The distinction from pericardial murmurs is set forth in Table I. In view of the great tendency to recurrence of acute rheumatism and of rheumatic endocarditis, a careful history of previous attacks should be taken: a mitral stenotic murmur is evidence of an attack of acute rheumatism at least six months previously. Therefore, if signs of an acute endocarditis are also present, the acute attack now suffered from cannot be the first. Pyrexia, tachycardia, an increase in the size of the heart, and the intensity of the murmur and a raised erythrocyte sedimentation rate indicate active inflammation of the valve or valves. *Acute infective endocarditis* differs clinically (1) in the greater severity of the constitutional symptoms, with often a wide range of the diurnal temperature, and even rigors;

[1] For an exception to this, see Fig. 24, p. 64.

(2) in the occurrence of systemic emboli; (3) the presence of splenomegaly, clubbing of the fingers and a positive blood culture. Acute infective endocarditis may supervene on a valve previously damaged by rheumatism, and the diagnosis may become extremely difficult: persistent absence of embolism, a persistently negative blood culture and the presence of auricular fibrillation suggest an active rheumatic lesion. Malignant endocarditis rarely supervenes upon a recently active rheumatic lesion. The endocarditis resulting from syphilis or arteriosclerosis is an afebrile condition.

The *Prognosis* of rheumatic endocarditis, though the malady may last for many weeks, or even months, is favourable as regards life, but the damage to the cardiac valves is generally permanent, and then the prognosis turns on many important considerations (§ 61). A tendency to recur is one of the most striking features of the disease.

Treatment should be directed primarily to the rheumatic fever: sodium salicylate is usually thought to have no control over the cardiac lesion. Digitalis is occasionally of use in reducing the heart rate in cases of persistent tachycardia. Iron is useful as ferri et ammon. cit. 15 gr. t.d.s. for the anæmia which is so often present. Penicillin is of no value in this condition. *Perfect rest*—hardly allowing the patient to turn in bed—is absolutely essential. The patient should be confined to bed until fever has been absent for at least 6 weeks, the sedimentation rate has been normal for 4 weeks, the pulse rate has returned to normal and has remained normal for 3 weeks, anæmia has vanished, and the patient has begun to put on weight. Careful treatment of the condition may require prolonged rest in bed for 6 to 9 months (and see § 582). The erythrocyte sedimentation rate is of great value in assessing the arrest of the active process (§ 927).

§ 50. **Infective, Bacterial, Malignant or Septic Endocarditis.** This is an acute infection which attacks the heart valves or the endocardium already damaged by previous disease. It is characterised by the presence of the large vegetations, by local destruction of the valves, fever, bacteræmia and multiple embolism. The disease probably never attacks a healthy endocardium. The commonest predisposing cardiac lesions are rheumatic or syphilitic endocarditis, and such congenital lesions as a bicuspid aortic valve, a patent intraventricular septum, or a patent ductus arteriosus.

There are two chief clinical types, Acute and Subacute.

Acute Infective Endocarditis is generally a terminal event following pneumonia, osteomyelitis, erysipelas, typhoid fever, bacillus coli infection, septic wounds, gonorrhœa and meningitis. The illness is usually continuous with the infection which precedes it. The organisms most commonly concerned are: streptococcus pyogenes, pneumococcus, staphylococcus, meningococcus, gonococcus, B. typhosus and B. coli.

Symptoms.—There is an exacerbation of the previously existing fever. The patient is more severely ill, perspires profusely and may have rigors. Typhoid fever may be simulated by the onset of a somnolent state or a muttering delirium. If the infection is affecting the mitral or aortic valves, signs of embolism may be present. These may be gross or minute. Gross embolism affects most commonly the spleen, producing pain in the left hypochondrium; the kidney, producing hæmaturia and sometimes lumbar pain; the retinal artery, producing blindness; the cerebral vessels, producing hemiplegia or meningeal symptoms; and the arteries of the extremities,

producing sudden pain in an arm, leg, finger or toe, pulsation then being absent distal to the block. More rarely, coronary embolism, producing sudden death, and mesenteric embolism, producing acute intestinal pain, bleeding and meteorism, may occur. Minute embolism produces a petechial rash, subconjunctival and retinal hæmorrhages, and hæmorrhages into the muscle of the hands and feet, producing localised red tender swellings known as Osler's nodes. Rarely the lesion is on the right side of the heart producing periodic attacks of cough, pleurisy and sometimes hæmoptysis, due to small pulmonary emboli.

Signs.—The fever is generally of the hectic variety and the pulse rate is raised. The heart is more or less enlarged, and the valvular murmur of the attacked valve is present. The enlarged spleen may be palpable. Blood cultures are generally positive, but several may have to be taken before a positive culture is obtained.

Diagnosis.—Septicæmia without endocarditis, typhoid fever, tuberculosis, meningitis, acute leukemia and pernicious anæmia, often have to be considered in the differential diagnosis.

Prognosis.—Acute malignant endocarditis may be curable by penicillin (see p. 70).

Subacute Infective Endocarditis (Synonym: Subacute Bacterial Endocarditis).—In this form of the disease also a previously existing cardiac lesion is attacked by a secondary infection. The cardiac lesion is as described above, but the infecting organism is in the great majority of cases a normal inhabitant of the mouth or intestine, the streptococcus viridans. Rarely the influenza bacillus is present.

Symptoms.—The four cardinal findings are fever, a cardiac lesion, signs of embolism and a positive blood culture. The fever may continue for months (Fig. 125), the only complaint of the patient being undue lassitude, marked sweating at night, and transient pains in the joints or fingers. But sooner or later a doctor is called in, and in addition to the fever a definite mitral or aortic valvular lesion is found. Embolism may produce a more dramatic picture, producing the symptoms described under the acute form of the disease. An earthy pallor with moderate anæmia is characteristic. Particular search should be made for the petechial emboli into the conjunctiva, retina and skin. Clubbing of the fingers is often found. Rigors are less common than in the acute form. The patient is at first not incapacitated to any severe extent, and may have continued his usual occupation.

Diagnosis.—The diagnosis is generally comparatively easy, but cases occur where owing to the absence at first of signs of embolism, or owing to difficulty in obtaining a positive blood culture, it may remain in doubt for a period. Fever and a cardiac lesion occur in *acute* or *subacute rheumatism*, and pains in the joints are common, but a positive blood culture is never obtained. The joint lesions of acute rheumatism are brought to an end by full doses of salicylate, those of subacute bacterial endocarditis are unaffected. A cardiac lesion and systemic embolism occur in advanced mitral stenosis, but here the patient is afebrile, and auricular fibrillation is generally present; this irregularity is very rare in malignant endocarditis. Continued fever, possibly with a systolic cardiac murmur, may be present in other causes of severe *anæmia*, sometimes with splenic enlargement and petechiæ, but the blood count and

increasing the agony. The sense of suffocation, of bodily discomfort, constriction of the chest, and of impending dissolution is extreme. The attack lasts for a few seconds to a few minutes, and is liable to be aggravated if the patient ventures to move from the position which he may have assumed. (2) The heart's action, when examined during an attack, is sometimes found to be unaltered, though palpitation may be complained of. Electrocardiographic examination often shows some irregularity, usually due to premature beats. The blood pressure during the attack is raised. The pulse rate is in some cases increased. The heart usually shows some enlargement, and often some form of aortic valvular mischief is present (see Etiology below). (3) The mind remains clear throughout. Many attacks are accompanied or succeeded by a profuse flow of urine; others by profuse perspiration. The limbs and other parts which were the seat of pain may afterwards feel "numbed." (4) Patients are usually of the male sex, and over 50 years of age. The disease also appears to affect by preference hypersensitive individuals.

Etiology.—There is often a familial tendency. Coronary atheroma, aortic regurgitation, and aortic syphilis are the most common causes.

Prognosis.—The same considerations enter into the prognosis in spasmodic angina as in angina of effort, and death from coronary thrombosis is always a possibility.

Treatment of spasmodic angina.—The pain is relieved by amyl nitrite or nitroglycerin. Hot whisky and water is sometimes useful, especially at night. Morphia may be necessary. Great attention should be paid to keeping the rooms warm, and the bed well warmed, as many attacks are produced by passing from a warm room to a cold passage, and by getting into a cold bed at night. Good nights must be ensured by the use of bromidia, chloral, or even morphia when necessary. If the cause of the condition is an inadequate oxygen supply, the object of treatment is twofold, namely: (1) to reduce the work of the heart, and (2) to increase the oxygen supply, *i.e.*, the blood supply to the muscle. Between attacks, the treatment is identical with that described for angina of effort.

There are two methods in use for blocking the stimuli in anginal pain, sympathectomy of the middle and lower cervical ganglia, and alcohol injection of the five upper dorsal sympathetic *rami communicantes*. The choice of appropriate cases is a matter for expert opinion, but it can be stated briefly that the chief indications for mechanical interference are: (1) The pain has a coronary origin; (2) syphilis has been excluded; (3) the cardiac function of the patient is otherwise good; (4) gross disease is absent.

§ 52. III. **Coronary Thrombosis.**—In disease of the coronary arteries, as in other damaged vessels, thrombosis is very liable to occur. Moreover, as with thrombosis in other positions, it often takes place when the circulation is slowed, and hence frequently comes on in the night. When it occurs the patient gets a sudden violent attack of pain, usually in the lower part of the chest behind the sternum. It does not radiate so definitely as in angina, lasts on and off for a period of hours or days and is associated with marked restlessness. The main differences between

an attack of angina and coronary thrombosis are indicated and summarised as follows:

TABLE II.

	ANGINA OF EFFORT.	CORONARY THROMBOSIS.
I.	Attack comes on during exercise, cold or emotion. Especially after meals.	Attack comes on when circulation is slowed, especially therefore in the night.
II.	Patient is brought to a standstill by pain, which then soon goes.	Patient is restless, collapsed, sweating and often slightly cyanosed or flushed.
III.	Attacks last a few minutes.	Attacks may last some hours or days.
IV.	Pain referred definitely to left or both arms, throat or epigastrium.	Pain not so diffuse, is substernal, and usually lower down—even epigastric; is often more agonising.
V.	Pain relieved by vaso-dilators.	Pain unaffected by vaso-dilators.
VI.	Arterial B.P. usually rises.	Arterial B.P. falls markedly. Venous pressure very much raised.
VII.	No fever.	Slight fever within 24 hours.
VIII.	No leucocytosis.	Leucocytosis with the fever.
IX.	Erythrocyte sedimentation rate normal.	Erythrocyte S.R. raised.
X.	Friction sounds absent.	Pericardial friction often present after about the fourth day.
XI.	Electrocardiogram. Left-sided preponderance usually present. The tracing is unchanged in shape.	Electrocardiogram always shows a changing QRST (Fig. 27 and § 44).
XII.	Heart sounds clearly audible.	Heart sounds weak.

Diagnosis.—Conditions simulating coronary infarction are pulmonary embolism, perforated peptic ulcer, pneumothorax and gall-stone colic. Pulmonary embolism, if not fatal, may cause sudden pain, dyspnœa, collapse, and cardiac embarrassment. Here the neck veins are greatly distended, the electrocardiogram is like that of posterior coronary infarction except that in pulmonary embolism the "T" wave in the chest lead placed near the right sternal border (CR1) is inverted. The associated cardiac condition is often called "acute cor pulmonale."

Prognosis.—After coronary thrombosis it is improbable that the heart will be as efficient as before, but in certain cases where the myocardium is otherwise healthy, apparent recovery occurs. Points suggesting an unfavourable prognosis are: marked enlargement of the heart, signs of congestive failure and the presence of diabetes. A second thrombosis occurs in 25% of cases. Complications are cardiac irregularities, heart failure, occasionally hemiplegia from intra-ventricular clot, and rupture of the heart.

Treatment of Coronary Thrombosis.—Keep the patient in bed for six weeks at least; give oxygen for cyanosis; give full doses of morphia to relieve the pain and procure rest, and repeat it if necessary. After the second week given diuretin and phenobarbitone. Slow resumption of normal activities is encouraged up to the level of comfort: and mental and physical overstrain must at all times be avoided.

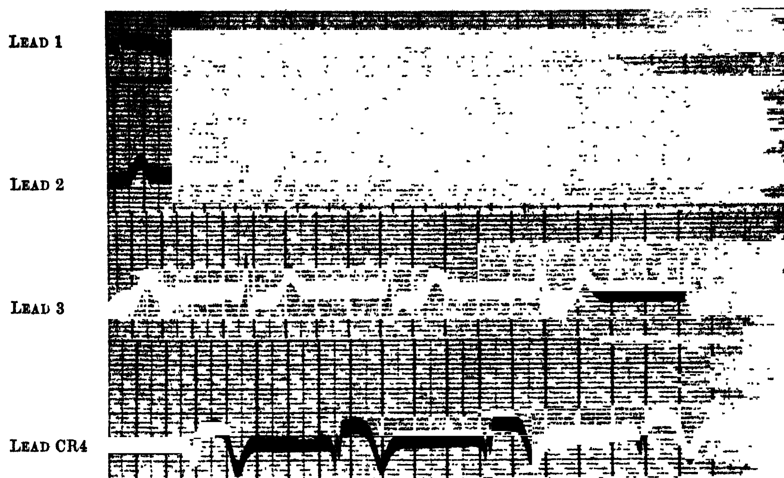


FIG. 25.—ELECTROCARDIOGRAM three days after an acute ANTERIOR CORONARY THROMBOSIS. Note S-T elevation in leads 1 and CR4, and S-T depression in leads 2 and 3.

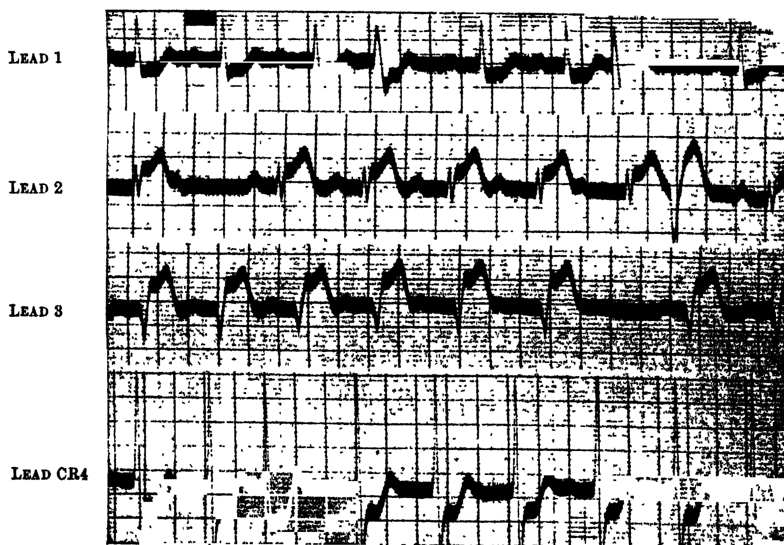


FIG. 26.—ELECTROCARDIOGRAM two days after an acute POSTERIOR CORONARY THROMBOSIS. S-T elevation is well shown in leads 2 and 3, and S-T depression in leads 1 and CR4.

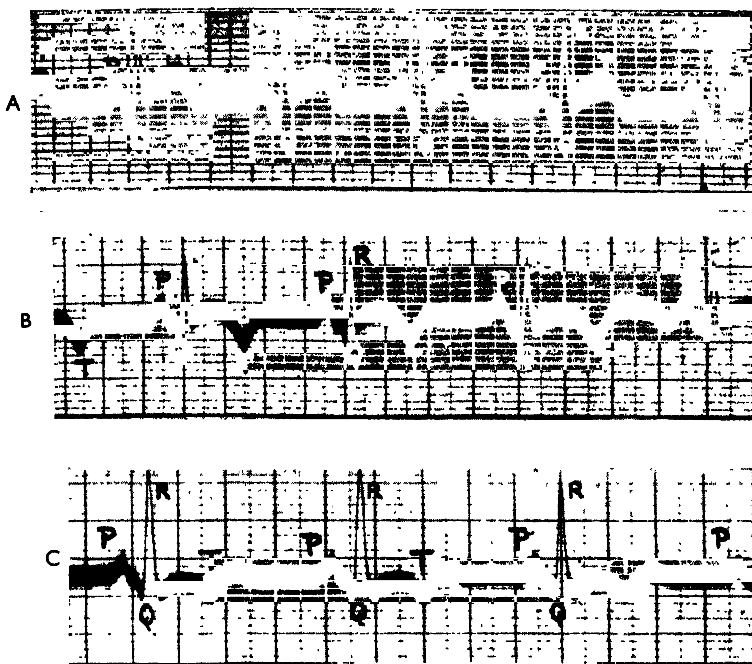


FIG. 27.—CORONARY THROMBOSIS (anterior branch).
All tracings are of lead 1.

A. Three days after coronary thrombosis; note the high "take-off" of the "T" from the "R" wave.
B. Ten days later. Note inversion and spiky shape of "T."
C. Three weeks after thrombosis. "T" is beginning to return to its normal upright shape; it is diphasic. Later, the RST curve may become quite normal again.

§ 53. IV. **Angina Innocens.** Cardiac pain of a functional type is common and is labelled angina innocens or *pseudo-angina*. It must be distinguished from pain due to an organic lesion. The safest criterion is the relation to exercise. If there is never pain at rest, if it is strictly proportional to exercise and only occurs during exertion, it is of organic origin. Other evidences in favour of true angina are: the presence of dyspnœa, of cardiac enlargement, hypertension or aortic disease, and the absence of præcordial tenderness. Evidences in favour of angina innocens are: an increase of pain after exertion—not during it, the presence of palpitation, giddiness and fainting attacks, the presence of præcordial hyperæsthesia, evidence of an unstable nervous system or a history of nervous breakdown. In angina innocens there is no cardiac enlargement, and no evidence of cardiac disease clinically, electrocardiographically, or by X-ray. However, there is an exception to this rule in cases of mitral stenosis with pain; these patients have pain of the angina innocens type.

There is a second type of pain occasionally found in patients with angina innocens. It is identical with that described by Gowers (Vaso-vagal

attacks of Gowers, see § 720). These attacks have a sudden onset, there may be a sense of impending death; severe præcordial pain may radiate to the arms, or tingling in the arms may be complained of. Marked pallor and bradycardia are the most striking signs. The patient generally faints.

Prognosis. In angina innocens, though the symptoms may persist for years, there is no danger to life and recovery always occurs in time. The vaso-vagal attacks have no bad prognostic significance.

Treatment of angina innocens consists in reassuring the patient, in removing obvious physical abnormalities such as marked dental or tonsillar sepsis, in attempting to adjust any psychological stresses, and in allowing a gradual increase of exercise taken by the patient. Treatment in a special psycho-therapeutic centre is often advantageous. The best drugs are phenobarbitone and bromides.

GROUP C. We now consider those conditions in which examination reveals **Enlargement** of the **Area of Præcordial Dulness.**

a. If there is a history of acute onset, and there is PYREXIA, the condition is due to ACUTE PERICARDITIS, which is fully described in Group I, § 46, or to DILATATION secondary to a febrile process.

b. If there is no Pyrexia, the enlargement may be due to

I. Cardiac Hypertrophy.
II. Cardiac Dilatation.
III. Chronic Pericardial Effusion.
IV. Adherent Pericardium.
V. Congenital heart disease (rare).

VI. Aortic Aneurysm and Mediastinal Tumours must be remembered, because their existence is often revealed by finding enlargement of the præcordial dulness, or dulness above, merging into that of the heart.

Chronic conditions which may be, but are NOT NECESSARILY, attended by ENLARGEMENT of the area of præcordial dulness should be borne in mind; their diagnosis may depend mainly on auscultation, and hence they are described under Groups D and E.

Method of Procedure.—It will be remembered that the routine examination of the heart consisted of (1) inspection; (2) palpation; (3) percussion of the præcordial dulness; (4) auscultation; and (5) in any patient in whom cardio-vascular disease is suspected, radiography and electrocardiography can give valuable help. The student should bear in mind the various *fallacies* which may give a false impression of cardiac enlargement, and also those conditions, such as emphysema, which obscure an enlarged heart (§ 40).

I. *The* APEX BEAT *is* BELOW *its normal position; the impulse is* FORCIBLE *and heaving; on auscultation, the first sound is* DULL *and prolonged. There is* HYPERTROPHY OF THE HEART.

§ **54. Hypertrophy of the Heart,** and the dilatation which not infrequently accompanies or follows it, are certainly the commonest conditions which produce an increased area of præcordial dulness.

Cardiac Hypertrophy is an increase of the muscular substance of the heart, and its weight, which is normally about 8½ ounces in women and 9½ ounces in men, may be increased to 10 or 12 ounces, and on rare occasions to 15 or 20 ounces. Its *signs* are as follows: (1) The increase in the præcordial dulness is downwards and outwards if the left ventricle be hypertrophied, outwards only if the right ventricle; (2) the apex beats below and outside its normal position; (3) the impulse is unduly forcible, heaving, or thrusting, the thrust of the hypertrophied right ventricle being generally felt in the epigastric notch; (4) on auscultation, the first sound is loud and prolonged. The pulse is firm, strong and bounding.

Symptoms may be altogether absent if the hypertrophy accurately compensates for the obstruction in the circulation which has caused the hypertrophy. The patient may, indeed, be unaware of any cardiac disorder.

Etiology.—Hypertrophy is caused by an increase in the work to be performed. The part of the heart which undergoes hypertrophy is that immediately behind the lesion: the signs of such a lesion will be additional to those caused by hypertrophy. Thus, there will be three sets of signs: (*a*) Signs of hypertrophy of the heart as a whole; (*b*) signs of enlargement of the chamber specially involved; and (*c*) signs and symptoms of the cause. The following causes will be more readily understood by consulting Fig. 28 (p. 86), and it must be remembered that the enlargement is rarely in actual practice strictly limited to one chamber of the heart.

(*a*) Hypertrophy of the Left Ventricle is indicated by displacement of the apex beat *below* and to the left of its normal position. The apical impulse is strong, sustained and heaving in character. There is also enlargement of the area of cardiac dulness to the left. The pulse is strong unless modified by the presence of a valvular lesion, and the carotids may be seen to pulsate.

Etiology.—Hypertrophy is always secondary, and is proportional to the extent of the causative lesion, provided that compensation has occurred. It is an illustration of the physiological law that increased use leads to increased growth. Hypertrophy of the left ventricle is due to one of the following causes:—hypertension (§ 94), aortic regurgitation or stenosis, mitral regurgitation, healed chronic myocarditis and adherent pericardium. The largest of all hearts are those in which chronic pericarditis with adhesions is present (§ 56, IV). The writer has seen one such case where the heart weighed more than the liver. The commonest cause of marked enlargement is aortic regurgitation. Aortic stenosis produces only a slight degree of enlargement, the whole of which is due to hypertrophy. *Aneurysm of the aortic arch*, if unattended by valvular disease or renal mischief or arterial disease, does not *per se* cause cardiac hypertrophy; if enlargement is present, it is due to an associated aortic regurgitation or hypertension. *Excessive muscular exercise*, whether athletic or laborious, may produce hypertrophy, and in support of this statement it may be mentioned that the normal increase with age is more noticeable in men than in women.

(*b*) Hypertrophy of the Right Ventricle is indicated by enlargement of the area of dulness to the right; and a heaving impulse in the epigastrium. It is the result of resistance to the emptying of the ventricle into the pulmonary vessels. This may occur in:

(i.) *Pulmonary diseases* attended by obstruction in the pulmonary circulation, of which *bronchitis with emphysema* is certainly the most frequent. This condition, a very common one, is identified by a history or evidence of lung mischief (§ 142).

(ii.) *Mitral stenosis* is the next most common cause, and should be borne in mind even in the absence of a presystolic murmur (§ 60).

(iii.) *Mitral Regurgitation* (§ 58).

(iv.) Raised pulmonary pressure from pulmonary arterio-sclerosis, Ayerza's disease (see § 31).

(v.) Congenital pulmonary stenosis.

(*c*) HYPERTROPHY OF THE LEFT AURICLE is always attended by dilatation. It is a difficult condition to recognise with certainty except with X-ray examination (Figs. 22 and 23).

It may arise in *mitral regurgitation*, but its chief cause is *mitral stenosis*. In the latter condition, palpation generally reveals a thrill over the apex, and careful auscultation may detect the presystolic or mid-diastolic murmur (§ 60).

(*d*) HYPERTROPHY OF THE RIGHT AURICLE gives rise to the following physical signs: (i.) Increase of dulness to the right of the sternum; (ii.) powerful jugular pulsation, which polygraphic records prove to be due to forcible auricular contractions.

(*e*) Extreme HYPERTROPHY OF BOTH AURICLES AND VENTRICLES arises in congenital heart disease, but may be confined to the right side. It also occurs with Adherent Pericardium of the external type (§ 56).

All these conditions can easily be verified by X-ray examination (Figs. 22 and 23).

Prognosis and Treatment.—Cardiac Hypertrophy is essentially a compensatory process for some condition which causes obstruction in the circulation. It is Nature's method of compensating for the increased work.

1. *If the cause be removable*, the prognosis is favourable. Treatment in such cases should therefore be directed to the removal of the cause —*e.g.*, high blood pressure.

2. *If the cause be not removable*, the prognosis of the case depends on the avoidance of myocardial failure, which will show itself symptomatically by dyspnœa and physically by dilatation. To accomplish the first, the general health should be improved by general hygienic measures. In order to relieve the heart of part of its work, and to aid the systemic circulation, baths, massage, passive and active movements are of the greatest use (see § 62).

3. The *existence of cardiac hypertrophy* indicates an element of risk to a person's life from one of two factors. In the first place, hypertrophy nearly always indicates that there is obstruction somewhere in the circulation, and this, whatever it be, is in itself an injury to health, and may shorten life. Secondly, it is an indication in many cases of associated arterial disease and hypertension, with the risks of coronary and cerebral disease and of renal failure (§§ 93 and 94).

II. *The* AREA OF DULNESS IS INCREASED; *the position of the* APEX BEAT IS INDEFINITE; *the impulse is diffuse, wavy and slapping; on auscultation, the first sound is short and sharp. The condition is* CARDIAC DILATATION.

§ 55. **Cardiac Dilatation** (an important indication of "Myocardial failure") suggests that the heart is "failing" to keep pace with the demand made upon it, that the reserve power of the muscle wall is becoming spent. Dilatation is the immediate physiological response of the heart to increased work. If increased work continues, hypertrophy normally follows. Physiological dilatation is limited by the pericardium. Strictly speaking, both the dilatation and the symptoms and signs of failure are due to a common cause, the underlying myocardial failure—which is usually due to toxæmia or to anoxæmia.

Myocardial failure, nearly always accompanied by dilatation, generally involves both left and right ventricles. Usually the left side is first affected and its failure later causes the right ventricle to fail also. In rare cases the right ventricle fails alone: orthopnœa is absent in pure right ventricular failure. Heart failure with clinical œdema is called congestive heart failure.

(I) Dilatation of the Left Ventricle indicates some degree of **left ventricular failure.**

Physical Signs.—In the early stages we find: (1) the cardiac impulse by palpation is wavy and diffuse, and is displaced outwards rather than downwards: it may be so feeble as to be hardly perceptible. (2) There is increase in the area of cardiac dulness in a transverse direction to the left. (3) On auscultation the first sound is sharp and clear due to the closure of the mitral valve and the usual muscular element in the sound is largely inaudible. Both first and second sounds are often faint and the period of systolic output shortened. (4) Murmurs may be present from co-existing valvular disease, but a systolic murmur—the "murmur of dilatation"—may be heard apart from actual valvular disease, because the dilated auriculo-ventricular orifice allows a reflux of blood (§ 58, Ia). (5) The pulse may be feeble and rapid. *In the later stages*, when left ventricular failure is more severe, there may appear (1) pulmonary congestion and œdema (*cardiac asthma*). This is most noticeable in the night: it is revealed by orthopnœa, dyspnœa, cough, expectoration of mucus sometimes tinged with blood, or actual hæmoptysis. The physical signs are abundant râles and, sometimes, scattered patches of dulness at one or both bases. (2) *Fœtal rhythm.* In this the systolic and diastolic intervals are almost identical, making it difficult to identify the first and second cardiac sounds. (3) In *delirium cordis* or auricular fibrillation the heart is so rapid and irregular that it becomes difficult to make out the relations of sounds and murmurs. (4) In *gallop rhythm* there is rapidity of action, together with a distinctly reduplicated first or second sound.

Gallop rhythm is a condition in which three heart sounds, instead of two, are heard in each cardiac cycle. *Pathological* causes are (i.) a Bundle-branch lesion (Fig. 38). Owing to the fact that conduction is unequal down each branch of the Bundle of His, the contraction of the two ventricles is slightly asynchronous. The first heart sound at the apex is thus reduplicated. (ii.) A reduplicated first

sound may be present with left ventricular dilatation, generally in association with a failing heart in a patient with hypertension. (iii.) A third heart sound may be produced in the early stage of mitral stenosis by the discharge of blood from the left auricle through the mitral valve in mid-diastole. *Apart from disease*, three heart sounds may occur in two conditions: (i.) A physiological third heart sound heard as a very faint sound in the middle of diastole. (ii.) When the ventricular systole, by mechanical means, produces a sound outside the heart. This exocardial sound generally disappears either during full inspiration or during full expiration.

(II) Dilatation of the Right Ventricle follows left-sided heart failure or is due to obstruction of the blood flow through the lungs. It causes enlargement of the area of cardiac dulness to the right, and often marked epigastric pulsation. Sooner or later the symptoms and signs of **right-sided heart failure occur :**

(i.) A *bruit* over the tricuspid orifice is sometimes heard (see p. 89).

(ii.) *Fulness* and *pulsation* in the neck veins, due to tricuspid regurgitation.

(iii.) *Dropsy*, which indicates congestion of the whole venous system. Cardiac dropsy *starts and predominates in the legs* or *the back*, whichever may happen to have been in the most dependent position. The skin is tense, and is very liable to be attacked by erythematous, erysipelatous, and inflammatory conditions (cellulitis, ulcer, etc.). *Ascites* in varying amount is generally present. It is often an early and prominent sign in mitral stenosis. *Cyanosis* and a general lividity of the surface are consequences of the same venous stasis. A case of mitral disease, therefore, presents a marked contrast to one of aortic disease, where the countenance is often pale.

(iv.) *Engorgement of the liver* is evidenced by pain and tenderness in that region, and later jaundice of the skin and sclerotics. The organ is enlarged, and it may extend to the umbilicus. Sometimes pulsation of the liver may be made out by placing one hand on the epigastrium, and pressing the other beneath the back in the dorsal region. In cases of *dropsy with albuminuria*, when in doubt whether the dropsy is of renal or cardiac origin, hepatic enlargement is a valuable diagnostic aid; its presence is usual in cardiac cases.

(v.) *Indigestion*, want of appetite, a sense of discomfort in the stomach after meals, nausea or actual vomiting, with streaks of blood, indicate congestion of that organ.

(vi.) *Albuminuria*, with high-coloured scanty urine of high specific gravity (and possibly casts and some blood in long-standing cases), point to congestion of the kidney.

The *Causes of Cardiac Dilatation* are of extreme importance as bearing on the prognosis and treatment of cardiac valvular disease and other circulatory disorders. The *clinical conditions* which produce dilatation are practically identical with those which produce cardiac hypertrophy (§ 54), when they are persistent and *are associated with some condition*

which impairs the nutrition of the heart (see Etiology). Undoubtedly the commonest causes of cardiac hypertrophy with dilatation are CARDIAC VALVULAR DISEASE, HYPERTENSION WITH MYOCARDIAL DEGENERATION (producing left-sided dilatation), and CHRONIC BRONCHITIS WITH EMPHYSEMA (producing right-sided dilatation). These are the possibilities which should first suggest themselves to the mind in a case where dilatation is evident.

Etiology.—Pathologically speaking, Dilatation falls under two heads:—Compensatory Dilatation and Dilatation due to failure.

Examples of *compensatory dilatation* are found in aortic and mitral regurgitation. In each case the left ventricle has to accommodate first the normal amount required for each normal systole, and secondly the quantity which will regurgitate through the damaged valves. *Dilatation due to failure* occurs whenever the heart muscle is sufficiently poisoned, or becomes anoxæmic. The commonest causes of this myocardial failure are: (*a*) Infections such as acute rheumatism, diphtheria, typhoid fever, septicæmia, pneumonia, influenza, tuberculosis, malaria and possibly syphilis. (*b*) Poisons such as arsenic and alcohol. (*c*) Metabolic states such as hyper- and hypo-thyroidism, beri-beri, scurvy.

The commonest causes of cardiac *anoxæmia* are: (*a*) Severe anæmia, of the primary or secondary type; (*b*) Coronary artery disease.

Any *sudden strain on an apparently normal heart* may produce acute dilatation. But no really normal heart will dilate under strain. In the cases where this occurs, some mild infection or other lesion is present. Thus severe muscular exertion in athletes or soldiers who have not had any previous training may seem to cause the heart to fail. Instances are met with in hill-climbers who are "out of form," and others who take sudden and unaccustomed exercise. Breathlessness may date from incidents of this kind, from which the patient may never, or only with difficulty, recover. Rest and gentle exercise are indicated. Prolonged fatigue may similarly overtax the heart muscle if it is already diseased.

The *Prognosis* and *Treatment* of Cardiac Dilatation are fully dealt with under Cardiac Valvular Disease (§§ 61 and 62).

III. *The area of dulness is* INCREASED UPWARDS, *and its shape is pyramidal, with the point upwards; the apex beat is raised, and the impulse is weak and undulatory; on auscultation, the sounds are feeble. The signs are of long standing. The disease is* CHRONIC PERICARDIAL EFFUSION.

§ 56. In **Chronic pericardial effusion** (Synonym: Hydropericardium), the *Symptoms* are due to increasing intrapericardial pressure: there is increasing respiratory distress. *Signs:* (1) There is congestion of the neck veins. (2) The shape of the dulness is very characteristic, being pyramidal, with the narrow end upwards. (3) The apex of the heart is *raised*, and *to the right* of its normal position, because the roof of the pericardium is raised by the fluid, and takes the heart with it. (4) For the same reason, the left margin of præcordial dulness extends *beyond* the apex beat. (5) On auscultation, the heart sounds are distant and muffled. There may be irregularity and rapidity of the pulse, and difficulty of breathing from the impeded action of the heart and lungs. (6) Pulsus paradoxus may be present; this is an inspiratory diminution in pulse volume. (7) The physical signs at the base of the left lung are identical with those described with acute pericarditis (§ 46).

Etiology.—Chronic effusion into the pericardium may originate in one of three ways. (1) As the result of Acute Pericarditis (§ 46), of which a history is generally obtainable, but by no means always (see Latent Pericarditis, § 48). (2) True hydropericardium seldom occurs excepting as part of a general dropsy due to renal or cardiac disease, and therefore the urine should be carefully examined. In these circumstances

dyspnœa is the most obvious symptom complained of and an X-ray examination should always be made if there is any doubt as to the existence of fluid. (3) If hydropericardium be not preceded by pericarditis, or be not part of a general dropsy, new growth or tubercle should always be suspected.

The *Diagnosis* from Cardiac Dilatation should be readily accomplished by the shape of the dulness, which is square instead of pyramidal in dilatation; and by the heart sounds, which are clear and sharp in dilatation, muffled in effusion. X-ray examination is of assistance. Pleural effusion is attended by pulmonary symptoms.

The *Prognosis* of hydropericardium depends on its causation, being favourable in Cause 1, adding only a little to the gravity of the primary malady in 2, and being necessarily fatal in malignancy: tuberculous cases often recover.

Treatment.—The treatment of inflammatory effusion is dealt with in § 46. If part of a general dropsy, our efforts must be directed to this. Counter-irritants are sometimes useful. Paracentesis should not be considered unless the cardiac embarrassment is urgent.

IV. **Adherent Pericardium** may exist in three forms: (i.) *Constrictive*, in which the visceral and parietal layers are adherent in varying degree. This condition may be latent, or may produce the signs of venous obstruction as a result of the presence of fibrous or calcareous interference with the venous filling of the heart. The heart may be constricted rather than enlarged. Ascites and peripheral œdema are the commonest signs. Pericardectomy may produce a striking cure. If the pericardial fibrosis or calcification does not obstruct the auricular filling there may be no signs or symptoms. (ii.) *External* adhesions may bind the pericardium to all the surrounding structures. The condition is a sequel to pericardial effusion, with softening and dilatation of the sac. In many cases some degree of internal adhesion is also present. The condition may cause congestive failure; the heart is dilated, and is so tethered that it cannot return to its normal size. The signs are many, but not very reliable. They are (1) a systolic tug at the apex; (2) fixity of the cardiac apex during respiration and with change of position; (3) systolic recession around the apex and along the attachments of the diaphragm, either in front along the lower costal border, or behind in the ninth and tenth spaces (Broadbent's sign); (4) signs of hypertrophy, greater than can be accounted for by the severity of any valve disease which may be present; (5) pulsus paradoxus, or diminution or disappearance of the radial pulse during inspiration of normal depth; (6) signs of incompetence of auriculo-ventricular valves; (7) a diastolic shock in the veins of the neck. Rheumatism is the commonest cause of the condition, and mitral stenosis is often present. The subjects of this condition seem unable to acquire more than a slight degree of "compensation"; slight improvement is soon followed by more complete exhaustion of their cardiac reserve. No permanent improvement is to be expected. Cardiolysis or removal of ribs has been successfully performed for this condition.

(iii.) PICK'S DISEASE or chronic adhesive mediastino-pericarditis. In this condition a diffuse mediastinitis is associated with effusion into one or more of the serous cavities of the body. The *etiology* is obscure, but in some cases tuberculosis is present. The *prognosis* in Pick's disease is not good. Although remissions often occur, and may last for some months, inflammation and effusion recur in one serous cavity or another. *Treatment* consists in removing the fluid by paracentesis, whenever it becomes physically embarrassing, and in nursing the patient in conditions as pleasant and as airy as possible. Diet should be full and well balanced, with an adequate ration of milk, cream and butter.

V. IN CONGENITAL HEART DISEASE the enlarged area of præcordial dulness varies with the lesion. There are usually characteristic murmurs. For the differential signs of this condition, see § 59.

VI. In ANEURYSM of the first part of the aortic arch, the upper part

of the dull area is increased transversely, and there is dulness over the sternum. Auscultation may reveal a systolic or diastolic murmur and a loud, sharp, ringing aortic second sound (see § 80).

GROUP D. We now turn to those *conditions in which there is found an* **alteration of the heart sounds, or a murmur.** It is well to bear in mind several fallacies referred to on pp. 48, 53, 87, 88, 89 and 95.

In the absence of these, if *the heart sounds are* FAINT, the disease is MYOCARDIAL DEGENERATION, unless a thick chest wall or marked emphysema is present.

The second sound is ACCENTUATED *or* DOUBLE. This may be due to (i.) High blood pressure; (ii.) aortic aneurysm; (iii.) pulmonary stasis in mitral disease.

The first sound at the apex is unduly LOUD. This may be due to: (i.) Nervousness of the patient; (ii.) mitral stenosis; (iii.) a thin chest wall; (iv.) hypertrophy of the ventricle; (v.) adjacent air-containing cavity acting as a resonator.

The first sound is unduly SHORT. This may be due to: (i.) Dilatation of the ventricles due to myocardial degeneration or toxæmia; (ii.) rapidity of the pulse; (iii.) incomplete filling of the left ventricle (mitral stenosis), resulting in an unduly hurried emptying.

I. *On auscultation the* HEART SOUNDS ARE VERY FEEBLE; *the impulse is weak and slapping. No murmur is heard.* MYOCARDIAL DEGENERATION *may be suspected.*

§ 57. **Myocardial Degeneration.**—Previously sometimes called Fatty Heart and Fibroid Heart.

The changes in the heart muscle may cause: (1) failure of tonicity, causing dilatation; (2) failure of contractility, causing circulatory inadequacy; (3) changes in the primitive musculature, causing irregularities of rhythm (see § 63). The disease should be suspected when cardiac symptoms arise in a patient with cardiac enlargement, and when other diseases (such as of the valves, or hypertension) can be excluded.

Symptoms and Signs.—The disease will be suspected when the heart is enlarged and there is evidence of arterial disease elsewhere. On exertion there is: (i.) undue dyspnœa; (ii.) marked lassitude; (iii.) palpitation; or (iv.) a tight feeling across the chest which goes on rest; (v.) cardiac irregularities (§ 63) such as premature beats may be present at rest, and are often exaggerated by exercise. Auricular fibrillation, and occasionally auricular flutter occur. When the disease is more advanced (vi.) dyspnœa may be present at night ("cardiac asthma"); (vii.) the cardiac impulse is feeble, the heart sounds poor and the radial pulse weak; (viii.) congestive failure may occur. Myocardial degeneration usually affects both left and right ventricles in fairly equal degree. If the left ventricle is the more diseased, pulmonary congestion and œdema are prominent. When emphysema is a causative factor, the failure chiefly involves the right ventricle.

The degree of degeneration is difficult to estimate clinically. Guidance

on this point may be obtained from (1) Estimation of the amount of exercise which can be carried out without distress, such as pain, dyspnœa, palpitation. (2) The cardiac enlargement is always detectable radiologically, or clinically. (3) The character of the heart sounds: especially shortening of the first sound. (4) The systolic, and to a much less extent the diastolic, blood pressures fall, from ventricular weakness; this produces a diminution in pulse pressure. (5) Electrocardiographic changes, such as flattening or inversion of the "T" waves in leads 1 and 2, or the presence of a bundle-branch lesion, or R–T deviation due to associated coronary disease, are common.

Causes.—Myocardial degeneration is a consequence of interference with the nutrition of the heart wall. This in the majority of cases is the result of coronary disease associated with a progressive interstitial fibrosis. The fibrosis and the fatty degeneration are part and parcel of the same process.

The Prognosis is uncertain. The earlier stages of the malady are insidious, so that by the time pronounced symptoms appear the mischief may be irreparable. Some patients survive for years: those with marked Cheyne-Stokes' respiration, or pulsus alternans die sooner. In the early stages of cardiac degeneration plenty of fresh air, exercise and good sleep are essential for increasing the reserve power of the unaffected muscle fibres, and if the patient responds to this treatment he may live for many years (Mackenzie). Prognosis and treatment are discussed more fully in §§ 61, 62.

While auscultating the heart three questions should be in the physician's mind: (1) What is the character of the first sound? (2) What is the character of the second sound? (3) Is a murmur present?

GROUP D. II. *A* **Murmur** *is present.* Its source may be:

(1) Endocardial—Endocarditis of valve or wall; narrowing or dilatation of orifice of valves; congenital abnormalities.

(2) Atonicity murmur due to muscular stretching of a valve ring.

(3) Cardio-pulmonary, Cardio-respiratory (§ 59).

(4) Pericardial—friction of Pericarditis; and see §§ 46, 49.

(5) Functional and Hæmic murmurs (see §§ 42 and 535).

The chief points to be considered in diagnosing the *source of a murmur* are given in § 42 and § 49 (Table I).

§ 58. Chronic Endocarditis—Cardiac Valvular Disease—Cardiac murmurs.—Disease of the valves of the heart is revealed on auscultation by the presence of a bruit or murmur, which is added to, or replaces, a normal heart sound.

Method of Procedure. Five features must be carefully investigated in any murmur: TIME OF OCCURRENCE. Whether it REPLACES or merely ACCOMPANIES the first or second sound; POSITION of maximum intensity; direction in which it is CONDUCTED; and CHARACTER. The last named is relatively least important. In order to be quite sure of the time of a bruit, it is wise to place the thumb on the carotid artery while auscultating the chest.

The characters of PERICARDIAL MURMURS have already been given (§ 46); and their diagnosis from endocardial murmurs (Table I, p. 67).

Valvular disease is most commonly due in early life to endocarditis (acute or chronic), and in older persons, to chronic degenerative change. The effect is a thickening or puckering of the valves and ring, which results in one or both of two conditions: (*a*) *Stenosis—i.e.*, a narrowing (*στενοω*, to contract) of the orifice, which prevents the blood flowing freely through it; or (*b*) *Regurgitation*, in which the valves are incompetent and allow a reflux of the blood to take place from imperfect meeting and closure of the cusps. The remote effect of these two conditions is practically the same—viz., a retardation or obstruction to the circulation of blood through that orifice.

It simplifies diagnosis very much that cardio-valvular disease arising *after* birth is practically *confined to the left side of the heart—i.e.*, to the mitral and aortic orifices. Thus it happens that there are four principal valvular lesions—MITRAL REGURGITATION, MITRAL STENOSIS, AORTIC REGURGITATION, and AORTIC STENOSIS.

TABLE III.—DIFFERENTIATION OF CARDIAC VALVULAR DISEASES.

			Auscultation.	Pulse.	Other Symptoms special to the Disease.	
C.V.D.	**Mitral** (apical murmurs).	*Regurgitation.*	*Systolic* murmur conducted into axilla.	Usually regular.		
		Stenosis.	*Presystolic* murmur localised to apex beat area.	Small, moderately firm; very irregular with onset of auricular fibrillation.	Dropsy, enlarged liver and ascites, etc., Hæmoptysis; emboli,	with signs of congestion of organs.
	Aortic (basal murmurs).	*Regurgitation.*	*Diastolic* murmur conducted down sternum.	"Water-hammer," rapid and compressible.	Throbbing of arteries of neck,	with symptoms of cerebral anæmia and anginal attacks.
		Stenosis.[1]	*Systolic* murmur conducted into vessels of the neck.	Slow, regular, small and hard.	No special symptoms.	

The student should study Fig. 10, p. 50, so as thoroughly to comprehend the various events which occur during one complete cardiac cycle. He should also bear in mind that the left side of the heart is behind the right, and that the left ventricle comes nearest to the surface only at the apex, immediately behind or just below the fifth rib (Figs. 11 and 12, pp. 51 and 52). He should also remember that a cardiac murmur is not produced *in* a diseased orifice, but by the eddies in the blood-stream beyond, and is conducted in the direction of the stream of blood which is causing the murmur. For these reasons a murmur is not usually heard

[1] Real aortic stenosis is somewhat rare, but atheromatous roughening is common.

loudest directly over the orifice diseased. The student should also consult the diagram of the circulation (Fig. 28).



FIG. 28.—Scheme of the Circulation of the Blood.—The superior and inferior venæ cavæ (6) bring the blood back from the organs and tissues into the right auricle (1). Thence it passes into the right ventricle (2), through the pulmonary artery (7) into the lungs. Returning from the lungs by the pulmonary veins (9), it passes through the left auricle (3) and left ventricle (4), and is distributed by means of the aorta (5, 5) and the carotids (8) to the organs and tissues of the body. Notice that the blood from the stomach and intestines passes through the liver before joining the general circulation. (From Huxley's "Physiology," modified.)

Diagnosis of Cardiac Murmurs.—The first thing to determine is whether a given murmur is related to the first or second sound of the heart—*i.e.*, whether its time is systolic or diastolic—and this will form a convenient basis of classification of cardiac murmurs.

A. **Systolic Murmurs** [1]—*i.e.*, bruits added to or replacing the first sound—may be produced by the following causes, which are mentioned more or less in order of frequency: mitral regurgitation, hæmic and functional conditions (see § 42, and Anæmia, § 535), aortic stenosis, dilatation of the aortic ring, congenital lesions, and tricuspid regurgitation: pulmonary stenosis, patent interventricular septum and patent ductus arteriosus.

I. In **Mitral Regurgitation** the systolic murmur is characterised by (i.) being loudest at the apex; (ii.) being conducted to the axilla, and often audible behind, at the angle of the scapula. When regurgitation is marked, the apex is displaced downwards and outwards owing to the hypertrophy and dilatation of the left ventricle. There is accentuation of the second sound in the pulmonary area, due to congestion in the pulmonary circulation. The pulse is soft, there is a characteristic florid physiognomy, failure occurs late.

The ultimate mechanical effect of Mitral Regurgitation upon the heart and lungs is as follows: (1) owing to the reflux of blood from the ventricle during ventricular systole the auricle becomes dilated.

[1] The term systole is used by clinicians to designate ventricular, not auricular, contraction.

(2) In order to drive on the increased volume of its contents the auricle hypertrophies. (3) The effect of a hypertrophied auricle driving an increased volume of blood into a flaccid ventricle at each auricular systole is to produce dilatation of the ventricle. (4) When the power of the auricle begins to fail it is unable to empty itself properly, and there is difficulty in the free passage of blood from the pulmonary veins. Thus pulmonary blood stasis tends to occur. (5) To overcome this stasis it is necessary for the right ventricle to hypertrophy. In cases of failure, right-sided dilatation supervenes, often with the onset of tricuspid incompetence. (6) So that in cases of Mitral Regurgitation there may occur: (*a*) dilatation and hypertrophy of left auricle and ventricle; (*b*) pulmonary congestion; and (*c*) dilatation and hypertrophy of the right auricle and ventricle. These changes are less marked and develop more slowly in

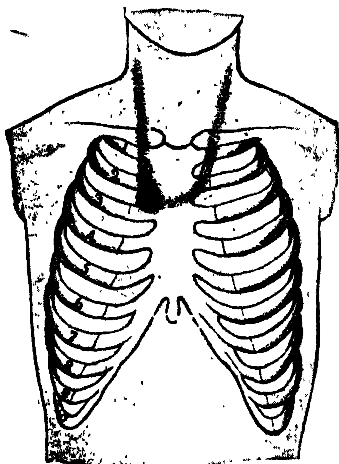


FIG. 29.—The systolic murmur of **aortic stenosis.** Depth of shading indicates intensity of murmur.

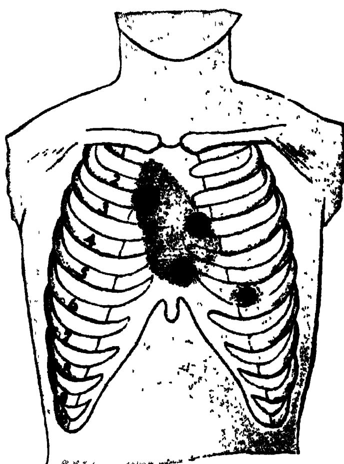


FIG. 30.—The diastolic murmur of **aortic regurgitation.** Depth of shading indicates intensity of murmur.

mitral regurgitation than they do in mitral stenosis, for in the latter condition the left auricle is less able to empty, and is kept in a state of more persistent tension. In mitral regurgitation the outlook is relatively good.

I*a*. A MURMUR OF DILATATION (**Atonicity M.**), systolic in time, having all the above characters, and, like it, due to mitral regurgitation, may occur without definite disease of the valve, when the *left ventricle becomes dilated*, and the muscular ring around the valve *fails to complete* the closure of the mitral valve. This condition is especially apt to occur (i.) in the stage when dilatation of the left ventricle supervenes on hypertrophy, (ii.) in anæmia, and (iii.) in acute myocardial disease, *e.g.*, diphtheria and acute rheumatism.

II. **Aortic Stenosis** is another lesion producing a systolic bruit. The same bruit is produced by endocarditis or atheroma of the aortic orifice.

True narrowing of the aortic ring should not be diagnosed unless five

signs are present: (i.) a systolic bruit in the aortic area and conducted into the carotids. It is harsh, sometimes musical, and may be audible at the apex. (ii.) A systolic thrill in the aortic area, second R. interspace, often best felt when the patient sits forward; (iii.) hypertrophy of the left ventricle; (iv.) a slow-rising, well-sustained, small pulse, often anacrotic in character; and (v.) a weak second sound.

General Symptoms are almost wanting in aortic stenosis—other than anginal pain, due to coronary atheroma, pallor or sallowness of the face, and faintness or giddiness.

The detection of aortic stenosis is sometimes as difficult as that of mitral stenosis and the characteristic murmur may be absent. It may then be suspected when the patient, generally an elderly man, presents persistent dyspnœa, bradycardia, nervousness, and occasionally anginal attacks, which are not otherwise accounted for. In true stenosis the second sound is short and not very loud; whereas in cases of high arterial pressure with a systolic murmur the second sound is loud.

III. In Dilatation and in Aneurysm of the commencement of the Aorta a systolic murmur is the most common one heard. The condition is a "relative stenosis," *i.e.*, though the aortic ring is normal in diameter it is small as compared with the diameter of the enlarged aorta. A peculiar ringing character of the aortic second sound is the most constant cardiac physical sign (§ 80).

§ 59. IV. **Congenital Heart Disease** is comparatively rare.

There are two groups—the cases without cyanosis and those with cyanosis. Those *without cyanosis* include bicuspid aortic valve defect, imperfect ventricular septum, and patent ductus arteriosus. Those *with cyanosis* that survive generally have multiple defects which usually include pulmonary stenosis, or other abnormality in the size or position of the pulmonary artery. Patency of the inter-auricular septum (atrial septum defect) does not usually cause cyanosis, but does so when the left ventricle fails later in life.

(1) Bicuspid aortic valve is not possible to diagnose during life, but should be suspected when aortic infective endocarditis develops in a patient whose heart previously was free from murmurs.

(2) Imperfect ventricular septum is characterised by a loud systolic murmur close to the sternum in the third and fourth left intercostal spaces. The murmur is usually accompanied by a thrill. There is usually no marked limitation of the cardiac function.

(3) Patent ductus arteriosus is characterised by slight ventricular hypertrophy, a loud continuous murmur throughout systole and diastole, loudest in systole, and present in the second left interspace near the sternal border. A thrill usually coexists. The diastolic pressure is often lowered. The X-ray shows a typical enlarged pulmonary conus. If infective endocarditis supervenes, cure is possible by ligature of the ductus (§ 50).

(4) Congenital pulmonary stenosis is characterised by dyspnœa, cyanosis, clubbed fingers, and a loud systolic murmur and thrill over the pulmonary base. Polycythæmia is usually present. It is usually found in combination with three other lesions, when the condition is known as Fallot's tetralogy—pulmonary stenosis, interventricular defect, dextroposition of the aorta, and hypertrophy of the right ventricle. Surgical anastomosis of the subclavian to the pulmonary artery (Blaylock's operation) greatly benefits some cases.

(5) In atrial septum defect the whole heart is enlarged, especially the pulmonary conus, and the pulmonary arteries are enormous and are readily visible in the X-ray picture. A systolic murmur and sometimes a thrill are present over the pulmonary area. Cyanosis is absent at rest, but may appear during exertion: it may become permanent when failure sets in.

Prognosis.—A congenital lesion may remain latent for years, though few cases survive to middle age. The acyanotic cases often die of a superadded septic endocarditis; the cyanotic group develop pneumonia or tuberculosis. The prognosis is best if there is no sign of pulmonary stenosis, no clubbing of the fingers and only polycythæmia. In childhood fatal bronchitis and broncho-pneumonia are common. The prognosis is serious in proportion to the degree of dyspnœa and cyanosis, pointing to deficient aëration of the blood, and in proportion to the other symptoms of "cardiac failure" (§ 61).

The *Treatment* is the same as that of Cardiac Dilatation (§§ 55, 62).

V. Tricuspid Regurgitation takes place when that orifice is diseased or dilated. Some maintain that if the valve be healthy, though dilated, no bruit can be heard, but it is certain that in cases of confirmed bronchitis a murmur is often present which comes and goes under treatment, and which is not found to be attended with any marked changes in the tricuspid valve after death. The murmur is characterised by (i.) being heard best at the tricuspid area—*i.e.*, on the left side of the lower part of the sternum; (ii.) it may be heard as far out as the right nipple; (iii.) the pulse is of low tension, often irregular; (iv.) owing to the accompanying hypertrophy or dilatation of the right auricle, the area of cardiac dulness extends to the right of the sternum.

General Symptoms, as above indicated (p. 80), result from tricuspid regurgitation. By far the commonest cause is Chronic Bronchitis, which thus presents a clinical picture readily recognised.

VI. Cardio-pulmonary or Cardio-respiratory Murmurs are fairly common, and are probably produced by the expulsion of air from the adjacent lung tissue by the movements of the heart. They do not indicate any cardiac lesion, and the lung is usually healthy. They are heard in various parts of the antero-lateral region of the chest. They have a blowing, whiffing, or "sipping" character, are usually systolic in time, and in rare cases double, though the systolic element is always loudest. Often they are not loudest at the apex, and come rather between the two sounds than with the first sound. A common variety is audible at the apex, getting louder as it is conducted into the axilla, and is only heard during inspiration. Sometimes they disappear when the patient alters his position or stands up. When he stops breathing, they may be weakened, abolished, or unaltered. These murmurs have no pathological significance.

VII. The Exocardial Murmur is possibly due to a localised thickening of the visceral pericardium. Usually it is unattended by symptoms, but it may be of importance clinically, for it is apt to be mistaken for valvular disease. The exocardial murmur (based on twenty-three observations, verified by autopsy) is generally a prolonged rough bruit, systolic in time, though occasionally double; it is *strictly localised* to a circle of 1 or 1½ inches radius, whose centre is situated in the third left interspace, close to the sternum, which is also its position of maximum intensity. Another important feature is that *at one time it is very rough and loud*, and a day or so later it may have completely disappeared. These features, and the absence of the concomitant symptoms of cardiac valvular disease, or of anæmia, enable us to differentiate the exocardial murmur from other conditions. It was found more often in hypertrophied hearts than in those of normal size. The condition is more frequently met with in adult or advanced life. A history of pericarditis was obtainable in only one of the twenty-three cases.

VIII. A rare cause is *coarctation of the aorta*; this produces systolic murmurs in the enlarged superficial arteries; a systolic murmur produced over the left internal mammary artery may be confused with a cardiac murmur. Similar murmurs may be heard over enlarged arteries on both sides of the chest and over the back in these cases.

§ **60.** B. **Murmurs** heard in the **diastolic interval** may occupy either (*a*) the first half of that interval, replacing, accompanying, or following the second heart sound (*Diastolic* murmurs); or (*b*) they may occupy the

second half of the interval, preceding and leading up to the first heart sound (*Presystolic* murmurs; see Fig. 31). The latter can be accurately defined and described as auriculo-systolic.

TABLE IV.

Early Diastolic Murmurs	*Late Diastolic Murmurs (Auriculo-systolic)*
Aortic regurgitation	Mitral Stenosis
Mitral Stenosis	Austin Flint Murmur.
Dilatation of the Aortic Ring (Aneurysm)	
Pulmonary regurgitation (rare)	Tricuspid Stenosis (very rare)

I. In **Aortic Regurgitation** the murmur is *diastolic* (Ventricular Diastolic).[1] (i.) The diastolic murmur at the aortic valve (Fig. 30) must be listened for: (*a*) over the lower part and to the left of the sternum. It may be audible as far as the apex and indeed over the whole heart. This murmur may or may not be accompanied by a systolic murmur and is found typically in those cases of aortic regurgitation where the valve is the site of endocarditis or of aortic valve dilatation. (*b*) Over the junction of the second right costal cartilage with the sternum. Here the murmur may be loud, and just beneath the stethoscope. (*c*) Over the third left costal junction. Here the murmur is soft, blowing but distant, never harsh in character. This murmur should be carefully listened for in any case of mitral stenosis where the left ventricle is large. The diastolic aortic murmur of rheumatic valvulitis is best heard along the left sternal margin, that of the syphilitic lesion at the aortic base. (ii.) Owing to the amount of dilatation and hypertrophy of the left ventricle, the apex is displaced downwards and outwards more than in any other form of valvular disease. The increase in the size of the left ventricle is proportional to the amount of blood regurgitating. (iii.) The carotids visibly pulsate. Capillary pulsation is generally present, and is detected by drawing a line across the forehead, or by lightly pressing on the fingernail or on the lips with a glass slide; the alternate blush and pallor due to the pulsation in the capillaries is thus well brought out. The retinal arteries may also show visible pulsation. (iv.) In aortic regurgitation the pulse and the blood-pressure changes are of great diagnostic importance. The pulse wave is very forcible but is ill sustained, and this gives a marked pulsation in the carotids and the vessels of the limbs: it may be sufficiently marked to cause the head to nod to and fro with each heart-beat. At the wrist it is best felt by placing the flat of the hand and fingers just above the patient's wrist: pulsation is then felt in both radial and ulnar arteries and is increased by raising the forearm above the patient's head. Owing to the leakage back through the aortic valves during diastole, the

[1] *Diastolic* murmurs are sometimes spoken of as V.D. murmurs, being produced during the ventricular diastole. Similarly, *presystolic* murmurs are spoken of as A.S. murmurs, being produced during the auricular systole.

volume of blood suddenly expelled into the aorta at the commencement of systole is markedly increased, with a forcible systolic wave in the whole arterial system, and a high systolic pressure : this is ill sustained, as during diastole blood leaves the arteries not only distally, but also by leaking back through the aortic valves, with a correspondingly low diastolic pressure. The pulse therefore becomes " collapsing " or " water-hammer " (" Corrigan pulse ") : the pulse pressure is raised much above normal, and the diastolic pressure is lowered in accurate proportion to the diastolic leak. The systolic pressure in the leg is higher than in the arm, due to vasomotor hypertonus ; the difference between arm and leg pressure is usually taken as a measure of vasomotor reserve. A falling systolic pressure in aortic regurgitation usually means a failing myocardium. There is (v.) a pistol-shot sound over the arteries, and (vi.) a diastolic murmur (Durozier's murmur) over the large arteries.

The *appearance* of a patient with aortic regurgitation is often characteristic. (1) The rheumatic type occurs in children and young adults. There is marked pulsation in the neck, while the whole chest may be seen to pulsate with the heart-beat. The brachial arteries stand out prominently and seem to be definitely hypertrophied. (2) The syphilitic aortic case is usually middle-aged, and often presents signs of premature old age ; the apex beat is slapping, less heaving in character, and more diffuse ; dyspnœa is frequently present, and often signs of arterial degeneration. (3) In the arterio-sclerotic type the lesion is due to the associated atheroma.

As regards the *general symptoms*, pallor is generally stated to be characteristic but in fact this is not true (Lewis). Faintness and giddiness may occur, usually brought on by change of position ; frontal headache and consciousness of the heart's action may be complained of, especially on first lying down at night. Pain may be present in the chest on exertion, but until failure sets in, beyond the consciousness of a forcibly acting heart, the patient is usually fit and able to do a large amount of physical work.

II. **Mitral Stenosis** is characterised by narrowing of the mitral orifice with obstruction to the free passage of blood from the left auricle to the left ventricle. It is always caused by a rheumatic infection.

The *appearance* of the mitral stenotic patient is often characteristic. It is most frequently met in women. The face is more or less pinched, there is a marked malar flush, whilst the tip of the nose, ears and the extremities are cold and blue. Respirations are frequently rapid, and the jugulars are often prominent. The patient has a typically thin face, pale or yellowish, and the jugular veins may be engorged. The " mitral face " is typically a red face, but not infrequently Mitral Stenosis appears with a pale face. The pale Mitral Stenotic should always be regarded with suspicion as, generally speaking, one of the following conditions is present : (*a*) recrudescence of the rheumatic infection ; (*b*) supervening malignant endocarditis ; (*c*) an associated aortic leak, or (*d*) some independent condition such as associated renal disease or anæmia.

Physical Examination.—(1) The *pulse* in Mitral Stenosis may be characteristic, and gives a guide to the condition of the systemic circulation and left side of the heart. The *volume* is small (estimated by "lift" plus duration of wave), due to the diminished output. The *force* is small (estimated by impact against the finger), due to small output from the left ventricle. The *tension* is low (estimated by obliteration force), owing to diminished output and diminished force. The *rhythm* may be regular or irregular; in the latter case the irregularity is due either to ectopic beats or to auricular fibrillation. The *rate* is often rapid (round about 90). (2) The blood pressure is usually low, owing to diminished output and force of the left ventricle. It is sometimes reduced in volume after auricular fibrillation has set in. In older patients the blood pressure may be raised in proportion to the amount of associated arterio-sclerosis. Should mitral stenosis become complicated by aortic regurgitation, the blood pressure tends to rise and the pulse volume and force increase. (3) *Cardiac Signs.*—*Inspection:* Epigastric pulsation is often visible owing to the dilatation and hypertrophy of the right ventricle; the apex beat itself can often be seen inside the nipple line somewhat diffuse in character. *Palpation:* The apex beat is slapping in character and well inside the nipple line. Typically, a presystolic thrill, and in advanced cases a diastolic thrill, is felt at the apex (§ 39). The presystolic thrill may be intermittent and only brought out by exercise, deep breathing, rest or lying on the left side. The pulmonary valve closes so forcibly that it can usually be felt. *Percussion:* The cardiac dulness is increased slightly, if at all. The right cardiac dulness measured from the mid-sternal line is increased. On the left, the cardiac dulness does not extend out to the nipple line unless there is some other associated condition such as mitral or aortic regurgitation, pericarditis, etc. *Auscultation:* In the early stages a faint mid-diastolic murmur is audible, which after exercise, or in the left lateral position, may develop into a typical crescendo presystolic murmur.[1] Later, the typical presystolic murmur appears at rest: then the first sound at the apex is sharp, reduplicated or markedly accentuated. This presystolic murmur, typical of mitral stenosis, is heard only over a limited area, and is not conducted outwards. A long rumbling diastolic murmur is also audible in most cases, especially if the patient lies on the left side (Fig. 31). The second sound at the apex is inaudible in a well-established mitral stenosis, while at the base the pulmonary second sound is markedly accentuated and reduplicated.

General Symptoms.—The commonest symptoms associated with mitral stenosis are: (1) Dyspnœa, at first only on exertion after meals, later after ordinary exertion (*e.g.*, stairs). It then becomes continuous

[1] It may be difficult to differentiate early mitral stenosis from the overacting heart common in excited or neurotic young people. At this stage the X-ray or electrocardiogram may give no help, and the best guide is the character of the first sound. In the excitable heart, although loud and roughened, the first sound is low-pitched; in mitral stenosis the pitch is higher and approximates to that of the second heart sound.

and progressive, so that the patient is unable to lie down at nights (orthopnœa). (2) Palpitation is at first intermittent, occurring after exertion, and is simply of the nature of a physiological tachycardia. Later on it becomes more or less continuous, occurring independently of effort. (3) Cough is a common symptom, induced by exercise or change of position. It may be associated with marked cyanosis or the spitting of blood. The

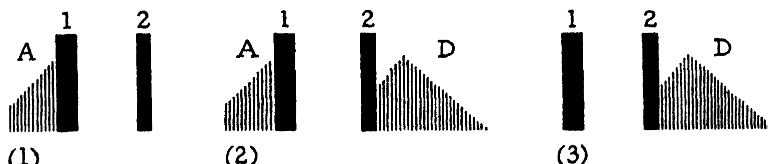


FIG. 31.—In mitral stenosis there are two murmurs, which occupy different parts of the diastolic interval—the presystolic or auriculo-systolic (A), and the diastolic (D) murmurs. The presystolic is present when the auricle is contracting; the diastolic is due to passive blood flow. The presystolic disappears when the auricles fibrillate. The figure shows:—(1) the presystolic murmur; (2) both murmurs; (3) the early diastolic alone as in fibrillation, the presystolic having disappeared. The reduplication of the 2nd sound is omitted for clearness.

underlying cause is congestion of the lungs. A severe type of cough is sometimes met with in embolism or infarction. Symptoms developing later are: (4) auricular fibrillation, flutter, or premature beats; (5) hæmoptysis, embolism, right-sided failure with liver engorgement and ascites are common (§ 55). Embolism, generally cerebral producing hemiplegia, sometimes renal producing hæmaturia, may occur, from the liberation of fibrinous clots formed in the appendix of the dilated left auricle.

In order to understand the progressive variations of the physical signs and symptoms met with in Mitral Stenosis, it is necessary to say a few words about the anatomical changes which develop in this lesion. Broadly speaking, narrowing of the mitral orifice from any cause results in: (*a*) a tendency to dam up the blood in the left auricle, pulmonary and venous circulation in this order, so that the pressure tends to rise; (*b*) there is a reduction in the left ventricular filling, with a reduced size of the left ventricle, a low tension pulse and a slapping first sound. The output and force are thus reduced and the systemic blood pressure falls. The damming of the blood in the pulmonary circulation is responsible for the physical signs met with in the chest, *e.g.*, accentuated pulmonary second sound, basal crepitations, etc., and subsequently it produces engorgement of the systemic veins, enlargement of the liver, peripheral œdema and other manifestations of congestive failure.

The progressive changes which occur in the myocardium in Mitral Stenosis and its actual condition at any phase of the disease can be followed by observing the changes in the electrocardiogram. The first result of Mitral Stenosis is increased auricular work and contraction, and this is reflected in the electrocardiogram by increase in the amplitude of the P or auricular waves, which still however retain their normal form. As the condition progresses, the two auricles become slightly divorced in their action, so that the P waves become flattened, widened and partly divided. Sometimes by the time this has occurred rheumatic fibrosis has invoved the Bundle of His, with impairment of its conductivity, as is shown by an increase in the P–R interval (Fig. 16). Usually also, by now right ventricular "compensation" has occurred, so that the electrocardiogram shows a distinct right axis deviation. The narrowed mitral orifice results in imperfect filling and therefore incomplete stretching of the left ventricle, which consequently contracts poorly (hence the slapping character of the apex beat). The condition continues to progress, the

muscle fails, the rhythm becomes irregular—the first irregularity noticed usually being due to premature beats of auricular origin (Fig. 15). This indicates myocardial hyper-irritability of the auricle, and is often succeeded by a complete irregularity due to auricular fibrillation (Fig. 14) which may rarely be preceded by a state of flutter (Fig. 35). The fibrillation at first is of the coarse variety, but with time it becomes finer and ultimately flat as the muscle gradually degenerates and its co-ordination and contractibility become more impaired. Definite heart block is not uncommon.

III. In Aortic Aneurysm a *diastolic* murmur is sometimes heard if the aortic ring shares in the dilatation of the aorta.

IV. An Austin Flint murmur is a presystolic apical murmur occasionally heard with aortic regurgitation. It is diagnosed from that due to mitral stenosis by its not being followed by an accentuated first sound, by the position of the cardiac impulse, and by the absence of the other signs of mitral stenosis.

V. Regurgitation through the pulmonary artery is met with very rarely; it may be produced by congenital malformation of the heart.

VI. Tricuspid Stenosis is a very rare condition, but it is occasionally met with in young women, and is recognised by (i.) a presystolic murmur, heard loudest over the fifth right costal cartilage, close to the sternum. (ii.) Dropsy is an early effect, but in other respects the consequences are the same as those of regurgitation through this orifice. Dyspnœa is less than the degree of venous and hepatic distension would seem to warrant. Orthopnœa is absent.

Fallacies in the Diagnosis of Diastolic Murmurs.—1. A diastolic murmur due to *aortic regurgitation* may be heard at the *apex*. It must not be mistaken for that of mitral stenosis. In addition to the fact that the aortic murmur is heard louder at the base than at the apex, it has a rushing character, whereas a mitral diastolic murmur is low-pitched and rumbling, and the character of the pulse and the blood pressure is different.

2. *Mitral stenosis* is sometimes hard to detect in the stage of auricular fibrillation, when the characteristic murmur may be *altogether absent*. It may, then, be strongly suspected when there is—(i.) a loud, clear, sharp first sound at the apex, with marked accentuation of the pulmonary second sound; or (ii.) hypertrophy of the right ventricle, chronic pulmonary catarrh, and hæmoptysis, especially if the second sound is reduplicated.

C. **Double Murmurs** may be produced by a combination of any of the above systolic and diastolic murmurs.

(*a*) Double murmurs most audible at the **base** (other than hæmic):

I. Combined Aortic Obstruction and Regurgitation is the most common condition, and causes a loud double to and fro murmur, heard best in the second right interspace.

II. Aneurysm of the Aorta may be attended by a double murmur having the same characters as in disease of the aortic valves. This is heard loudest in the second right interspace, but it may also be heard at the back, to the left of the fourth dorsal vertebra.

III. A double murmur occasionally occurs in the dilated aorta of the aged, but with less marked features.

IV. A double murmur, loudest in the pulmonary area, usually indicates Congenital Heart Disease, especially with patent ductus arteriosus.

(*b*) A double murmur most audible at the **apex** may be heard when both Mitral Stenosis and Regurgitation are present. It consists of a typical presystolic murmur running up to the first sound, immediately

followed by a systolic mitral murmur which is conducted outwards to the axilla. A third murmur, a diastolic mitral stenotic murmur, often coexists.

FALLACIES IN THE DIAGNOSIS OF DOUBLE MURMURS.—1. When a double murmur can be heard both *at the base and apex*, do not take for granted that mitral regurgitation exists, as well as aortic disease. Remember that a systolic mitral and a systolic aortic may be alike in character, and that aortic murmurs can often be heard at the apex, as well as the base. To arrive at a conclusion is often difficult, but one must rely on the position in which the murmur is loudest, and on the other features which distinguish mitral and aortic lesions.

2. When a *double aortic* murmur is present, the lesion may be regurgitation, or stenosis, or both together. A diagnosis is made by examining the pulse (§ 83), the time of the thrill, if one is present, and the position of the apex beat. In regurgitation the apex is displaced farther downwards and outwards than in any other form of valve disease. In aortic stenosis the left ventricular wall is hypertrophied, with but little enlargement of the cavity, but as emphysema is so often associated with it, the apex may be hard to find.

3. Murmurs of *pericardial friction* may easily be mistaken for a double aortic murmur; but whereas endocardial murmurs begin synchronously with the first or second sounds, the rub of pericardial friction often lags slightly behind them.

4. *Hæmic, cardio-pulmonary* and exocardial murmurs are occasionally double.

§ 61. SYMPTOMS RESULTING FROM CARDIAC VALVULAR DISEASE.—The first effect of valvular disease is *hypertrophy* of the heart, as already mentioned, and so long as there is adequate compensatory hypertrophy there may be no concomitant symptoms at all.

But, sooner or later, in most cases hypertrophy gives way to *dilatation*, and then a series of characteristic symptoms ensue. Those special to each form of valvular lesion have been referred to in the preceding section. Certain *general symptoms are common to all forms of chronic valvular disease* when this has produced myocardial failure.

1. *Breathlessness* on walking uphill, or even on very slight exertion, is a constant feature. No serious enfeeblement of the heart wall or disturbance of its function can exist without this symptom; and it cannot be too much insisted on that breathlessness is not only a symptom, but, in general terms, is the most accurate measure of the extent of the cardiac failure.

2. *Dropsy* occurs early in mitral, late in aortic, disease.

3. *Palpitation* is of less diagnostic import, for it generally occurs without organic heart change.

4. *Pain* is by no means always present in cardiac dilatation, but few cases run their entire course without some præcordial discomfort. Pain is a fairly common feature of aortic disease, and sometimes is anginal in type; it is then due to interference with the coronary blood flow.

5. *Insomnia*, in advanced cases, is frequently a troublesome symptom, and in aortic regurgitation may be the first symptom of failure. Sometimes the patient, when dropping off to sleep, suddenly starts with the terror of suffocation, and gasps for breath. *Headache* and *delirium* are also met in advanced cardiac disease. The former is often due to variations

in the blood pressure. Delirium in heart disease is usually due to cerebral anoxæmia, resulting from slowing of the circulation rate (see § 35).

6. *Embolism* may occur, as described under Acute Endocarditis (§ 49), but without evidence of general infection. It is most frequent in mitral stenosis and in auricular fibrillation, and can occur in aortic disease. Emboli commonly occur in one of the middle cerebral arteries.

The chief ETIOLOGY OF CARDIAC VALVULAR DISEASE in *youth* is rheumatic endocarditis, which has a special tendency to attack the mitral valve. In *advancing years*, the commonest cause is an atheromatous degeneration. Rarer causes are: infective endocarditis (§ 50) and syphilis. The latter attacks only the aortic valve.

1. *Acute Endocarditis* of rheumatic origin is by far the most frequent cause, and a large majority of "heart cases" date their symptoms from an attack of that disease in youth or early adult life.

2. *Chronic Endocarditis* may come on insidiously. Syphilis causes aortic regurgitation between the ages of 40 and 60, often in association with arteriosclerosis. Chronic rheumatic endocarditis more often supervenes upon acute endocarditis—attacks of which may have been overlooked. Rheumatic heart infection may begin during rheumatic fever, chorea, rheumatic tonsillitis, or scarlet fever, and may be of insidious onset.

3. *Degenerative* changes (*e.g.*, atheroma) are the lesions chiefly met with after middle life. They affect especially the aortic orifice, either by injuring the valves or by causing dilatation of the aorta, which, extending to the situation of the valves, prevents them from meeting during diastole.

4. Any prolonged *high blood pressure*—*e.g.*, that which accompanies arteriosclerosis —may lead to valvular strain, usually aortic. Persistent obstruction in the lungs (*e.g.*, chronic bronchitis), or in the general systemic circulation, may have the same effect as persistent high tension on the right or left side of the heart respectively.

5. *Congenital* conditions are referred to in § 59.

Prognosis. In a case of chronic heart disease the fundamental factor is the heart muscle. Is the heart muscle handicapped? If so, is the handicap removable? If not, is a handicap likely to arise in the future? These questions must all be answered in any one case. In actual practice the prognosis is good in proportion to *the amount of exercise a patient can take without producing breathlessness*. The factors which influence the function of the heart muscle are toxins, metabolic factors, oxygen supply, and certain mechanical considerations, which include occupation or exertion, valvular defects and cardiac irregularities.

Toxins. Diphtheria, influenza, pneumonia, typhoid fever and certain septic foci produce a myocarditis which is completely recovered from if the patient survives the disease. Acute rheumatism not only produces permanent myocardial changes; it is apt to recur repeatedly during a patient's life, thus injuring the heart muscle ever more severely. If, however, the rheumatic process ceases, and if mitral stenosis has not been produced, a complete functional recovery is possible. If toxæmia of any kind is present, the myocardium will fail as a result of increased work, and the severity of the failure will be in proportion to the amount

of toxæmia and the amount of work. A healthy, non-toxæmic heart muscle will never fail, whatever the severity of the exertion undertaken. The factors which cause an athlete to be " rowed out " are not myocardial, but vasomotor and nervous.

Metabolic factors producing myocardial failure are hyper- and hypothyroidism and beri-beri. Complete cure is here possible.

Oxygen supply to the myocardium is deficient either in anæmia or in coronary disease. In the former case the prognosis depends upon the removability of the cause. The prognosis in coronary disease depends upon the following factors: (i.) with advancing years, as Gross has shewn, the collateral coronary anastomoses become freer; (ii.) coronary disease is often very localised, but it may or may not be part of a generalised arteriosclerosis; and (iii.) any individual coronary lesion tends to be progressive. Thus a coronary thrombosis in a man of 50, who has no hypertension, may heal and leave him with no dyspnœa; then a complete recovery is possible and the prognosis is good. On the other hand, a man with cardiac pain on exertion, due to coronary sclerosis and associated with hypertension, will be likely to lose ground progressively. Pulsus alternans, gallop rhythm and Cheyne-Stokes' breathing are bad signs.

Mechanical factors. The *valvular* lesions which seriously hamper cardiac efficiency are mitral stenosis and aortic incompetence. In mitral stenosis an increasing check is placed upon the amount of blood allowed to enter the left ventricle, and since this chamber can only expel what it receives the circulation rate is progressively reduced. Aortic incompetence is a mechanical handicap, for the left ventricle has extra work to do per beat, the diastolic coronary flow to the myocardium is less efficient, and the propulsive effect on the circulation of the aortic recoil is lost; also the factors which cause injury to the aortic cusps are apt to obstruct the mouths of the coronary vessels. The prognosis in aortic stenosis is much better. Cardiac irregularities chiefly cut down the circulation rate by the associated tachycardia, which reduces the diastolic time and thus interferes with ventricular filling.

The prognosis in *valvular lesions* depends upon the myocardial condition, and the size of the valvular defect. A case of arrested aortic valvulitis, from rheumatism or syphilis, may live a healthy life for many years. As a brief generalisation the least dangerous valvular defect is mitral regurgitation; next in order comes aortic stenosis, then mitral stenosis; and most dangerous of all is aortic regurgitation. But in every case the etiological factor must be taken into account. The extent of the lesion can be best gauged from the size of the heart chamber affected by it. The enlargement of the left ventricle is proportional to the extent of the lesion in aortic stenosis or regurgitation and in mitral regurgitation. Hypertrophy of the right ventricle from valvulitis is only found clinically in advanced cases of mitral stenosis. Hypertrophy is a compensatory function; dilatation is of definitely bad omen.

As regards individual examples: in *aortic regurgitation*, a good prognosis may be given in young rheumatic individuals with a good exercise tolerance, a not unduly large heart, a slow resting pulse rate and a normal diastolic blood pressure with a comparatively small pulse pressure. Combined with the above there must be no sleeplessness and an occupation in which there is a healthy amount of muscular exercise, which is essential for an efficient venous return. A relatively bad prognosis should be given in aortic regurgitation when the case is of syphilitic origin, where the heart is considerably enlarged, the apex beat slapping (not heaving in character), where the pulse is rapid, where the systolic pressure is high but tending to fall, and the diastolic pressure low; when the patient is sleepless, gets vertigo on changing his position or after exercise, when the heart is irregular, whether the irregularity be due to extra systoles (left ventricular fatigue), to alternation (failing contractility), or to auricular fibrillation. Associated conditions, such as pregnancy, of course, add to the gravity of the prognosis.

In *mitral stenosis*, the prognosis is relatively good when the patient is capable of leading a sheltered life; when the pulse is regular and of good volume; when there is no marked dyspnœa or cyanosis, no raised pulse rate, and a relatively good exercise tolerance. The prognosis is bad when the pulse is small and irregular; when there is marked cyanosis and dyspnœa on mild exertion, or orthopnœa; when the blood pressure is very low, the pulse is rapid, the liver is palpable, and when there are crepitations at the bases of the lungs and œdema of the feet.

The occupation, sex, and temperament of the individual are of importance. In heart cases it is essentially the pace that kills. If the patient is peacefully occupied with work which he can carry out in his own way, in his own time, the prognosis may be relatively good, but if he has to work against time, especially at an occupation that he is not used to, the prognosis becomes relatively bad. The placid individual who takes things as they come and does not worry, usually lives longer than the worrying person who meets trouble half-way. Generally speaking, the prognosis is better in women than in men, but here again it is largely a question of the lives they lead and the amount of rest they are able to take.

The prognosis of *Heart Disease as affected by Pregnancy* demands special notice. In pregnancy the maternal heart is loaded (1) owing to the increased nutritive demands of the fœtus or embryo, and (2) by the mechanical embarrassment caused by the growing uterus. The former of these operates from the commencement of pregnancy; the latter becomes of increasing importance as pregnancy proceeds. In conditions such as mitral stenosis, where the right heart is in a continual state of strain, special care must be taken. In the case of aortic regurgitation, where the left heart is under a strain, the early months of pregnancy are attended with no particular risk, but towards the end of pregnancy (the 8th and 9th months) the risk of failure is gradually increased. Furthermore, after delivery it is essential to keep the patient still and to support the splanchnic area, as sudden vasomotor collapse is liable to occur. With proper precautions heart cases stand pregnancy well. Decision as to the safety of pregnancy is a matter of experienced judgment and varies with individual cases. Briefly, it may be said that if there are no symptoms

or signs of failure, pregnancy can be considered. If such symptoms have previously existed, pregnancy is possible but is a definite risk. If signs of failure are present, pregnancy must not occur, or must be terminated.

§ 62. The **Treatment** OF CHRONIC HEART DISEASE (including Myocardial Degeneration and Valvular Disease) may be considered under three heads: (*a*) When compensation is fully established; (*b*) when compensation begins to fail; (*c*) when compensation has broken down.

(*a*) When there is efficient compensation, no symptoms are present and no active treatment is needed, but much may be done to prolong the patient's life, and to avoid the supervention of cardiac failure. Subjects of chronic valvular disease should be enjoined to lead quiet, regular, and orderly lives. With regard to exercise, it may be said, in general terms, that the patient himself is the best judge, provided always that he does not exert himself sufficiently to cause palpitation, undue dyspnœa, or præcordial pain. Some sports are more permissible than others; thus cricket, tennis, and golf may often be enjoyed, whilst football, racing, and rowing must generally be forbidden. Climbing, especially to high altitudes, must be disallowed. Alcohol, tobacco, and tea are all myocardial poisons if taken to excess, and should be used only in strict moderation. The skin should be kept active by the daily bath, and the bowels regular by means of purgatives if necessary. Whenever possible, a means of livelihood should be chosen in which the heart is subjected to but little strain. A sedentary occupation with moderate exercise in the intervals, is more suitable than one which entails earning a living literally by the sweat of the brow. Lifting or carrying heavy weights, climbing ladders, wielding heavy hammers, and physical labour in constrained positions, are liable to overtax the powers for compensation of the cardiac muscle. Meals should be regular, and heavy meals should be avoided. The diet should be easily assimilable, and contain only a moderate amount of fluid. A small quantity of stimulant with meals may be called for, but should not be used unnecessarily, because of the reaction afterwards, and of the tendency to excess, which exists in cardiac cases.

(*b*) When compensation is beginning to fail, the condition of the heart should be noted frequently; rest, drugs, and exercises being prescribed in accordance with the variations in the circulation and the capability of response to treatment by the cardiac muscle.

Drugs.—In cardiac failure, especially in auricular fibrillation, when the pulse becomes feeble, rapid, and irregular, *digitalis is par excellence* the remedy. It is especially indicated in failure of the right heart in mitral stenosis or regurgitation, whether primary or secondary to aortic lesions. It is contra-indicated when there is full compensatory hypertrophy, and the pulse is fairly strong, regular, and slow, or if vomiting is present. By its action on the vagal nerve endings in the Bundle of His, it reduces the number of auricular impulses reaching the ventricle. Its action is not quite so efficacious if fever is present. There are two methods of administering it—massive or intensive dosage, or maintenance dosage.

If a patient is very seriously ill with heart failure and auricular fibrillation, digoxin 1 mgm. injected intravenously is often used, and can be repeated in 6 hours. In a patient less ill tincture of digitalis can be given 6-hourly in three doses of ♏ 90, ♏ 60, and ♏ 30. After these emergency measures, a maintenance dose is given, 10, 15 or 20 minims t.d.s., p.c. according to circumstances. The two things to watch are the heart rate and the signs of digitalis overdose. The apex rate, not the pulse rate, is the only safe guide to the correct dose of digitalis in auricular fibrillation, owing to the apex-pulse deficiency which occurs. No nurse should be allowed to record the pulse figures in these cases who cannot count the heart rate by stethoscope; pulse figures alone are valueless. The result to be obtained is a reduction of the heart rate to 70–80 per minute. The signs of overdose to watch for, in order of severity, are: reduced urinary output, coupled beats, nausea, vomiting, headache, and visual disturbance. In less severe cases of failure, it is sufficient to give ♏ 30 t.d.s., p.c. for 2 to 4 days, subsequently giving a maintenance dose of about ♏ 10 t.d.s., p.c.

The most useful preparations of digitalis are, in institutions, the tincture; for outpatients, digitalis pulverata, one grain being equal to 10 minims of the tincture, or digitoxinum (*B.P.C.*), which is similar to Nativelle's digitaline; and for cases where rapid absorption is desired, digoxin B.P. (0·25 mgm.), one tablet being equal to 15 minims of the tincture. The latter drug has removed one of the chief deficiencies in digitalis medication, namely, the slowness of its action. It may be given in doses up to 1 mgm. intravenously. Strophanthin has an action similar to that of digitalis, and can also be used intravenously. It must never be so given to a patient who recently has been taking digitalis, as sudden death has been known to occur. Digitalis can be continued indefinitely, as tolerance is not acquired. In regularly beating hearts it probably has a definite but slight effect by improving contractility. The action of digitalis and many other cardiac remedies is expedited by an occasional dose of calomel. Formulæ 54, 57, 67, and 84 are useful. The absorption and action of digitalis are slow, and in very acute cases of heart failure recourse is often had to rapidly acting stimulants, such as brandy, caffein citrate gr. 2–3, or leptazol B.P. (cardiazol) subcutaneously, but it is questionable whether any direct cardiac effect is produced by them. These preparations commonly used act on the vasoconstrictor and respiratory centres and not on the heart. Digitalin injections have little action unless given intravenously. Strychnine is useless as a cardiac stimulant, but stimulates the respiratory centre and increases tone generally. In *aortic valvular disease* and in the early stages of *mitral stenosis*, digitalis is not so valuable a drug; but in the later stages of these affections, when compensation begins to fail, and especially when auricular fibrillation is present, digitalis gives relief. In aortic cases, where the blood pressure is high, or where angina is present, the vaso-dilators are often of use, such as nitroglycerin in the form of liquor trinitrini ♏ i. t.i.d., erythrol tetranitrate or sodium nitrite. Theophylline ethylene diamine

cardophylin) intravenously once a day is a useful drug for cases of an arteriosclerotic nature. Sugar in doses of 1 to 5 ounces two or three times daily is useful for a failing myocardium from whatever cause.

The various symptoms may be met by appropriate remedies. For the *pulmonary congestion*, venesection is by far the most efficacious form of treatment. Pulmonary œdema is benefited also by injections of mersalyl. Nitroglycerin is useful for the headache and sleeplessness due to *hypertension.* Phenobarbitone is valuable for anxiety and restlessness, but in cases with cerebral arteriosclerosis it is apt to cause mental changes and even delirium. For the paroxysms of *dyspnœa*, morphia, cardophylin (0·48G. in 20 c.c. of sterile water, intravenously, daily for 7 days), or oxygen inhalations by mask or nasal catheter are useful. *Cough* is relieved by drinks of hot milk, and drugs such as codein, small doses of opium, and chloroform or ether. For *palpitation,* alcohol is a valuable sedative. The quantity should always be moderate. Other causes of palpitation which may be present should be treated (§ 34). For *sleeplessness,* in more acute cases, opium or morphia hypodermically is most useful, and should be given without hesitation. In children or in cases where the insomnia is not obstinate, other drugs may be employed, such as potassium bromide, phenobarbitone, sulphonal, trional, and paraldehyde. Chloral is harmless. The *hæmoptysis* of heart disease is best left alone, as it relieves the congestion. The *gastric* symptoms may be relieved by acting on the congested liver with calomel, ½ to 1 grain every night, with sodium sulphate and sodium bicarbonate (30 grains in 2 ounces of hot water) in the mornings. Digitalis must be stopped if it causes sickness. For the treatment of *pain* and *syncopal attacks, vide* §§ 33 and 35. Formula 56 is useful.

Massage and Systematised Exercises.—At one time rest was regarded as imperative for all forms of cardiac disease. But the advance of physiological knowledge has shown what an important part the skeletal muscles play in the circulation of the blood, by squeezing the fluids out of the soft-walled veins and lymphatics, while they cannot compress the lumen of the firm-walled arteries. There are three varieties of this treatment, which are invaluable for different degrees of cardiac failure. *First,* for the worst cases, *gentle massage,* combined perhaps with *passive movements.* These are available where any kind of voluntary movement on the part of the patient is attended with breathlessness. *Secondly,* slow *voluntary movements* of flexion and extension on the part of the patient while standing or sitting. In the Nauheim system these voluntary movements are gently resisted by the operator—"resistance gymnastics." These movements, combined with *baths* (see below), constitute the essence of the system. *Thirdly,* Oertel's method, which consists of three parts: first, reducing the amount of fluid taken to 31 ounces per diem (to include the amount contained in the solid food) and promoting perspiration; secondly, a diet largely consisting of proteins [1]; and thirdly, graduated exercise in

[1] Oertel's dietary is as follows:—*Morning:* 6 ounces of coffee, 3 ounces of bread. *Noon:* 3 to 4 ounces of soup, 7 to 8 ounces of roast meat or poultry, salad or green

the form of walking uphill, each day a little farther. Cases with plethora and obesity are the most suitable. In cases of early failure who are still ambulatory, much good can be done by reducing the weight when this is excessive. A diet poor in fat and low in carbohydrate provides the best method. Breathing exercises, by helping the venous return, are valuable in chronic heart disease.

Saline and effervescent *baths* may be usefully added. They act by relaxing the arterioles of the skin directly, and of other parts reflexly. By these means blood is transferred from the venous to the arterial system, and its flow accelerated (see Nauheim baths.)

(*c*) When compensation has broken down and marked cardiac failure is present, absolute rest is necessary. The patient is usually unable to lie down, but has to be propped up with pillows, and in severe cases sleep can be obtained only when the legs are hanging down. A special "heart" bed is valuable in such cases, as the degree of dorsal support and dependence of the legs can be adjusted to each case. In severe failure of the right heart, as indicated by distended jugular veins, cyanosis, the liver dulness extending well below the costal margin and the cardiac dulness extending far to the right, *venesection* is called for, and brings prompt relief. The rapid removal of from 10 to 20 ounces of blood is usually sufficient; this may be repeated. Three to six leeches may be applied to the right lower ribs in children, in whom venesection is more difficult to perform. In the treatment of cardiac œdema, mersalyl, 1 or 2 c.c. injected intramuscularly or intravenously, is one of the most useful remedies known for removal of fluid and usually renders tapping unnecessary. Ammonium chloride (20 gr. t.d.s., p.c.) should be given for one day before mersalyl is begun, and should be continued during the treatment. The mersalyl injections can be repeated every 4 or 5 days. Signs of mercurial poisoning such as salivation and stomatitis must be watched for. Mersalyl may be dangerous if renal disease is present. Similar compounds can be used rectally; in common use are suppositories of mersalyl. Theophyllin and sodium salicylate (diuretin), 10 gr., and theocin sodium acetate, 3 gr., may be useful. The diet should contain a minimum of salt. Dropsy may be treated by draining the legs by Southey's tubes (§ 29) or multiple superficial incisions, asepsis being maintained by penicillin cream and sterile dressings. Aspiration of a pleural effusion or paracentesis abdominis may be necessary. Pleural effusions are common in congestive heart failure: treatment by removal of the fluid will increase the expanding area of the lung, decrease the anoxæmia and benefit the myocardium. They should be looked for, and removed by aspiration even though comparatively small. Diaphoretics are of little use in cardiac dropsy. Digitalis and caffein should be employed in conjunction with

vegetable, a little fish, 1 ounce of bread or farinaceous pudding, 3 to 6 ounces of fruit; no liquid (excepting in hot weather, 6 ounces of light wine). *Afternoon:* 6 ounces of tea or coffee (1 ounce of bread occasionally). *Evening:* One or two lightly boiled eggs, 1 ounce of bread, salad, fruit, sometimes a small piece of cheese, 6 to 8 ounces of light wine, with 4 to 5 ounces of water.

diuretics, calomel, and hydragogue cathartics, such as pulv. jalapæ co. and cream of tartar. The digitalis, squill and mercury pill is useful at this stage; so also Formula 55.

Complete thyroidectomy has been successfully used in order to reduce the basal metabolic rate of cardiac patients, and thus to lighten the burden of the heart. It has been used for repeated attacks of congestive failure in myocardial degeneration; but it is most useful in carefully chosen cases of anginal pain of organic origin; for this sympathectomy has also been performed (§ 51).

§ **63.** Group E. We now turn to the consideration of those cardiac disorders the recognition of which depends upon **Alterations in the Rate or Rhythm of the Pulse.** In all cases *it is essential to compare the radial pulse with the heart sounds,* and to observe the pulsation in the veins of the neck.

The Electrocardiograph and, rarely, the Polygraph (§ 44) may be required to make an exact study of a case presenting pulse alterations; but it is often possible to make a correct diagnosis without their aid.

I. With an occasional PAUSE in the radial pulse	Premature Beat (Extrasystole) (§ 64). Early Heart Block (§ 69). Sino-auricular Block (§ 64).
II. With RHYTHMIC alteration of rate DEPENDENT ON RESPIRATION	Sinus Arrhythmia (§ 65).
III. With INCREASED rate	Tachycardia, Physiological (§ 84) or Paroxysmal (§ 66). Auricular Flutter (§ 67). Auricular Fibrillation (§ 68).
IV. With DISORDERLY RHYTHM	Auricular Fibrillation (§ 68) or Multiple Premature Beats (§ 64).
V. With DECREASED rate	Bradycardia (§ 85). Complete Heart Block (§ 69).
VI. Coupling of the Pulse Beats	Premature Beats (§ 64). Pulsus Alternans (§ 71). Extreme Dicrotism (§ 70).

The various causes of altered rate and rhythm of the pulse, other than cardiac disease, are considered in § 84 *et seq.* Here we consider only the cardiac conditions to which attention may first be called, and in which the diagnosis may be largely made, by alterations in the pulse rate and rhythm.

A Pause in the Pulse, which is Otherwise Regular, is due to three conditions: Premature Beats, Heart Block, and Sino-auricular Block. The first of these is very common, the second somewhat rare, and the third very rare. The pause in the pulse caused by premature beats is due to the fact that the ventricle is prematurely stimulated early in diastole, and the output from it is therefore generally insufficient to

produce a pulse wave. Following the premature beat there is a compensatory pause. During the pause in the pulse, as felt at the wrist, the stethoscope, if placed over the heart, can detect the sound of the premature beat. This differentiates premature beats from heart block and sino-auricular block.

I. *There is an occasional pause in the radial pulse, during which the heart gives a short premature beat which can be heard over the præcordium.* The condition is PREMATURE BEAT.

§ **64. Premature beats (Extrasystoles)** are due to hyper-irritability of the myocardium, causing early contraction in some part of the heart.

The normal beat of the heart always starts at the most irritable point, normally the sino-auricular node. Should any other point of the heart become more irritable it initiates the contraction; such a beat is said to be ectopic in origin, and inasmuch as the contraction occurs earlier in the cardiac cycle than the normal beat, it is called *premature.* Ectopic beats may arise either in the auricular, nodal or junctional tissue (*i.e.*, the tissue between auricles and ventricles) or in the right or left ventricles. Ectopic auricular beats give rise to a ventricular contraction of normal

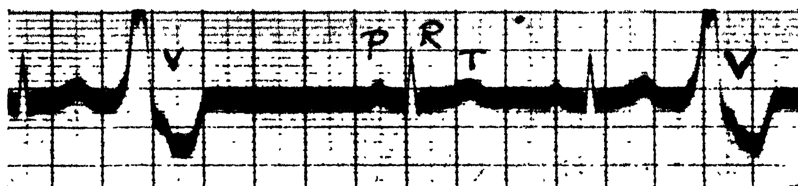


FIG. 32.—Premature ventricular beats (V). Note the prematurity and the large amplitude of the complexes.

type. Should the ectopic beat start in the nodal tissue, it travels upwards towards the auricles and downwards towards the ventricles, so that the auricles and ventricles contract more or less simultaneously.

Symptoms. The patient may or may not be conscious of the altered heart beat. The symptoms are palpitation, a catch in the breath, a thump, a sinking sensation, or even a sudden momentary stab of præcordial pain.

Broadly speaking, the *causes* of hyper-irritability which give rise to premature beats or extrasystoles are of (1) extrinsic, or extra-cardiac; or of (2) intrinsic, or cardiac origin. Extrinsic, *e.g.*, a distended stomach causing premature beats starting in either the right ventricle or auricle: such beats disappear on relief of the distension. In the eighth month of pregnancy the heart is often irregular, due to the distended abdomen.

The commonest intrinsic causes of hyper-irritability are: (1) Toxæmia; (2) fatigue; (3) inflammation; (4) degeneration.

(1) The connection between premature beats and *toxæmia* is seen with such conditions as malaria, typhoid, influenza, excessive tobacco, etc. Thyrotoxicosis, an abnormal susceptibility to coffee or tea, and digitalis poisoning are also common

causes. Septic foci may be causal. Generally speaking, all parts of the heart are involved, so that the irregularity is considerable, sometimes the auricles, sometimes the right and at other times the left ventricle showing premature beats. The abuse of tobacco as producing cardiac irregularities is well known and is a recognised method of evading military service.

(2) The relationship between premature beats and *fatigue* is especially interesting. The premature contractions may be associated either with a general fatigue and only occur after actual physical effort, or they may be associated with myocardial disease. Premature beats which appear or increase only after physical effort, are suggestive of myocardial damage. Any irregularity of the heart which develops with or after exercise, whether due to premature beats, auricular fibrillation, auricular flutter or any other cause, must always be looked upon as due to some definite myocardial disability. Even when premature beats are more frequent *after* exertion, they will generally be found to disappear during the period of exertion: this distinguishes them clinically from auricular fibrillation, where the rhythm is more irregular *during* exercise.

(3) *Inflammation.*—In acute myocarditis premature beats are common. In chronic forms of myocardial disease they frequently occur independently of any endocardial or pericardial involvement. Persistent premature beats increased by exercise always indicate myocardial damage. If auricular, they often precede fibrillation; if ventricular, they are often associated with acute local or diffuse myocardial disease. The association between premature beats and inflammation is often well seen in the course of an attack of rheumatic fever. The patient, perhaps, has well-marked signs of endocarditis or pericarditis, but as far as one can tell the myocardium has escaped damage. The heart then becomes irregular, and owing to the development of premature beats, one knows that the inflammation has involved the myocardium.

(4) In *myocardial degeneration* premature beats most commonly occur after fifty years of age, when the cardio-vascular system is degenerated; they are frequent antecedents to such conditions as auricular fibrillation or heart block, associated with generalised myocardial change.

Prognosis.—The essential point to remember first is that the majority of individuals who have ventricular premature beats do not suffer at the time or subsequently from heart failure or gross disease. The prognosis in all cases with premature beats must be decided independently of the presence of this sign. The prognosis has been briefly mentioned under each cause. Ectopic beats are important according to (1) the underlying condition on which they depend, and (2) the amount of circulatory disturbance they produce. Premature beats are occasionally so distressing to the patients as to require treatment. If so, a useful mixture contains quinidine sulphate gr. 2 or 3; atropine sulphate gr. $\frac{1}{150}$; acid. sulph. dil. ♏ 10; aq. chlorof. ad $\frac{1}{2}$ fl. oz., t.d.s., p.c.

The first stage of **heart block** (§ 69), in which the only change is lengthening of the "P–R" interval, is not diagnosable clinically (Fig. 16). The second change is that of an occasionally dropped beat, the "P–R" interval lengthening from normal progressively, until eventually an auricular stimulus fails to reach the ventricle. A ventricular beat then drops out. In this case the pause is completely silent (Fig. 17). The common causes of this condition are acute rheumatism, diphtheria, pneumonia, and occasionally other febrile states.

Sino-auricular block is a rare condition of no especial clinical significance, in which there is a complete obliteration of the whole of a cardiac cycle, both auricular and ventricular beats being equally affected. It is sometimes accompanied by other evidences of excessive vagal tone.

II. *The patient is* YOUNG, *and presents a* REGULARLY RECURRING *alteration of the pulse rate, usually dependent upon* RESPIRATION. *The condition is* SINUS ARRHYTHMIA.

§ 65. **Sinus Arrhythmia** is a condition in which the discharge of perfectly normal impulses from the sino-auricular node occurs periodically, producing a rhythmic irregularity of the heart.

Symptoms.—The pulse rate increases with inspiration, but there is no great difference between the strength of any two successive beats. The regular waxing and waning of the pulse is often accentuated when the patient breathes deeply. Auscultation reveals no alteration in the heart sounds. This irregularity is without symptoms.

Causes.—The condition is common in the young and during convalescence from diseases in which the heart rate has been rapid. It is of vagal origin in individuals in whom there is exaggeration of the normal inspiratory increase and expiratory slowing of pulse rate.

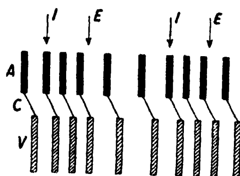


FIG. 33.—Sinus arrhythmia. The heart mechanism, auricular contraction (A), conduction (C) and ventricular contraction (V), is normal. The heart rate during inspiration (I) is increased, and during expiration (E) is slowed.

Prognosis.—The condition is of little importance; it ceases when the pulse rate quickens from any cause, *e.g.*, after exercise. When found after fevers, it is a good sign, inasmuch as it suggests the absence of extensive damage to the heart wall. No treatment is indicated.

§ 66. III. *The Cardiac conditions in which an Increased Rate forms the most striking feature, are:* PAROXYSMAL TACHYCARDIA, AURICULAR FLUTTER, AURICULAR FIBRILLATION.

In the majority of cases of regular TACHYCARDIA the increase is physiological in character. In two conditions, paroxysmal tachycardia and auricular flutter, the heart beat starts from a new focus.

These two forms of tachycardia may be differentiated by the following features:—In the physiological type the pulse rate is (i.) affected by posture, falling 10 to 30 beats when the patient passes from a standing to a recumbent position; (ii.) the pulse rate increases with exercise, and is affected by emotion, meals, fever, and sleep; (iii.) the onset and termination are gradual; (iv.) electrocardiograms are normal; (v.) jugular tracings show no exaggeration of the force of the auricle. The causes of this form of tachycardia are dealt with in § 84.

The pulse rate is REGULAR, 130 to 200; *the rate is unaffected by exercise or posture. The condition is* PAROXYSMAL TACHYCARDIA OR AURICULAR FLUTTER.

Paroxysmal Tachycardia is a term reserved for cases of rapid action of the heart presenting the following characters: (1) the onset of the tachycardia is abrupt; (2) the duration varies from seconds to days; (3) the relief is sudden, and the pulse returns to its normal rate in the course of a few beats, which are often irregular in force and rhythm. During the paroxysm violent jugular pulsation may be visible.

The *symptoms* complained of by the patient depend upon the duration of the paroxysm. Many of the short paroxysms, lasting a few hours, are accompanied only by a fluttering or throbbing sensation in the chest or at the root of the neck, and a feeling of lassitude. Some attacks cause pain of an anginal nature. When the attack is prolonged over several days, grave cardiac embarrassment with dilatation, cyanosis, œdema of lungs, and engorgement and enlargement of the liver occur. Occasionally there is great distress and discomfort. The general disturbance of the patient, the rapidity of the pulse, and the severity of the abdominal pain dependent upon the engorgement of the liver may be so extreme as to simulate an acute abdominal condition calling for surgical interference. Cases are on record of exploratory laparotomy having been performed owing to such an error of diagnosis. The rapidity of the disappearance of the abdominal symptoms on cessation of the tachycardia is a very striking feature. The immediate *prognosis* depends chiefly upon the presence or absence of dilatation of the heart. The most severe symptoms may disappear in less than an hour if the heart muscle is healthy.

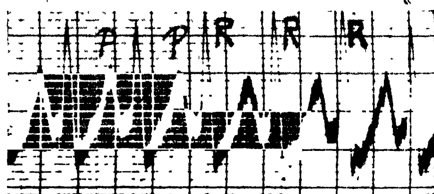


FIG. 34.—Auricular paroxysmal tachycardia. The "P" waves are abnormal in shape showing an ectopic origin, and at a rapid rate. The "QRS" waves are normal.

Etiology.—The condition is due to sudden rhythmic activity of some ectopic focus (usually in the auricle), which for a time overcomes and replaces the normal activity of the sino-auricular node. It is most common in young adults, but may occur in early childhood or old age. The attacks may be excited by exertion, emotion, flatulence, or change of posture. The disturbance is often not ascribable to physical disease, but is sometimes a consequence of rheumatism, scarlet fever, syphilis or coronary disease (see § 68, Etiology). Often no valvular lesion is present; if valvular murmurs are present they become unrecognisable during the paroxysm. Post-mortem examination has shown fibrosis, pallor, friability of the heart-muscle and sometimes coronary disease.

The *diagnosis* from tachycardia of purely *nervous* origin depends upon: (i.) the abrupt onset and relief; (ii.) the presence of violent jugular pulsation; (iii.) occasional presence of a few premature beats in the intervals between the paroxysms. Many attacks of so-called "Paroxysmal Tachycardia" are really paroxysms of Auricular Fibrillation or Auricular Flutter (see below).

Treatment.—The brief paroxysms which produce no subjective symptoms call for no treatment. For the prolonged attacks the patients often discover for themselves some simple procedure which cuts them short, such as holding the breath, compressing the abdomen with a tight binder, or the assumption of some special posture. If these are inadequate, pressure may be made upon the vagus in the neck. Morphia may be called for, and should embarrassment of the right heart become extreme, venesection and the removal of fl. oz. 10–20 of blood may give relief. Drug treatment is useful during the attack and between attacks as a prophylactic measure. For the former

purpose digitalis in full doses (see § 62) can be tried. In a case of emergency quinidine sulphate can be given in increasing 2-hourly doses of gr. ii., iii., iv., v., vi., and vii.; but it is definitely dangerous in this condition, and should only be given if the danger of the attack itself seems the greater. Acetyl choline in the form of carbachol B.P. (doryl), intravenously, will often cut short an attack: 1 c.c., diluted in 10 c.c. of saline, is given slowly into a vein. Prophylactically, quinidine sulphate in doses of gr. 2 or 3 t.d.s., p.c. is a safe and useful remedy.

§ 67. **Auricular Flutter.**[1]—This name has been given to a condition in which "the normal beats of the auricle are submerged by contractions of this chamber in response to a series of new, rhythmic, and pathological impulses varying in rate from 200–350 per minute" (Lewis). The distinction from Paroxysmal Tachycardia is arbitrary as regards auricular rate, and is drawn at 200 beats per minute. Flutter differs, however, from Paroxysmal Tachycardia in that it is due to a circus movement and is almost invariably associated with some degree of heart block (§ 69); the auricle may, for instance, be beating at the rate of 300 per minute, while the ventricle responds with only 150 beats per minute. In other cases a higher grade of heart block (4–1) may be present, and the

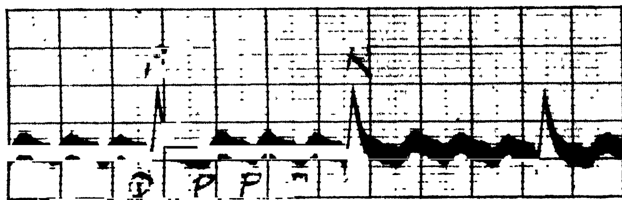


FIG. 35.—Auricular flutter. The auricular waves are undulatory at the rate of 300, and the ventricle responds only to every 4th auricular beat. One of the "P" waves is periodically obscured by the "QRS" waves.

pulse rate be about 75 and regular. The rate of the auricle is absolutely regular; that of the ventricle may be regular or irregular, depending upon the constancy of the degree of heart block present (and see p. 105). It will be readily seen what difficulty there may be in making a clinical diagnosis of this serious heart condition.

Symptoms and Signs.—The symptoms of auricular flutter are generally those of heart failure; palpitation is sometimes complained of and the pulse rate is found to be unexpectedly feeble and rapid (see Fig. 35). For the certain recognition of the disorder polygraphic or electrocardiographic records are necessary, but its existence may be suspected when (i.) the pulse rate is 130–160, regular, and maintained for long periods, especially if associated with syncopal attacks; (ii) the ventricular rate shows no quickening with exercise or slowing on lying down. This constancy of the rate on alteration of posture and with exercise is an important

[1] Flutter has been placed under this heading because it is usually associated with a regular ventricular pulse. When varying grades of heart block are present this regularity in rate disappears, but in contrast to the pulse of auricular fibrillation regularity in force and volume persists.

diagnostic feature. (iii.) A visible jugular pulse of greater rate than the apex should lead one to suspect auricular flutter.

Etiology.—The condition is usually associated with chronic myocardial degeneration, but occasionally accompanies mitral stenosis. The patients are often past middle life, and are subjects of arteriosclerosis. The irregularity is occasionally produced by quinidine when given in auricular fibrillation (and see p. 110).

Prognosis.—The duration of the condition varies. It may occur only shortly before death; more frequently it persists for long periods, even for years. This long duration is a further diagnostic point of great importance. There is a danger of the ventricle suddenly assuming a rate equal to that of the auricle—a condition which can lead to syncopal attacks which may be fatal.

Treatment.—Digitalis in 20 minim doses four times a day will often effect a cure; it acts by producing auricular fibrillation (see below). If the drug is then withdrawn, fibrillation may suddenly cease and the heart resume its normal rhythm. Even if digitalis fails to produce fibrillation, it may be relied upon to reduce the ventricular rate. If large doses of the drug are given, the period of fibrillation may be cut short. A daily dosage of 90 minims for two or three days is often necessary. Quinidine sulphate can be used as an alternative method of treatment, as described in § 68.

IV. *The heart rate is irregular, with* COMPLETELY IRREGULAR *rhythm, and in failure may beat at a rate as high as* 180 *per minute; the condition is probably* AURICULAR FIBRILLATION.

§ 68. **Auricular Fibrillation** is recognisable by (i.) Complete irregularity of the pulse; (ii.) the difference between the apex and the pulse rates; (iii.) increase in the irregularity by exercise; (iv.) absence of the "A" wave on the venous curve or absence of the "P" wave on the electrocardiogram (Figs. 14, 36). (v.) Auricular fibrillation is usually associated with a low blood pressure, due to the small output, but in cases where the peripheral resistance is raised (*e.g.*, arterial sclerosis, renal disease) the blood pressure may be high: it always varies from beat to beat. Furthermore, auricular fibrillation is almost invariably associated with (vi.) increase in

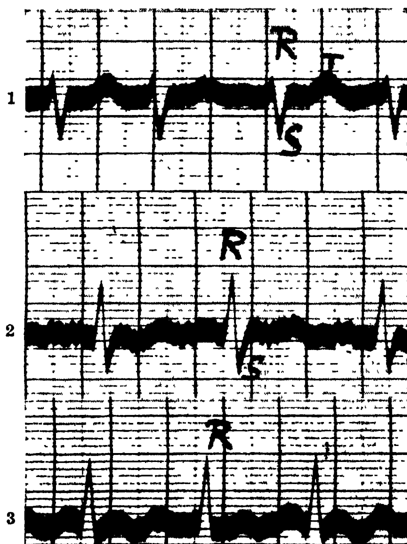


FIG. 36.—Right-sided preponderance. "S" is deepest in lead 1, and "R" is tallest in lead 3. (Auricular fibrillation is present.)

the cardiac dulness. (vii.) Auricular fibrillation is sometimes paroxysmal in character, the attacks lasting from a few minutes to hours, days, or even weeks: permanent fibrillation usually supervenes later.

Etiology.—The auricle is composed of a number of intimately connected muscle fibres, so much so that some people regard them as forming a syncytium; and a normal systole of the auricle is initiated at the sino-auricular node, and the stimulus travels from one muscle fibre to another, and gives a systematic and co-ordinate contraction of the auricle from above downwards, *i.e.*, from the sino-auricular to the auriculo-ventricular node. In auricular fibrillation the underlying process is a " circus movement " in which the sino-auricular node ceases to function and is replaced by a stimulus which makes a circuit of the muscle surrounding one of the large venous orifices 400–500 times a minute. The auricular muscle derives its stimulation from this circus movement in an entirely irregular fashion, for much of the auricular muscle is refractory to such a rate of stimulation. In consequence co-ordinate auricular contraction no longer occurs, but individual bundles of fibres, or even individual fibres, contract inco-ordinately, so that the whole auricular wall appears to be trembling or quivering in a state of diastole. The ventricle therefore receives impulses at very irregular intervals, and so the force and the rhythm of the ventricle varies from beat to beat. The stronger of these stimuli, at irregular intervals, traverse the Bundle of His and excite a ventricular contraction. According to the number of auricular stimuli which are transmitted to the ventricles, the rate of these will be fast or slow; the apex rate may vary between 180 and 40 per minute. Auricular fibrillation usually occurs: (1) secondary to valvular disease, especially mitral stenosis; (2) in myocardial degeneration, *e.g.*, cardio-vascular sclerosis; (3) in toxic conditions, *e.g.*, Graves' disease; (4) in the course of acute inflammatory conditions, *e.g.*, acute carditis; (5) very rarely as a result of syphilis.

The rate of circus movement in auricular fibrillation at 400–500 times per minute, is faster than in auricular flutter (200–350 per minute) because the path of the circus movement in fibrillation is shorter than in flutter.

The *prognosis* depends chiefly upon (1) whether the underlying cause is removable or not. In myocardial degeneration the cause is not removable but progressive; the prognosis is consequently bad. In Graves' disease, provided the muscle is only in the toxic and not in the degenerative stage, removal of part of the thyroid will remove the hyperthyroidism and the auricular fibrillation will cease. In mitral stenosis the immediately exciting cause of the fibrillation is a rise of pressure in the auricles. (2) The condition of the ventricular muscle. If the auricular fibrillation is due to a more or less local condition of the auricle, the outlook is comparatively good. But if the cause of the fibrillation has seriously affected the ventricular muscle, the prognosis is bad. (3) The extent to which the ventricle is overstimulated by the erratically acting auricle. Obviously the greater the ventricular rate and the larger the number of ineffective

beats, the greater the over-work of the ventricle and the worse the outlook. (4) Where the cause cannot be removed, how far it is possible to control the fibrillation. In some cases, *e.g.*, with mitral stenosis, it may not be possible to stop the fibrillation, but it may be possible to control it by treatment so that the ventricular rate is slowed and the number of ineffective beats few. In such cases the prognosis is good, the fibrillation making little difference to the conditions of life.

Treatment.—It is thus obvious that the treatment of auricular fibrillation first lies in attempting to remove the underlying cause. In some cases this is comparatively easy, in others it may be impossible. In hyperthyroidism it can be stopped by removing part of the over-active thyroid. In mitral stenosis, on the other hand, the removal of the cause is impossible. If it is impossible to stop the actual fibrillation, it is usually possible to control its effect on the ventricle. This is done by giving the patient that dose of digitalis which will keep the heart rate, at rest, between 70 and 80. If the heart rate is already as slow as this, digitalis is not needed. If there are signs of failure the patient must be treated in bed according to the scheme described above (see § 62). Digitalis must be continued, if needed, so long as the fibrillation remains, *i.e.*, usually for the rest of the patient's life.

Quinidine, an isomer of quinine, is sometimes useful in stopping auricular fibrillation. Contra-indications for its use are established mitral stenosis, signs of congestive failure, and marked cardiac enlargement. Where fibrillation is known to have persisted for many months, quinidine is generally not used. Thyrotoxic and early arteriosclerotic cases are the most suitable. Before quinidine treatment digitalis must be stopped; during quinidine treatment patients must be nursed flat, strictly confined to bed, and must not be allowed to sit up, for the drug is a myocardial poison. Dosage is as follows: gr. ii. t.d.s., p.c., for one day, to exclude the possibility of hypersensitivity to the drug, and on successive days eight two-hourly doses of gr. 2, 3, and 4, the final total daily dose being gr. 32. This final dose can be maintained for 2 or 3 more days, and is then stopped. If the auricular fibrillation ceases during treatment the course can be stopped, and a maintenance dose of gr. ii. or iii. t.d.s., p.c., continued for 2–3 months. In cases where auricular fibrillation is paroxysmal, a dose of 3–5 gr. a day will usually prevent attacks.

Quinidine sulphate acts as a myocardial depressant, and slows the rate of conduction as well as increasing the refractory period of the heart muscle undergoing circus movement. When the latter effect predominates, circus movement ceases.

V. *Conditions of the heart which are associated with a* SLOW PULSE.

§ 69. Slow pulse—between 40 or 50 or below—occurs in four more or less common conditions. (1) Personal idiosyncrasy; (2) The heart of the well-trained athlete; (3) Debilitating and exhausting diseases; (4) Conditions of complete or partial heart block.

(1) In the bradycardia of the athlete, the subject usually looks and seems physically very fit; on exercise he shows no symptoms of distress;

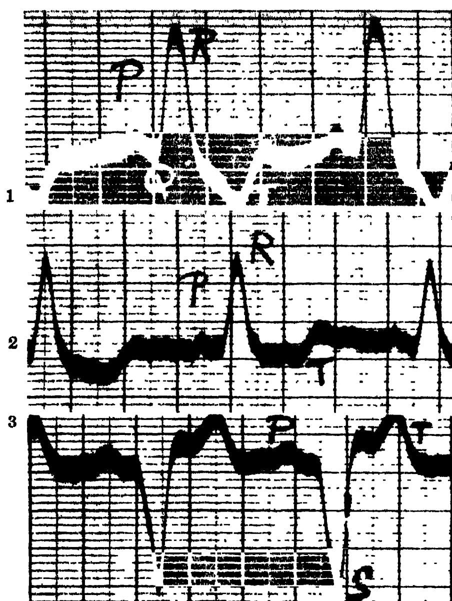


Fig. 37.—Lesion of the left bundle branch. Note (1) the widening of QRS to 0·12 sec. (normal less than 0·10 sec.), (2) the slurring of QRS, (3) the direction of T_1 and T_3, *i.e.* opposite to R and to S.

and his pulse rate, instead of climbing as the exercise is increased, suddenly doubles.

(2) Bradycardia may occur in conditions of lowered metabolism, such as inanition, exposure to cold, and myxœdema. It is also seen in some toxic states, as in jaundice.

(3) Digitalis may cause it in three ways—by producing heart block, by producing premature beats which fail to reach the arterial pulse, and by central vagal stimulation. In vagotonic conditions (*e.g.*, in increased intracranial pressure) and in shock bradycardia is often seen.

(4) In complete or partial **heart block** the rate is unaffected by exercise, and signs of cardio-vascular degeneration are frequently present. Periods of unconsciousness (Stokes-Adams' fits) may occur.

Etiology.—Conductivity is specialised in the Bundle system described in the introduction to this chapter. Any part of this conducting tract may be damaged, a whole series of conditions arising therefrom (Fig. 38). Sometimes the stimulus appears to be blocked in the sino-auricular node, when the whole heart is silent and misses a beat—a condition spoken of as sino-auricular heart block (§ 64). If the Bundle of His is damaged, then either partial or complete auriculo-ventricular block occurs. Furthermore, either the right or left main branch may be damaged, right or left bundle block resulting (Fig. 37). Bundle block not infrequently occurs in cardiac atheroma, with aortic regurgitation, in coronary thrombosis, in chronic lung affections and in certain cases immediately prior to death. All these conditions can be easily made out by means of the electrocardiograph.

The varieties clearly depicted in Fig. 38 are here summarised :—

1. *Supra-auricular* (Sino-auricular) (§ 64).
2. *Auriculo-ventricular :*
   (*a*) Temporary { Complete. / Incomplete.
   (*b*) Permanent { Complete. / Incomplete.
3. *Ventricular :*
   Main Branch Block :
   (1) Right } Temporary or permanent.
   (2) Left }

The conditions of the Bundle which can produce heart block fall into three groups : (1) infective processes, (2) degenerative processes, and (3) the influence of

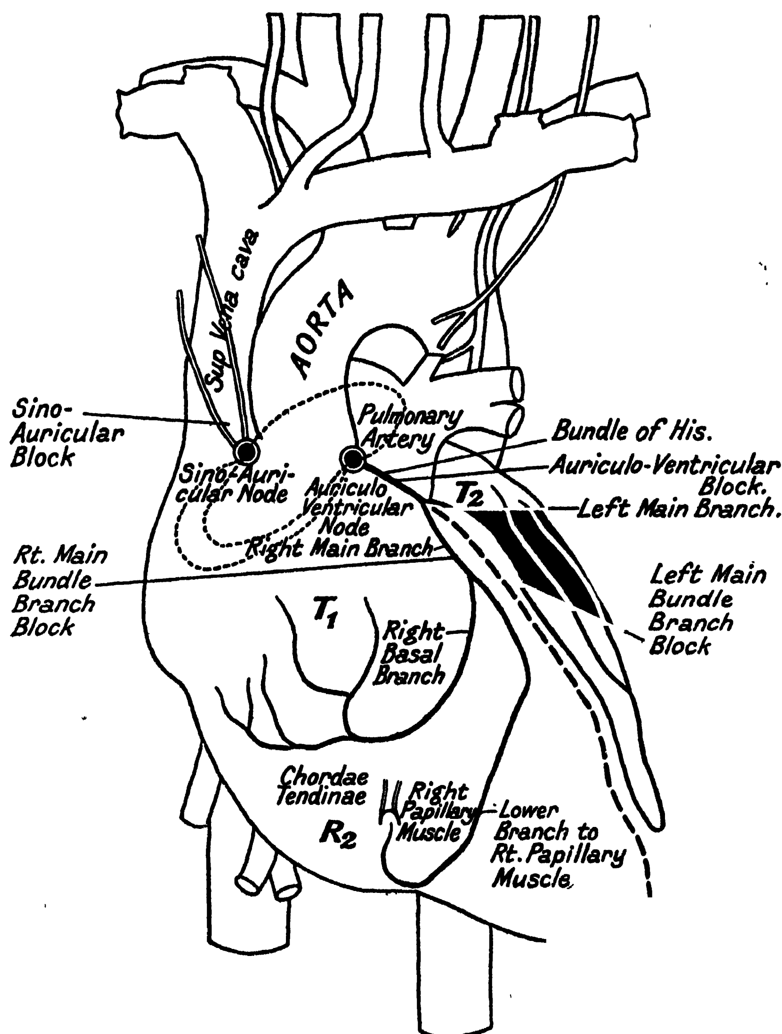


FIG. 38.—Diagram illustrating the positions of the lesions in the different varieties of Heart Block.

drugs. The infective processes are numerous: rheumatism is the chief; diphtheria, pneumonia, influenza, scarlet fever and syphilis are all common. Degenerative processes include fibrosis, the result of old rheumatic fibrotic processes, interference with nutrition through disease of the coronary vessels, and tertiary syphilitic lesions of the heart muscle itself. Digitalis has a pronounced action in lowering conductivity of the Bundle, an effect which is most marked when the Bundle is already diseased, and which is usefully employed in the treatment of auricular fibrillation.

In the milder forms the *prognosis* and the *treatment* are those of the associated disease. Digitalis is usually contra-indicated, owing to its action in lowering conductivity, but in severe cases, where signs of gross heart failure are also present, and rest in bed proves insufficient, digitalis may be serviceable by virtue of its beneficial

action upon the ventricular muscle. In cases in which the possibility of syphilitic changes can be entertained active anti-syphilitic remedies should be employed.

Complete Heart Block implies total interruption of impulse between auricles and ventricles (Fig. 18). When this occurs the ventricles initiate their own rhythm. This is regular, and at a rate which varies from 24–36 (usually 28–30) in different patients, but remains remarkably constant in any individual case. Complete heart block is not infrequently associated with syncopal attacks—**Stokes-Adams' Disease**, first described by R. Adams in 1827. The patients are usually advanced in years, complain of dyspnœa, and have marked bradycardia, the pulse rate ranging from 20 to 40. Any mental excitement is liable to bring on an attack. The attack is due to cerebral anæmia resulting from ventricular standstill, and lasts from four to thirty seconds. The shorter attacks are characterised by transient dizziness, longer ones by brief syncopal periods; in the more severe seizures the breathing becomes stertorous, the face cyanosed, there is dilatation of both pupils, rigidity of the body, accompanied by clonic movements of the limbs. The pulse occasionally ceases for a few seconds, the jaw drops, and for a brief period the patient is to all appearances dead. No pulse is felt in either wrist, and on auscultation the cardiac sounds are inaudible. Then a feeble sound is heard, followed by a stronger, and a second later the pulse begins beating at about 30 per minute (one can feel the artery fill), the cyanosis lessens, the pupils contract, and consciousness returns. Many such fits may occur in succession, from six to ten in a single night.

The *prognosis* in complete heart block depends upon the presence of dyspnœa and other signs and symptoms of failure. Life is often prolonged for years, for the slow heart rate saves the heart muscle.

Treatment.—The attacks may be prevented by the subcutaneous injection of adrenalin which in all probability acts directly on the Bundle of His viâ the sympathetic. Adrenalin in oil (1 in 100) 1 c.c., 3 or 4 times daily, is best for this purpose. The underlying cause must, however, be treated; for this purpose potassium iodide is often very useful. Ephedrine in doses of gr. ½ or gr. 1 three or four times a day by mouth is also useful.

§ 70. *The Heart-beats occur in couples, with a pause after every alternate beat.*

This condition may be apparent or real, and may be due to:

(1) Regularly recurring PREMATURE BEATS; a common cause of which is DIGITALIS overdose. (Fig. 39.)

(2) PULSUS ALTERNANS. (Fig. 39.)

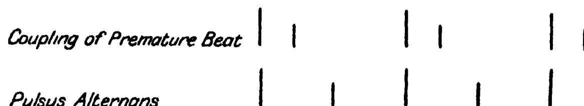


FIG. 39.—In Premature Beat the interval between the beats is unequal; in Pulsus Alternans the interval is equal.

(3) Extreme DICROTISM produces an appearance of coupling, but is distinguished from that due to premature contractions by the fact that the apparent second beat occurs synchronously with the closure of the aortic valves, and is unaccompanied by a systolic heart sound. It occurs where the diastolic pressure is low but the aortic valve competent (§ 88).

§ 71. Pulsus Alternans is a condition in which every second ventricular beat is feebler than its predecessor and the rhythm remains regular. This sign is an evidence of exhaustion of contractility: it may be constant, or only appear occasionally

after premature beats. To distinguish from a coupled pulse see Fig. 39. The condition is of very grave significance when it occurs with a slowly acting heart. With a quick pulse rate it need not be regarded with such grave apprehension, but its appearance is always a warning sign of cardiac exhaustion. Pulsus alternans is most readily shown in a pulse tracing, but can be easily diagnosed by the sphygmomanometer. If the pressure is raised above the systolic figure and allowed slowly to fall, only the alternate stronger beats will at first be heard by the stethoscope. At a lower pressure the weaker beats, evenly spaced between the stronger ones, then appear.

CHAPTER IV

ANEURYSM OF THE AORTA AND OTHER INTRATHORACIC TUMOURS

Anatomy.—The mediastinum is the irregular space in the chest which lies between the two pleural sacs. For descriptive purposes it is divided into four parts—viz., the *middle mediastinum*, which is occupied by the heart and pericardial sac; the *anterior*, which is the space in front; the *posterior*, the space behind; and the *superior*, the space above the pericardial sac. The most important structures contained in these spaces are: The thymus or its remains; the arch of the aorta with its branches (innominate, left subclavian, and left carotid); the superior and inferior venæ cavæ, with the innominate and azygos veins; the pulmonary vessels, the trachea and bronchi; the vagus, recurrent laryngeal, phrenic, and splanchnic nerves; the cardiac and pulmonary plexuses; the roots of the lungs; the œsophagus, thoracic duct, lymphatic glands and vessels, and loose cellular tissue (Fig. 11). The lymphatic glands are important on account of the occurrence of lympho-sarcoma and other glandular enlargements which may form mediastinal tumours.

If, on percussing over the sternum,[1] or just beside it, the præcordial dulness is found to be **increased irregularly upwards**—the morbid condition may be PERICARDIAL EFFUSION, ENLARGEMENT OF THE PULMONARY CONUS, RETRACTION OF THE LUNG, AN ABDOMINAL SWELLING PUSHING UP THE HEART AS A WHOLE, OR AORTIC ANEURYSM OR SOME OTHER MEDIASTINAL TUMOUR. The two last named are generally to be distinguished sooner or later by the presence of pressure symptoms (p. 121). With the aid of X-rays the diagnosis of all mediastinal conditions is usually made clear.

If there is **abnormal dulness** *near the base of the heart, which is accompanied by* PULSATION, *and on auscultation, there is a* REINFORCED OR RINGING SECOND HEART SOUND—*perhaps a systolic or diastolic murmur—the disease is probably* ANEURYSM OF THE AORTA.

§ **80. Intrathoracic Aneurysm.**—In regard to the anatomy of this serious and important malady, the student should study Fig. 11 (p. 51). Aneurysm of the aorta used to be the commonest of intrathoracic tumours. Since the introduction of organic arsenical compounds in the treatment of syphilis, this form of cardio-vascular syphilis has become comparatively rare.

The arch of the aorta is the favourite seat for aneurysmal dilatation. Any part of it may be affected—the ascending, transverse, or descending part of the arch. The dilatation may assume either a fusiform or saccular shape, the former being the more frequent. Fusiform dilatation arises as a rule in the first part of the aorta, and may lead to stretching of the valves and aortic incompetence. The fusiform aneurysm gives rise to practically no physical signs, andthe ensuing description refers, unless otherwise stated,

[1] Remember, in percussing over the sternum, the note elicited is of a much higher pitch than that just beside the sternum.

o saccular aneurysm. It may make its way in various directions, and ones, cartilages, and other hard structures may become eroded and bsorbed under its pressure. According to its position, aneurysm of the orta may be either very easy or very difficult to detect. If it involves he ascending aorta, near the *front* of the chest, it is soon revealed by efinite *physical signs.* If the transverse or descending parts are involved, nd the tumour extends backwards, there may be no physical signs, nd even the *pressure symptoms* may be obscure. Thus the clinical mani-stations belong to two categories—physical signs and pressure symp-oms; and we have two varieties of aneurysm: (*a*) The *aneurysm of hysical signs,* when the ascending aorta is involved; (*b*) The *aneurysm f pressure symptoms,* when the transverse and descending parts of the rta are involved.

The *Symptoms Common* to aortic aneurysm in all positions will be nsidered first, because these are the symptoms which will probably st attract our notice. Then we will turn to certain others *special* to e ascending, transverse and descending parts of the aorta respectively.

Symptoms Common to All Positions:

1. Dyspnœa is often one of the earliest complaints which the patient akes. When it is due to pressure on the trachea, as in aneurysm affect-g the transverse aorta, it is persistent and stridulous in character. hen it is due to narrowing of the coronary arteries by the diseased rtic wall, it is often paroxysmal. Orthopnœa of a marked degree ay be present.

2. Cough is often present and has a characteristic brassy sound ander cough). Pressure upon the recurrent laryngeal nerve is common, th consequent paralysis of the left vocal cord, and there may be hoarse-ss or even aphonia from the same cause. *Paralysis of the left vocal cord* the absence of central nerve lesions, suggests aortic aneurysm or a oplasm. Laryngoscopic examination should be a matter of routine in suspicious cases, because abductor paralysis occurs before complete ralysis, and the former may be unattended by any alteration of voice.

3. Pain is another common symptom; it is felt in the chest, or in the ck. It is frequently worse at night, or on exertion. It may be in e form of angina of effort if the coronary orifices are involved, shooting wn one or both arms, usually the left, especially in aneurysm of the ending aorta. The pain may be neuralgic when there is pressure nerves; or it may be of a dull boring character when due to erosion bone, such as occurs in connection with aneurysm of the ascending or scending aorta. This variety of pain is often worse at night. Short definite anginal attacks of this kind, patients with aortic aneurysm liable to feelings of suffocation, constriction, or "spasm" in the est, and nameless dreads come over them from time to time with-t cause. Such attacks may in many cases be brought on by nding the head backwards, or by any movement which stretches the k.

4. Palpation of the chest provides two signs. Diastolic shock is felt over the aortic base and is synchronous with the second sound. Systolic pulsation may be locally palpable on the thoracic surface, or may be observed by sitting the patient upright and by placing the palm of one hand firmly and flat over the chest and the palm of the other over the corresponding area of the back.

5. A reinforcement of the aortic second sound is the most constant of the auscultatory signs of aortic aneurysm. Dilatation of the first part of the arch gives rise to a ringing aortic second sound, which is quite characteristic.

6. Inequality of the radial pulses is a fairly frequent sign. It is present whenever the aneurysm is so placed as to cause a difference in the arterial pressure in the great vessels which spring from the aorta. The typical aneurysmal pulse occurs in the vessel just beyond the sac, and its characteristic is a decrease of the pulse wave, the blood tending to flow in one continuous stream. Owing to the fact that the radial arteries are often of different sizes the blood pressures on the two sides should be carefully compared to confirm the impression obtained by feeling the pulses. The sign, however, is not diagnostic, for atheroma of the vessels may cause it.

7. Inequality of the pupils occurs from inequality of carotid blood pressure and corresponding inequality of blood-pressure in the vessels of the iris. In the early stages the irritation of the sympathetic nerve causes dilatation of the pupil on the same side. Later on there is paralysis, contraction of the pupil, enophthalmos, ptosis, sometimes vascular dilatation and unilateral sweating of the face and neck (Horner's syndrome).

8. The heart may be displaced when the aneurysm is large, usually to the left.

9. Hæmoptysis may occur; profuse, from rupture, and slight, from the associated pulmonary congestion.

10. Congestive heart failure may occur.

(*a*) Symptoms peculiar to aneurysm of the **ascending** or **first part of the arch.** Aneurysm of this part of the arch is usually easy of detection, and in marked cases the *Physical Signs* are unmistakable. (i.) On inspection, pulsation is often visible over the right upper thoracic spaces. (ii.) On palpation, a diastolic shock may be felt unless the aortic ring is stretched by the aneurysm. Supra-sternal pulsation and a tracheal tug may be felt. The pulsation is felt to be expansile. A systolic thrill may be palpable. (iii.) On percussion, dulness is present, continuous with that of the heart and expanding upwards and outwards from this organ to the right. (iv.) On auscultation, a systolic murmur may or may not be audible. Since the aortic ring is frequently involved, all of the signs and symptoms of aortic incompetence may be present in addition (see § 60). (v.) The right bronchus may be pressed upon, leading to diminished or absent respiratory murmur of the right lung. In severe cases there may be pressure on the superior vena cava, with œdema of the neck

and arms. (vi.) The dyspnœa is paroxysmal; and the right recurrent laryngeal nerve may be involved, with *right laryngeal paralysis.*

(*b*) The symptoms of aneurysm of the second or **transverse part of the arch** may be equally easy to detect when it makes its way forwards. But when the posterior part is affected it may present considerable difficulty in diagnosis, especially from other intrathoracic tumours. (i.) The dyspnœa may be either paroxysmal or continuous, with inspiratory stridor, owing to the pressure upon the trachea. (ii.) Pressure upon the left bronchus may lead to diminished breath sounds in the left lung, partial collapse or bronchiectasis, and symptoms (2) and (5) above are especially marked in aneurysm of the transverse arch. (iii.) Tracheal tugging is a very characteristic sign of aneurysm in this situation. Standing beside the patient, whose head is held level and straight, the examiner defines the cricoid with finger and thumb, and lifts it upwards without backward pressure, away from the thorax. If the aorta is in close contact with the bronchial tree, either by pressure of an aneurysm or by adhering to it by growth, a systolic downward tracheal tug will be felt. (iv.) The physical signs—which are in this situation less marked, or may be absent—consist of a thrill felt on palpating the suprasternal notch; dulness on percussion over the manubrium, continuous with that of the heart, and extending from the middle line to the left of the sternum.

(*c*) Aneurysm affecting the **descending aorta** may be very difficult to diagnose. (i.) Pain in the back and dysphagia are the most constant symptoms. The pain may pass to the side, following the course of an intercostal nerve. It is due to erosion of the vertebræ, which can be demonstrated radiologically. (ii.) Other pressure symptoms are dysphagia, from pressure upon the œsophagus; wasting, from pressure upon the thoracic duct, and signs in the left lung, from pressure upon its bronchi. (iii.) The most diagnostic sign when present is the "Lateral Thoracic Jerk" (Bourne).[1] The whole thorax is jerked to the left during systole. This can best be seen by inspection from the foot of the bed. The jerk is caused by the fact that the ventricles, the aneurysm, and the vertebral column are in direct propinquity, and a thrust is thus transmitted laterally to the left chest wall from the left side of the vertebral bodies when the first two structures become hardened during systole. (iv.) If the swelling enlarges, physical signs on auscultation and percussion may become apparent in the left (occasionally the right) scapular region; and in advanced cases there may even be a pulsating swelling without the knowledge of the patient. Osler found that in some cases there is absence of pulsation in the femoral arteries.

Etiology.—(1) Aortic aneurysm is far more frequent in men than in women, especially in those in the prime of life—namely, between the ages of thirty-five and fifty. (2) It is especially frequent among soldiers, blacksmiths and others who do laborious work, probably due to the fact that these classes are subjected to sudden and severe muscular exertion and

[1] The *Lancet*, 1932, II, 68.

heart-strain at certain times. (3) Syphilis accounts for the large proportion of cases of thoracic aortic aneurysm, and for many of those of the abdominal aorta. (4) Atheroma is the cause in a very small minority of cases. Atheromatous disease generally produces a fusiform aneurysm, and is responsible for a small minority of cases of aneurysm of the descending thoracic aorta and of a higher percentage of aneurysms of the abdominal aorta (§ 263). (5) Some cases of aneurysm date from a period of over-exertion, exposure, and destitution, or from an injury as an exciting or secondary cause.

Diagnosis.—The diagnosis of a deep-seated aneurysm is sometimes difficult in the early stages. The diagnosis from *cardiac valvular disease* is made by the pressure symptoms. Moreover, aneurysm does not cause cardiac enlargement unless there is secondary aortic incompetence. Many of the local signs of a saccular aneurysm may be produced by a *dilated and rigid aorta,* but here the pressure symptoms are wanting. The *throbbing aorta* of hypertension and of aortic regurgitation, as felt in the suprasternal notch, is apt to be mistaken for aortic aneurysm, and it is sometimes difficult to differentiate these conditions. The throbbing aorta in Graves' disease and severe cases of anæmia may also give rise to difficulty. *Mediastinal growths* may have the same pressure symptoms as aneurysm. Pressure upon the veins is more common with growth than with aneurysm, and may only be diagnosed by the absence of the physical signs referable to the heart. There is no murmur on auscultation over the dull region, the area of dulness is usually not so limited or defined, there is usually no expansile pulsation over the tumour, and there are signs of collateral circulation. The course of mediastinal tumours rarely lasts longer than eighteen months. X-ray examination, together with a Wassermann or Kahn test, help to distinguish aortic syphilis and tumour.

Prognosis.—Treatment can do much to prolong life, and the patient may live a good many years if his occupation does not necessitate much exertion. Death may occur from rupture, exhaustion, congestive failure, or complications. Rupture usually leads to a sudden copious hæmorrhage, which terminates life ; but sometimes there is a slight leakage, which may recur each few days. With aneurysm of the *ascending* aorta rupture usually takes place into the pericardium, pulmonary artery, or superior vena cava ; with aneurysm of the *transverse* arch, into the trachea (a very frequent situation) or bronchi ; and, when the *descending* aorta is involved, the blood usually finds its way into the pleura or œsophagus. The process may be so gradual that there is no sudden onset of symptoms, such as dyspnœa, cyanosis, or bleeding, and death may not occur for some time. The severity of any case is measured by the amount of dyspnœa present and the rapidity of the evolution of symptoms. Other consequences or complications are usually due to the effects of pressure—such as collapse of the lung or a low form of pneumonia, hydrothorax, and œdema of the head and neck.

Treatment.—Give a concentrated, nutritious diet ; Tufnell's régime is

very severe.[1] Clear up any septic condition and remove carious teeth where necessary. In syphilitic cases, as there is danger of sudden death if treatment is begun with arseno-benzol, the course of N.A.B. or bismuth injections should be preceded by six weeks of full doses of potassium iodide and mercury. This therapeutic programme should be repeated at six monthly intervals until the Wassermann reaction becomes negative. If the myocardium or liver are defective, arsenic injections should never be given. Large doses of potassium iodide, starting with gr. xx. t.d.s., used to give good results before other antisyphilitic measures were known, *i.e.*, before it was realised that these aneurysms were commonly of syphilitic origin. Penicillin may prove valuable in syphilitic aortitis: as it is likely to produce Herxheimer reactions its use needs careful control. For the pain, morphia injections are used; if of anginal character, nitroglycerin. Even if the dyspnœa is very urgent, tracheotomy is not called for. If there be an external swelling, some elastic support is needed. Phenobarbitone is valuable for palpitation. For venous distension or severe dyspnœa, venesection may be performed. Surgical measures have been adopted from time to time in the treatment of superficial aneurysms, but they are not free from danger.

Other Mediastinal Tumours

§ 81. The *Symptoms and Signs* of **Mediastinal Tumour** belong to three categories—namely, (*a*) the signs of displacement of organs; (*b*) the physical signs of tumour; (*c*) the symptoms of pressure. There are also (*d*) certain symptoms special to the different kinds of tumour.

(*a*) The **displacement** of organs is sometimes the first intimation we receive. The liver is rarely displaced, but the lungs and heart are often moved to one side. The tumours may compress a main bronchus, collapse the lung, and thus draw the trachea and heart towards the same side. These organs will be displaced to the opposite side (i.) if the tumour is very large; (ii.) if it causes a secondary pleural effusion, when the signs of fluid will be present on the affected side.

(*b*) The **physical signs of tumour** may appear on the anterior or posterior aspects of the chest, and consist of: (1) Dulness on percussion, corresponding to the position of the tumour; (2) auscultatory signs, which differ somewhat with the position and nature of the tumour. If it be solid, the breath sounds will be tubular and perhaps differ on the two sides, and there may be an increased conduction of the heart sounds. If it contain fluid (such as aneurysm or, more rarely, hydatid) there will be a diminished respiratory murmur, and in the case of aneurysm a characteristic murmur (§§ 65, 80). (3) Auscultо-percussion will aid in defining the boundaries of the tumour. (4) Radiography is used for defining the nature and position of mediastinal growths, but is generally useless if a pleural effusion is also present. A lipiodol examination of the bronchial tree should be made.

(*c*) The symptoms of mediastinal tumour which are due to **pressure** on the various structures around are as follows:

(1) **Dyspnœa** always appears sooner or later, and may be of a type peculiar to mediastinal tumours when there is pressure upon the trachea and larger bronchi; it has a stridulous character, which resembles tubular breathing heard without the aid of the stethoscope. The breathlessness is often paroxysmal or asthmatic when there

[1] The solids consist of well-cooked meat or fish and biscuit, and for the fluid 10 ounces of milk are permitted per day. From 12 ounces to 18 ounces of solid food may be permitted, but the fluid must not exceed 16 ounces.

is pressure upon the heart and cardiac plexuses; or it may be of a Cheyne-Stokes' nature. But the character of the dyspnœa depends upon whether it is the heart, the great vessels, the bronchi, or the nervous apparatus of the heart, lungs, or larynx, which is pressed upon by the growth of the tumour.

(2) Cough, sometimes of a laryngeal brassy character, is also present, and it is accompanied by expectoration if, as is usual, there is also bronchitis or congestion of the lungs. There may be laryngeal paralysis from pressure upon the recurrent branch of the vagus, and hoarseness, or even aphonia, may result. Hæmoptysis may occur.

(3) Cardiac and circulatory symptoms, such as palpitation, cyanosis, or a difference in the pulses of the two sides in the neck or radial arteries. There may be signs of collateral circulation, with enlarged superficial epigastric and mammary veins.

(4) Dysphagia, from pressure on the gullet, is present chiefly with posterior mediastinal growths, and when the œsophagus is the site of the primary growth.

(5) Inequality of the pupils may appear, owing to pressure on the sympathetic. Usually the pupil on the affected side is contracted from paralysis of the sympathetic, but it may be dilated during the stage of irritation. Other signs of sympathetic paralysis are malar flush, ptosis of the upper lid and enophthalmos.

(6) Pleural effusion occurs if there be pressure on the thoracic veins or if there be growth in the pleura.

(7) The inferior vena cava is rarely compressed, but cyanosis or œdema of the head, neck, and arms may occur from pressure on the superior vena cava.

(8) In suspected tumour of the superior mediastinum, it is well to remember that when the head is thrown back, the veins of the neck become distended, owing to the increased thoracic pressure producing venous obstruction. Dyspnœa is marked, and the sternum may bulge forward.

(9) Pain down the arms and in the back occurs when there is pressure on the spinal nerve trunks. A persistent dull pain is often one of the first symptoms.

(10) Pyrexia is fairly common.

(*d*) **Causes.**—There are certain symptoms which are **special to the nature and situation of the tumour.** There are seven clinical groups of tumours, in addition to aortic aneurysm.

I. Malignant Tumours, which may be primary or secondary. If, in addition to the above physical signs, the expectoration is coloured red or brown by blood, and if on paracentesis a bloody fluid is drawn off from the pleura, the presumption is strongly in favour of malignant tumour. The fluid may contain cells recognisable as malignant. Out of 520 cases of mediastinal tumour, Hare found 134 were cancerous. *Cancer* is a common mediastinal tumour, secondary to cancer of the bronchus or œsophagus. In the latter case it lies in the posterior mediastinum. Primary cancer, as of a bronchus, tends to affect secondarily the posterior mediastinal glands. *Sarcoma*, especially lymphosarcoma, may start in the mediastinal glands as a primary growth, or originate from the pleura and from the thymus remains. Primary sarcoma is most frequent in the superior mediastinum. If secondary in origin (as when the abdominal viscera or sex organs are the seat of the primary tumour), it occupies chiefly the posterior mediastinum. In primary mediastinal sarcoma enlargement of the glands above the clavicle and elsewhere may occur. When the diagnosis of the cause of pressure is obscure, glandular enlargements suggest malignant disease in the mediastinum.

II. Innocent Mediastinal Tumours, though more rare than the foregoing, are sometimes found, *e.g.*, fibroma, dermoid cyst, hydatid. Lipoma, gumma, and enchondroma, the latter growing from the sternum, are also occasionally met with.

III. Enlargement of the Mediastinal Glands.—With these there is often a dulness posteriorly in the upper half of the interscapular space, but occasionally there is dulness over the sternum. Paroxysms of coughing, "croupy" or like whooping-cough, may be present, especially at night, together with stridulous breathing from pressure upon the trachea. The *causes* of enlarged bronchial glands are:

(*a*) As described above, *malignant disease* of the glands is the most common cause.

(*b*) *Tubercle*, generally secondary to tubercle of the lung, is more common in children than in adults. The condition may be suspected when concurrent disease of the lungs is present, and symptoms such as the above arise. If the glands suppurate, sweatings and intermittent temperature become more pronounced than when the lung only is diseased. An abscess may form and open into a bronchus (compare IV below).

(*c*) *Lymphadenoma* (*Hodgkin's disease*) may start in the mediastinal glands, and is then difficult to diagnose from lymphosarcoma. See also § 572.

(*d*) *Bronchitis* and the *pneumonia* which complicates measles, influenza, and whooping-cough, are often attended by enlargement of the bronchial glands, which may occasionally be recognised in children.

(*e*) *Whooping-cough*, without bronchitis or other disease of the lungs, may give rise to swelling of the bronchial glands, although the condition may be hard to make out. Some observers consider that it is the pressure of these glands which causes the paroxysms of whooping-cough.

IV. Suppurative Mediastinitis (abscess of mediastinum) is a rare condition which may affect the anterior or posterior mediastinum, or both, but more often the anterior. (i.) The most prominent symptom is pain, in the site of the inflammation, or passing down the nerves pressed upon. (ii.) Dulness, with œdema and redness, may be present over the upper part of the sternum if the disease be in the anterior mediastinum. Pulsation communicated from the aorta may be present, and lead to a diagnosis of aneurysm, but the pulsation is not expansile, and fluctuation may be felt. (iii.) Pyrexia is present, usually intermittent, with the rigors, sweats and weakness which attend all deep-seated inflammations. (iv.) The presence of leucocytosis is an important diagnostic feature. The causes of acute mediastinitis are trauma, carcinoma of the œsophagus or bronchus. The chronic form is usually due to tuberculous disease, rarely to actinomycosis. It may rupture in various directions.

V. Diffuse Gummatous Mediastinitis, especially affecting the mediastinum, may give all the symptoms and signs of a tumour. The Wassermann reaction and the response to antisyphilitic remedies are diagnostic.

VI. Enlargement of the Thymus.—A certain degree of enlargement is normal to childhood, and may cause dulness over the manubrium. It begins to decrease after the second year of life, and should have disappeared by adult life. In status lymphaticus (§ 37) the thymus may be found enlarged even in adult life. An enlarged thymus is also frequently found in myasthenia gravis, Graves' disease, and rarely in Addison's disease, myxœdema, and rickets. Inflammation, œdema, and tubercle may affect the gland. Tumours may occur—cysts, sarcoma, rarely epithelioma and thymoma. In lymphatic leukæmia a large thymic tumour may be present.

VII. When an enlarged thyroid grows behind the sternum, it may give rise to symptoms of mediastinal tumour.

Diagnosis.—X-rays may give valuable help.

Prognosis.—In cases of intrathoracic tumours which are large enough to produce symptoms the prognosis is unfavourable. Moreover, all of these conditions entail much suffering to the patient. Malignant tumours are fatal in six to twelve months, depending upon the site and progress of the growth. Innocent tumours may last for a long time or may be removable surgically. Syphilitic, tuberculous, and simple inflammatory glandular enlargements may recover under treatment, but even in these no confident prognosis of recovery can be given in any case. Suppurative mediastinitis may open externally or into the pleura, and run a course of a few days or weeks only ; other cases are chronic, and last for years, or lead to pulmonary gangrene and other serious complications when the pus burrows into adjoining organs. An enlarged thymus may lead to sudden death from pressure upon the trachea.

Treatment in intrathoracic tumour is almost wholly palliative. For aneurysm, see § 80. Abscesses, hydatids, dermoids, or growths connected with the sternum may be dealt with by the surgeon in some cases. X-ray and radium applications yield the best results in glandular and malignant tumours. Penicillin therapy is indicated for secondary infection.

CHAPTER V

THE PULSE AND ARTERIES

§ 82. The Meaning of "The Pulse".—The pulse is the wave of increased pressure which passes along the arteries with each contraction of the heart. It is important to distinguish between the transmission of pressure within the arteries and the movement of the blood itself.

The clinical features to be studied in palpation of the pulse are its (1) frequency, (2) rhythm, (3) tension and character, and (4) the state of the arterial wall. These features depend on the frequency and rhythm of contraction of the left ventricle, on the strength of the contractions, and on the output at each beat. They also depend on the elasticity of the arteries and the peripheral resistance encountered by the flow of blood, especially in the arterioles and capillaries. On account of the peripheral resistance the pulse generally ceases at the arterioles, but when the arterioles are relaxed the pulse is often transmitted through the capillaries and may even appear in the veins. *Capillary pulsation* is thus to be seen in a healthy person who has taken exercise on a hot day, and it is a clinical feature of aortic regurgitation (§ 60). In the great veins near the heart a pulse is normally present. Visible *venous pulsation* is to be seen in the veins at the base of the neck in congestive heart failure, and is due to tricuspid regurgitation (§ 59). Venous pulsation is sometimes seen in the veins on the backs of the hands in Graves' disease.

§ 83. Clinical Investigation.—Examination of the pulse provides evidence of great value both as to the state of the circulatory system and the general condition of the subject. Whatever examination is to be made, palpation of the pulse is the first observation to make. If the subject is nervous or emotionally disturbed, or has lately hurried, the observation is repeated later when the pulse has settled. For accurate record the pulse is always taken under similar conditions as to posture, time of day, relation to meals, etc. The radial pulse is generally chosen, since it is easily accessible and lies against bone (the radius). If it is aberrant, the opposite radial artery is palpated. Whenever disease of the cardiovascular system is suspected, both radial pulses should be felt simultaneously and carefully compared. The pulse can also be felt in other arteries near the surface, such as the temporal, facial, dorsalis pedis and posterior tibial arteries, and in the abdominal aorta. To feel the pulse three fingers are placed over the course of the radial artery, the index finger nearest the heart. Allowance is made for the thickness of the subcutaneous tissues.

The special features of the pulse may be brought out more clearly by holding the forearm up when palpating the pulse. The main points to note have been mentioned in § 41. After noting the frequency, rhythm

and character of the pulse (the term character refers to the nature of the pulse wave, its rise, summit and fall), the tension is estimated by the amount of pressure exercised by the forefinger in order to obliterate the pulse wave and prevent it reaching the middle finger. In case there is a return pulse wave through the palmar arches it may be necessary at the same time to obliterate the pulse with the third finger. Finally, after obliterating the pulse by pressure with all three fingers, the wall of the artery is felt by rolling the empty vessel under them.

The SPHYGMOGRAPH is an instrument employed to obtain a record on smoked paper of the characters of the pulse. With Dudgeon's instrument strapped to the wrist, a system of levers magnifies the pulse wave and records a tracing.

The SPHYGMOGRAM or sphygmographic tracing is useful as a graphic record of the pulse, but its readings can never be quite accurate. Fig. 40*a* is a normal pulse tracing. Fig. 40*b* shows the principal named parts of which it consists. The first or *percussion wave* is caused by the arrival of the pulse in the artery under the sphygmograph. Its form is determined by the output per beat of the ventricle, the rate at which the blood is ejected from the ventricle into the aorta, the peripheral

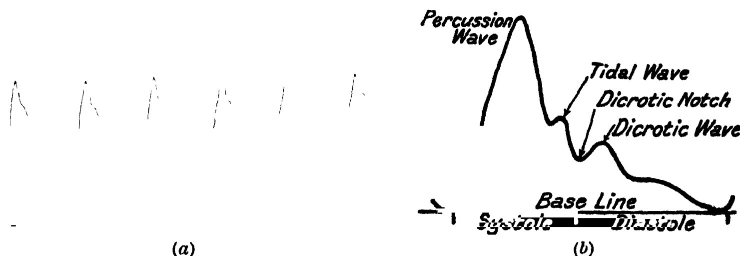


FIG. 40.

(*a*)—NORMAL PULSE TRACING, taken with Dudgeon's Sphygmograph. (*b*)—NORMAL PULSE TRACING magnified, with the names of the principal parts. The dicrotic (or aortic) notch indicates the closure of the aortic valves, and therefore the termination of the ventricular systole and the commencement of the ventricular diastole. The diastolic line is that part of the tracing from the dicrotic notch to the next percussion wave.

resistance and the extensibility of the arterial walls. The percussion wave is abrupt and the pulse is sudden when the diastolic pressure is low and the ventricle has little resistance to overcome in discharging its contents. The *tidal wave* represents, according to Crighton Bramwell, the summation of the outgoing percussion wave, and waves reflected back from the periphery of the arterial field. It is prominent in aortic stenosis and hypertension, for in both conditions ventricular pressure is well maintained throughout systole, and the summation of the percussion and the reflected waves may cause the tidal wave to be higher than the percussion wave. In aortic regurgitation, on the other hand, the ventricle ejects most of its contents during the early part of systole, because of the lower diastolic pressure. During the latter part of systole the ventricular output is much reduced, the falling pressure tends to neutralise the wave reflected from the periphery. Hence the tidal wave is inconspicuous. The *dicrotic wave* indicates the rebound of blood against the closed aortic valve. It is most marked with a forcibly beating heart, a low peripheral resistance, and an elastic arterial wall.

Many instruments have been devised for the **measurement of the blood pressure.** For practical purposes only the aneroid and the mercurial manometers need be considered. Of the former type the Tycos instrument is the best. It is portable and accurate, but it should be

checked against a mercurial manometer from time to time. The mercurial manometer is the more reliable, and of these the Riva-Rocci *sphygmomanometer* is the type on which all are modelled. The column of mercury should be open to direct atmospheric pressure, and unspillable. The armlet is at least 5 inches (12 cms.) wide, and tapers after the first 18 inches. The cuff is better made of fabric than leather, because fabric is more easily adjusted. The Baumanometer type of sphygmomanometer is very good. The armlet is wrapped round the patient's upper arm well above the bend of the elbow. The second turn fixes the upper limit of the armlet, the third turn fixes its lower limit so that, when the rubber bag contained in the armlet is inflated, an even pressure over the whole width of the armlet will be exerted. The sphygmomanometer is placed about the level of the heart. The patient's arm is placed extended on the bed or couch. The position of the brachial artery at the bend of the elbow is located by feeling the pulse in it, and the stethoscope is gently placed over it. The armlet is then inflated until the vessel is so compressed that no sound is heard. The pressure is then evenly released by turning the screw-valve attached to the pump. As the column of mercury falls, four different tones, or phases of sound, will be heard before there is complete silence. (1) The first phase consists of short sharp sounds, and the mercury level at which the first sound is heard indicates the SYSTOLIC PRESSURE. (2) As the column of mercury continues to fall the sound acquires the character of a murmur. This phase is often of short duration, and is on occasion absent. (3) It is succeeded in the third phase by loud and clear sounds; this is the longest and most distinct phase. (4) The clear sounds suddenly become dull and distant; the level of the mercury column at which this occurs is the DIASTOLIC PRESSURE.

The average reading in 150 young soldiers aged 23 to 27, in the fourteenth week of training, was—First phase, 135–125; second phase, 125–104; third phase, 104–80; fourth phase, beginning at 80. The average systolic pressure in this group was, therefore, 135 mm. Hg., and the diastolic 80 mm. Hg. In practice the blood pressure is always recorded in even numbers. Both systolic and diastolic readings in this group are somewhat above the average normal figure (say 10 mm. Hg.). The blood pressure in adolescence and early adult life is often somewhat raised. It returns to normal with maturity.

Having recorded the systolic and diastolic readings by the auditory method as described above, the systolic level is checked by taking the blood pressure again by the tactile method. With a finger on the radial pulse the pressure in the mercury column is quickly raised to 10 or 15 mm. Hg. above the systolic level determined by auscultation. At this level no pulsation is felt in the radial artery. The column of mercury is immediately allowed to fall gently. The level at which the first beat comes through to the radial artery is the systolic pressure. This reading must agree with the reading obtained by auscultation within 10 mm. Hg. The systolic reading as taken by auscultation is often raised above normal

in a nervous subject, and is then usually higher than the tactile reading: occasionally the auscultatory reading is lower than the tactile reading. When a succession of readings is taken the two usually approximate, but when there is still a discrepancy between them the final systolic level should be that obtained when a series of readings taken by the tactile method has given a constant figure.

The PULSE PRESSURE is the difference between the systolic and the diastolic pressure, *e.g.*, with 120 mm. Hg. systolic and 80 mm. Hg. diastolic, the pulse pressure = 40 mm. The DIASTOLIC PRESSURE may be regarded as representing the resistance to be overcome by the heart when the aortic valves are opened. The SYSTOLIC PRESSURE indicates the maximum work of the heart. The diastolic is the more constant, more significant and less liable to alter with nervous influence. The systolic reading normally is as three to two of the diastolic; *e.g.*, S. 120 to D. 80.

This is a rough rule only, the diastolic being more often about 74 mm. Hg. when the systolic pressure is 120. The systolic pressure tends to increase with advancing years. The saying that the systolic pressure should not exceed 100 plus the age is less true than the average generalisation Old people often have nearly normal systolic pressures and many live to a good age with a relatively high systolic pressure. In general terms, a systolic pressure above 170 mm. Hg. or a diastolic above 90 mm. Hg. is pathological. Pressures below these levels may, however, be too high for the particular individual, and of clinical importance.

The following figures give the average found in several thousand actual readings. The range of the normal limit is not more than fifteen millimetres above or below. Age 21–30: 124 (*Systolic*), 82 (*Diastolic*); 31–40: 126 and 84; 41–50: 130 and 86; 51–60: 134 and 90.

§ 84. **Rapid Pulse** (Tachycardia).—The rapidity or frequency of the heart beat varies considerably within the range of normal health due to variations in the rate of impulse production in the normal pace-maker (the sino-auricular node). This is called sinus tachycardia. The normal pulse rate is about 70 per minute. A few people have pulse rates under 60 or over 80, but such must not be accepted as within normal limits without careful consideration. Rarely a pulse rate of 50 or just under, or of 90 or just over, is compatible with perfect *health*. The pulse tends to be faster in the female than in the male. It varies at different ages. In the fœtus and new-born infant its average rate is 140 per minute; under 1 year, 120; under 3 years, 100; from 7 to 14, 90; from 14 to 21, 80; from 21 to 65, 70; in old age, 80 per minute. The pulse is *normally* more rapid during the menstrual period and menopause, in the evenings and after meals. After a severe illness and in asthenic states the pulse more easily becomes rapid. When the tachycardia is due to *simple causes*, not the result of myocardial changes, the number of the beats falls ten to twenty per minute when the patient alters his position from standing to lying. Exercise, emotion, meals, fever and sleep modify the rate, and the electrocardiogram is normal. These features differ-

entiate simple tachycardia from Paroxysmal Tachycardia, in which the pulse-rate is unaffected by posture, exercise, etc.

The **pathological** causes of sinus tachycardia are numerous. (1) *Pyrexia* is the most common. (2) Early *tuberculosis* should always be borne in mind. Any other *bacterial* infection is a common cause, *e.g.*, streptococcal and pneumococcal infections, whether generalised or focal. Pulse frequency is increased in the acute specific fevers, especially in scarlet fever. (3) Of *endogenous toxæmias*; (i.) Graves' disease is the most common; close observation for larval forms of this disease should be made in any obscure case of tachycardia; (ii.) uræmia; (iii.) malignant disease, especially when undergoing degenerative changes; (iv.) all blood diseases, including moderate and severe anæmia. (4) *Exogenous toxæmia* includes a large variety of drugs and poisons, such as tobacco, alcohol, tea, coffee, thyroid extract, belladonna and atropine. (5) *Nervous states*, including ordinary emotional disturbance, often of trivial kind; neurasthenia, anxiety neurosis and neuro-circulatory asthenia are common causes in which the border-line between physiological and pathological disturbance is hard to define. (6) Most forms of *heart disease*, toxic, inflammatory or degenerative, and whether acute or chronic. Increased pulse frequency is an important sign of heart failure. Forms of tachycardia in which the stimulus for contraction arises from an abnormal focus are described in § 66.

§ 85. Slow Pulse (Bradycardia). A slow pulse should be verified by counting the frequency of heart beats on listening to the apex. A frequency of 60 per minute or under requires careful consideration, as it may be the first indication of serious organic disease such as heart block or cerebral tumour. Bradycardia may be a personal idiosyncrasy. It is compatible with perfect *health*. It is sometimes familial. A slow heart rate is an advantage because it allows of an increased cardiac output without increase of heart rate. In a group of 28 Marathon runners examined by Bramwell and Ellis the average heart rate was 58, and 4 of these had heart rates under 50, while only 9 had heart rates over 60. In healthy subjects bradycardia is due to a slow rate of impulse production in the sino-auricular node. It is known as *sinus bradycardia*.

Pathologically, sinus bradycardia may be (1) the result of *reflex nervous effects*, via the vagus nerve; *e.g.*, in gastric disorders. (2) Bradycardia is one of the cardinal features of myxœdema, and other states of *lowered metabolism*, such as exposure to cold, starvation, anorexia nervosa, cachexia and melancholia except in the terminal stages of these conditions. It is associated with low B.M.R. (3) *Toxic conditions*: (*a*) endogenous, such as jaundice, diabetes and uræmia, and (*b*) exogenous, such as may be due to digitalis, strophanthus and opium. At first tobacco may slow the heart. (4) Bradycardia is not uncommon in *convalescence* from acute infection, *e.g.*, influenza, and in exhaustion states. A pulse rate low in proportion to the fever is found with infections by the typhoid and **salmonella groups**, B. coli, and sometimes staphylococcal infections

and influenza. (5) *Increased intracranial pressure* of whatever etiology. In meningitis a slow and irregular pulse is of diagnostic importance. (6) In *heart disease* a slow pulse may be found in aortic stenosis, fatty heart and senile heart. It is the rule, however, that the heart rate increases gradually with advancing years. Bradycardia in heart disease is generally due to heart block (§ 69). *Temporary slowing* of the pulse rate occurs with pressure on the vagus in the neck, and characteristically in an ordinary fainting attack (vaso-vagal slowing).

§ 86. The **Irregular Pulse**, apart from *Sinus arrhythmia,* indicates an abnormal action of the heart. Pulse irregularities are dealt with in § 41, and the heart conditions responsible for them in § 63. A few additional points may be noted here. Sinus arrhythmia (§ 65), which is probably due to rhythmic alterations in vagal tone, is most common in young persons and is generally regarded as physiological. Bramwell has observed the association of pronounced sinus arrhythmia with a liability to simple fainting attacks, and he attributes both to an over-active vagal mechanism. *Premature beats* (§ 64), unless very frequent, can be recognised by the fact of a regular pulse interrupted by an occasional irregularity recurring rarely, or say once in every 5 to 10 beats. The diagnosis is made by auscultation of the heart, and confirmed by electrocardiogram, especially if intrinsic disease of the heart is suspected. *Auricular fibrillation* (§ 68) is recognised by the irregularity in which no two beats or intervals are alike : exercise considerably exaggerates this irregularity and also the rate of the heart, whereas premature beats usually disappear with increased frequency of heart beat.

In auricular fibrillation the apex and pulse rates must be counted at the same time. The pulse rate is generally less than the apex beat—and the difference between the two rates (the " pulse deficit ") is represented thus, A/p. = 124/92. With recovery the pulse deficit becomes less, and it disappears when every ventricular beat reaches the radial artery.

Pulsus alternans (§ 71) denotes alternate weaker and stronger ventricular contractions. It is occasionally diagnosed by recognising an alternate weaker and stronger pulse, but it is readily diagnosed when taking the systolic pressure by the auditory method. At the top level at which the sounds are heard only alternate pulse waves are audible, for the weaker heart beats do not produce a pulse wave strong enough to overcome the resistance of the arm band. Pulsus alternans indicates exhaustion of the heart muscle and is of grave prognostic significance.

In *pulsus paradoxus* there is complete, or almost complete, disappearance of the pulse during inspiration. It is due to either (1) an increase of the " negative " intrathoracic pressure which normally takes place at the end of inspiration, or (2) to extreme weakness in the left ventricle, or to both. It can be produced in even healthy persons at the end of inspiration by so contriving that the negative intrathoracic pressure can be *suddenly* increased. It is met with in intrathoracic tumours, pleural effusion, mediastinitis, and adherent pericardium.

In the *anacrotic pulse* the tidal wave is higher than the percussion wave. It occurs in aortic stenosis and hypertension. It is due to the ventricle being forced to empty its contents more slowly, and so throughout the ejection phase of systole the rate of discharge of blood into the aorta is much more uniform. The pressure being thus maintained when the reflected wave returns from the periphery, it increases the height of the tidal wave. It is only recognisable by instrumental methods.

The *dicrotic pulse* is due to a marked dicrotic wave. It is said to simulate coupled beats, but once felt it is quite distinctive. It is common in asthenic states with a full soft pulse, as in typhoid fever.

§ 87. By **Blood Pressure** is meant the tension in the arterial system. In general terms, it refers to the pressure in the brachial artery. The pressure of blood in the veins is referred to as venous pressure. The blood pressure depends on two main factors—(1) the peripheral resistance; (2) the output of the heart: i.e. the force and frequency of the heart. Any gross variation in blood pressure is due to alteration in one or more of these factors. The determining factor is peripheral resistance.

High Blood Pressure (Syn., Hyperpiesis,[1] Hypertension) is due to many different causes. It is a symptom and not a disease *sui generis*. For the methods of measuring the blood pressure, see § 83. In a healthy adult the blood pressure is fairly constant, but the limits of normal variation are wide, namely—systolic, 100–146 mm., and diastolic, 64–84 mm. In older people the systolic pressure may be as high as 160 mm., but persistent pressures above 170 mm. systolic and 90 mm. diastolic indicate the presence of arterial disease and are certainly pathological.

Temporary hypertension (Symptomatic hypertension), may be due to emotional disturbance and to emotional or physical fatigue. It may occur during convalescence from acute infection and in certain cases of suprarenal tumour. The pressures are rarely higher than 170 mm. systolic and 90 mm. diastolic.

Persistent hypertension. (1) *With Renal Disease.* In any case of persistent hypertension the first object is to determine the presence or absence of kidney disease. Persistent hypertension is common in acute nephritis, in the several varieties of chronic nephritis, in polycystic disease of the kidneys and in toxæmia of pregnancy.

Pyelonephritis, hydronephrosis, renal calculus, renal tuberculosis, hypernephroma, fibrosis of the kidneys, and impaired blood supply from occlusion of the renal artery (*e.g.*, by severe arterio-sclerosis or aneurysm) also can cause renal hypertension. When unilateral, removal of the diseased kidney may allow return of the blood pressure to normal, provided the other kidney is healthy. Interference with emptying of the bladder (*e.g.*, by an enlarged prostate) may produce persistent hypertension.

(2) *Without Renal Disease.* Apart from the fact that heredity is an important factor, the etiology is ill-defined. (i) The patients may be obese, plethoric and jovial-spirited, or thin, pale, anxious and liable to depression. Persistent hypertension is uncommon under 35 years: particularly in younger patients, unilateral renal disease should be excluded by intravenous pyelography. (ii.) Associated with obesity, especially when due to over-eating or excessive drinking. (iii.) Modern city life, worry, anxiety and prolonged mental work, especially when combined with lack of regular exercise. (iv.) Endocrine disturbances, as at the climacteric or after removal of both ovaries. Hypertension may be associ-

[1] *Hyperpiesis* is a state of hypertension, whatever the cause, and may be temporary (as with emotion) or persistent. *Hyperpiesia* is a clinical condition associated with arterial disease (diffuse hyperplastic sclerosis, § 94).

ated with Graves' disease, or with myxœdema. It is part of the syndrome of pituitary basophilism and occurs with adenomata of the adrenal cortex. The relation of Graves' disease to hypertension is probably due to a constitutional predisposition rather than direct cause and effect. (v.) There is an ill-defined relationship with bacterial toxæmia. Chronic urinary infections may cause persistent hypertension: but syphilis, focal sepsis and other chronic infections are not causal. Diabetes mellitus and hypertension often occur together, and after the age of 40 may be due to arterio-sclerosis of the pancreatic vessels (Moschcowitz). Gout and osteo-arthritis are often associated with hypertension. (vi.) The only exogenous poisons are chronic alcoholism and lead. (vii.) For polycythæmia and its relation to hypertension, see § 31.

When a definite cause for hypertension exists, such as a chromaffin tumour of the adrenal or lead poisoning, the raised blood pressure is referred to as symptomatic hypertension, for removal may lead to a relatively normal blood pressure. When the etiological factor is concomitant or contributory, *e.g.*, obesity, or in all cases where no definite factor is found, other than heredity, the condition is called essential hypertension (hyperpiesia).

For the *symptoms* and *treatment* of essential hypertension see § 94.

§ **88. Low Blood Pressure** (Syn. Hypotension) in an adult is indicated by a systolic blood pressure persistently below 90–100 mm.. To the examining finger the pulse wave comes up rapidly, declines rapidly, and is easily obliterated. It may be suspected if the pulse, when counted with the patient erect, is rapid, and the rate falls 30 or 40 beats when the patient lies down. *Symptoms* may be absent. When present, headache, giddiness and sometimes syncope may be complained of, especially when rising from a recumbent posture: depression, lassitude and undue fatigue are usual. In cases of "postural hypotension," the systolic blood pressure is 20–30 mm. less when the patient is standing than when lying down.

Etiology.—In *health* a persistent state of low blood pressure is rarely a hereditary condition. It may be found also after meals, a warm bath or moist heat. In *disease*, the chief causes are (*a*) Cardiac disease, especially weakness of the left ventricle, such as occurs in coronary thrombosis and toxic myocarditis with diphtheria; (*b*) General conditions: (i.) suprarenal atrophy or tuberculosis (Addison's disease); (ii) pulmonary tuberculosis; (iii) cachexia, and deficient food; (iv) shock, collapse, hæmorrhage or dehydration; (v.) exhaustion due to mental or physical overstrain; or following asthenic types of fever, especially typhoid and influenza; (vi.) occasionally with certain types of advanced renal disease and with senile arterio-sclerosis.

The *treatment* of low blood pressure depends upon the cause, but special attention is given to the myocardium. The diet should be nourishing, easily digestible, and rich in vitamins, especially vitamin B. The avoidance of standing about for long, of free purgation, of *hot* baths, and of

mental and physical exhaustion, are all important. Give graduated exercise and an abdominal belt to support the splanchnic area. Nikethamide B.P. (coramine), cod-liver oil, and small doses of strychnine may be helpful. Vaso-constrictors (including ephedrine hydrochloride) may be used, provided the myocardium is not seriously damaged. Stimulants must be used with caution. See also Addison's disease (§ 560). Collapse is dealt with in § 35 and § 239. In severe cases rest in bed is advised.

§ 89. The Pulse in Relation to Prognosis and Treatment of Disease. Examination of the pulse affords valuable information both as to the general condition of the patient and the state of the cardio-vascular system. Indeed there is so much to be learnt from palpation of the pulse by the experienced finger that it should always be the first step in the general examination of a patient. The pulse frequency in *febrile diseases* should be charted four-hourly, so that it may be read in conjunction with the temperature and respiration rate. In an adult the pulse frequency increases 8 to 10 beats per minute for each degree rise of temperature. A pulse frequency increased out of proportion to the rise of temperature may be an indication of a *toxic myocarditis*, and a pulse rate over 130 per minute in *pneumonia* is evidence of severe toxæmia. In a *child*, the increase of pulse frequency with each degree rise of temperature is greater, namely, 12 to 15 beats per minute.

Slowing of the *pulse frequency* in relation to *fever* may be an indication of heart block. A sudden drop of temperature, pulse and respiration rates together takes place at the crisis in pneumonia; but a fall of temperature without a fall in pulse rate, or perhaps even a slight increase of pulse rate, is evidence of a complication. In *abdominal conditions* the pulse rate may decide a diagnosis between inflammation (rapid pulse) and colic (slow pulse), and fall in temperature with an increase of pulse frequency occurs with intestinal hæmorrhage, in perforation of the bowel, and with profuse diarrhœa complicating typhoid fever. The pulse rate may be of outstanding importance in the diagnosis and treatment of appendicitis; in a doubtful case, when the patient looks ill, has indefinite abdominal discomfort but no localised pain, and a soft abdomen, an increasing pulse rate observed half-hourly may be the deciding factor for immediate operation. The pulse-temperature ratio in abdominal disease is considered in § 239. Again, in a patient recovering from a severe *hæmatemesis* the temperature tends to oscillate about normal with an occasional rise to 99° or 99·4°; the pulse rate may be 100–110, gradually falling to 80. With recovery the temperature gradually becomes subnormal at a steady level, and the pulse drops to 70–80. A rise in pulse rate or an irregularity of the pulse curve may accompany further hæmorrhage, and thus provide an indication for more cautious treatment, or if the hæmoglobin is at the borderline of 30–40 per cent. it may determine treatment by blood transfusion. The pulse rate in *afebrile toxic states* is to some extent a measure of the degree of toxæmia, as in alcoholic poisoning, especially delirium tremens. In *Graves' disease* the pulse rate and

the height of the pulse pressure with the patient at rest in bed provide a fair index of the basal metabolic rate and the toxæmia. (See Tachycardia, § 84, for other toxæmias to which these observations also apply.) Pulse frequency to the extent that it is a measure of the *degree of toxæmia* thus provides important information as to prognosis and treatment. Rapidly rising pulse rate is a common terminal event in both febrile and afebrile diseases. A transient increased frequency is some measure of *emotional* reaction. The pulse rate in response to *exercise* and the time taken for its return to normal tells us something of cardiac efficiency. Variations in pulse volume are also of great importance. A full bounding pulse is characteristic of an acute febrile illness and asthenic state. In contrast is the small thready pulse which is felt in all states of shock, both medical and surgical. The pulse in relation to heart disease is discussed in §§ 41, 45.

ARTERIAL DISEASE

PART A.

§ **90.** SYMPTOMATOLOGY. The symptoms of arterial disease *per se* depend in the first place on changes in the function and structure of the blood vessels, and in the second place on the effects of these changes on the activity of the organs which the affected blood vessels supply. The symptoms vary according to whether the vascular affection is general or local, and necessarily according to the part which is chiefly affected. The CARDINAL LOCAL symptom of active arterial disease is **pain.** The outstanding example of this is the pain due to ischæmia of the myocardium; this may be due to structural disease of the coronary arteries, as in coronary thrombosis complicating arterio-sclerosis of the coronary vessels, or due to spasm of the coronary vessels, whether or not associated with structural disease. The pain of active arterial disease may be felt as a rheumatic pain in the limbs or trunk. Arterial disease in the cerebral vessels can cause neuralgic headache, vertigo or tinnitus. The pain of intermittent claudication is due to failure of the blood supply to meet the extra demands of muscular activity. It may occur as a result of healed vascular disease, and does not necessarily imply an active phase of it. Although often not possible of accurate diagnosis, arterio-sclerotic disease may on occasion be the cause of *dyspeptic symptoms*, especially in the form of flatulence or colic. The GENERAL symptoms depend upon the variety and location of the arterial disease. See Part C.

PART B.

§ **91.** The PHYSICAL EXAMINATION of the arteries has been described in the preceding pages which deal with the pulse. The condition of the arteries can be gauged by inspection of the retinal vessels, by palpation of the superficial arteries, by blood pressure observation and by X-ray examination which will show the size of the aorta, and calcification, whether

present in the aorta or in vessels of smaller calibre such as the limb arteries. Finally, examination of the urine may provide an indication of changes in the capillaries of the glomeruli. In fact, in order to arrive at a diagnosis, in certain cases a complete examination of the cardio-vascular and renal systems is necessary.

Physical Signs. (i.) Hæmorrhage is the cardinal sign of the active phase of arterial disease. The common sites are from the nose (epistaxis), uterus (menorrhagia), in the eyes (retinal hæmorrhages or hæmorrhage into the vitreous: conjunctival hæmorrhage has not this significance), and kidneys (microscopic and sometimes macroscopic hæmaturia). Hæmorrhages from the lungs (hæmoptysis), from the stomach (hæmatemesis), and bowels (melæna), are occasionally seen. Such hæmorrhages may be the forerunners of more serious events. Thus epistaxis may precede a cerebral accident, such as hæmorrhage or thrombosis, and is sometimes the first sign of incipient psychosis due to cerebral arterio-sclerosis. (ii.) Visible or palpable thickening or tortuosity of visible (retinal) vessels or palpable vessels, such as the temporal, radial, brachial, and sometimes the carotid and other arteries, indicates arterio-sclerosis, as commonly understood. This thickening of a palpable artery may be due to hypertrophy of the media in response to hypertension. In such a case on post-mortem examination the thickened artery may be found healthy except for the hypertrophy of its walls, which may involve the intima as well as the media. In other cases, such as the senile form of arterio-sclerosis, the thickening and tortuosity of the vessel felt by palpation may be due to degenerative and hyperplastic changes. The thickening may feel irregular, as in the Mönckeberg type of arterio-sclerosis, where there is much calcification of the media, with or without minimal changes in the intima. (iii.) The loss of elasticity in the arterial system in arterio-sclerosis is recognised by an increase of pulse pressure. (iv.) A rise in the diastolic pressure, as occurs in persistent hypertension, also causes a loss of elasticity and increase in pulse pressure. (v.) Accentuation of the aortic second sound is caused by sclerosis of the ascending aorta. With considerable clinical experience this may be distinguished from the accentuation of the aortic second sound due to a persistent raised blood pressure (persistent hypertension), because in the former the second sound often has a quality which is scrunching or clanging, while in the latter the sound is sharp and clear. (vi.) Calcification in the vessels can be demonstrated by radiography. (vii.) Obstruction of the arteries occurs with (*a*) thrombosis due to arterial sclerosis, or syphilitic endarteritis (especially of the cerebral vessels), and (*b*) embolism. Thrombosis less often occurs in a healthy vessel. When blocking has occurred, the arterial pulse is reduced or abolished beyond the obstruction (see §§ 577, 579). Minor degrees of obstruction and complete obstruction may be demonstrated by arteriography.

The arteries are commonly held to be more prone to disease than are the veins, but it is not as yet known in what proportion of cases major

vascular accidents, such as hæmorrhage or thrombosis, are located in the arteries, capillaries or veins. In those areas that are more available for observation, such as the vessels of the nasal septum and retina, capillary and venous accidents are certainly of great importance.

PART C.

The DISEASES OF THE ARTERIES which admit of clinical recognition are as follows:

I. Arterio-sclerosis, of which the following pathological types are recognised: *a*. Nodular arterio-sclerosis, including its terminal phase atheroma; *b*. Senile arterio-sclerosis, including the Mönckeberg type of arterio-sclerosis; *c*. Arterio-capillary fibrosis (Gull and Sutton), otherwise called Diffuse hyperplastic sclerosis (Jores). II. Thrombo-angiitis obliterans (see § 580). III. Periarteritis nodosa. IV. Chronic and acute endarteritis. V. Aneurysmal dilatation. VI. Complications, such as embolism and thrombosis. VII. Functional disease of the arteries.

§ 92. I. *a*. **Nodular Arterio-sclerosis,** including **Atheroma.** This word is derived from the Greek word *ἀθήρη*, which means porridge. It is the name applied to the terminal stage of nodular arterio-sclerosis when a patch of intimal thickening has undergone degeneration and necrosis (Andrewes).

Symptoms are generally absent. Atheroma and the senile type of arterio-sclerosis are commonly associated: the presence of atheroma *per se* cannot be clinically recognised and gives no symptoms (unless a dissecting aneurysm develops).

Atheromatous localised or patchy thickening of the tunica intima occurs for the most part in persons past middle age, and is unaccompanied, as a rule, by any obvious symptoms during life. It starts as a localised hyperplasia in the deeper (external) layer of the tunica intima, or secondary to localised medial degeneration (Virchow). The localised hyperplasia may remain as such, when it appears post-mortem as a raised circular or oval patch of pearly white or pale grey tissue. Or it may undergo fatty degeneration (when it appears yellowish in colour), or when it undergoes necrosis and caseation with or without calcareous degeneration, it becomes atheroma. When the necrotic process involves the superficial layers of the intima and the endothelial lining of the vessel wall an atheromatous ulcer is formed. In advanced cases the middle coat or media is always involved. The disease is more or less widespread, but generally commences and predominates in the larger vessels, *i.e.*, in the aorta and its branches. Although nodular arterio-sclerosis, when marked, tends to be widespread, when detected in the peripheral vessels, such as the radial or temporal, it cannot be concluded that it is also present in the visceral arteries, such as the arteries of the brain, heart and abdominal viscera. This statement also applies to the distribution of senile arterio-sclerosis.

§ 93. I. *b*. **Senile Arterio-sclerosis.** This is a diffuse form of arterial disease chiefly affecting the larger arteries and distributing trunks. It is characterised by widespread medial degeneration, as shown by fibrosis, fatty degeneration and more or less calcification. It is complicated by localised or at least uneven changes of the intima. These changes include

thickening of the subendothelial tissues with intimal hyperplasia, increase of the internal elastic lamina, which may or may not undergo fatty degeneration. It is commonly complicated by nodular arterio-sclerosis. The extreme form of this type of arterio-sclerosis is Mönckeberg's sclerosis.

The *symptoms* form a wide and varied group: (i.) Arterio-sclerotic arteries do not respond so readily to the extra demands made on them by organs in a state of increased activity. As a result, in muscular activity, for instance, anoxæmia may develop. Hence the muscular cramps in the legs on exertion (intermittent claudication) and angina of effort. (ii.) Diseased arteries are more liable to functional disturbance; therefore transient paresis and other cerebral disturbances are common in cerebral arterio-sclerosis. (iii.) The narrowing of the lumen in arterio-sclerotic vessels may lead to impaired vitality and degeneration in the organs they supply; hence the various forms of senile psychosis due to cerebral arterio-sclerosis. (iv.) Other examples are seen in myocardial degeneration, due to disease of the coronary arteries; in diabetes mellitus and in the varied forms of dyspepsia (including flatulence, colic and constipation) due to arterio-sclerosis of the splanchnic vessels. (v.) Arterio-sclerosis contributes to the loss of weight, fatigueability, debility, and many other signs of old age. (vi.) Lastly, arterio-sclerosis may be the determining cause of thrombosis, and thus lead to gangrene, myocardial infarction, cerebral softening and other well-known clinical syndromes.

The *physical signs* due to vascular disease *per se* have already been referred to: (i.) the thickening of the palpable arteries, (ii.) the irregularity of calibre of the retinal vessels with deviation of the veins and obstruction to the blood flow in them at the arterio-venous crossings. (iii.) Increased pulse pressure, generally associated with a rise in systolic pressure, and (iv.) an alteration in the second aortic sound.

Etiology.—(i.) *Heredity.* As Osler said, certain families seem to "inherit bad tubing." A history of arterial degeneration causing "strokes," angina, high blood pressure or "sudden death" is common; and sometimes "anticipation" occurs, so that subsequent generations show the essential changes at earlier ages. (ii.) *Age.* Arterio-sclerosis is rare before 40, apart from kidney disease. Age is an important etiological factor, as indeed the term "senile arterio-sclerosis" indicates. At the same time the changes, described under the heading of senile arterio-sclerosis, are not due only to age, but to other factors, constitutional, dietetic, metabolic, endocrine, infective or toxic, not as yet clearly identified. Senility alone is responsible chiefly for a diffuse thickening of the intima, with increased connective tissue formation in both the intima and the media, and the deposition of calcium salts. There is little fatty or necrotic change. Age is chiefly of importance in providing a longer opportunity for the causes of arterio-sclerosis, whatever they may be, to have their effect. (iii.) *Constitution.* In general terms there is an association between cardio-vascular sclerosis and diabetes mellitus, osteo-arthritis and gout. (iv.) The Mönckeberg type of arterio-sclerosis is chiefly found

in patients suffering from a serious disease such as cancer, chronic phthisis, advanced morbus cordis, and diabetes mellitus. It may be a different lesion from other forms of arterio-sclerosis, and possibly due to an error in calcium metabolism (Moschcowitz). (v.) Hypertension of sufficient degree and duration is an established cause of arterio-sclerosis. Therefore the etiological factors concerned in the causation of persistent hypertension are potential causes of arterio-sclerosis (see § 87).

Diagnosis.—The signs and symptoms of vascular disease already described in detail provide the diagnosis of arterio-sclerosis, but whether the patient who has arterio-sclerosis is or is not suffering from it, will depend on the signs and symptoms of activity of the disease. This is to be judged in the first place by the presence or absence of hæmorrhages and pain, and in the second place on an evaluation of symptoms referable to organs other than blood vessels, and the opinion as to what extent such symptoms are determined by a disorder in structure or function of the vessels which supply them.

Prognosis.—The question of prognosis depends firstly on the activity or quiescence of the arterial disease ; and secondly on the degree of cardiac and renal involvement. If there are signs of heart failure the prognosis is necessarily guarded, and with the development of retinitis and severe kidney involvement it becomes grave. The thickening of the artery, and indeed many of the structural changes which characterise arterio-sclerosis, are largely the result of recovery and repair. The structural pathology of arterio-sclerosis is to some extent comparable with that of fibroid phthisis, in which type of tuberculosis the lesion may be active, but the process of repair keeps pace with it or dominates it. If it is realised that the process of repair may keep pace with the smouldering vascular lesion, and perhaps overtake it, it will be readily understood how often patients have arterio-sclerosis without at any time suffering from it.

§ **94.** I. *c.* **Diffuse Hyperplastic Sclerosis** (arteriolo-sclerosis : arterio-capillary fibrosis) is the structural equivalent of persistent hypertension.

Functional Pathology.—Increased peripheral resistance is the cause of persistent hypertension. It is determined by narrowing of the very small arteries and arterioles, and is accompanied by an increased force of the heart beat. This means increased work for the heart and leads to left ventricular hypertrophy. The blood flow to the periphery is thus maintained in spite of the contraction of the vascular bed. Neither increased output of the heart, nor an increased flow of blood, nor increased viscosity, contribute materially to persistent hypertension. Goldblatt, Page and others have caused persistent hypertension in dogs either by reducing the blood flow to one or both kidneys or by the production of an aseptic capsulitis, thus causing a reduction of arterial pulsation in the kidney, and renal ischæmia. According to Page, the secretion of a pressor substance from the kidney depends rather on reduction of intra-renal pulse pressure than on renal ischæmia. As a result of this change in renal circulation the kidney secretes a substance known as renin, which reacts on a protein-like substance (renin-activator) in the blood plasma to form a heat stable pressor substance called angiotonin (Page and Helmer). It seems probable that other pressor substances are produced. The action of angiotonin is directly on the arterioles and no the heart. As to the mechanism by which angiotonin and like substances cause arteriolar hypertonus, it has been shown that nervous mechanisms have no part

but although the pressor substance seems to be quite distinct from adrenalin, integrity of the adrenal cortex is necessary to allow of the production of hypertension.

Normal kidney tissue prevents renin from exerting its pressor effect, and it would seem that the presence, persistence and severity of hypertension, experimentally produced, is a function of the ratio of ischæmic to normal renal tissue present (Katz). This holds out the hope of isolating an anti-pressor substance from normal kidneys for therapeutic use.

In an animal in which persistent hypertension, comparable to benign hypertension in the human subject, has been produced by an alteration of the intra-renal circulation in one kidney, removal of the other kidney will determine the development of a condition comparable to malignant hypertension in man. The same transition from benign to malignant hypertension may be determined on occasion by the large addition of sodium chloride or meat to the animal's basic diet. This observation gives support to the prescription of a vegetarian diet and restricted salt intake in certain cases of human hypertension.

Structural Pathology.—In contrast to atheroma, which is *localised*, the lesion in diffuse hyperplastic sclerosis (hyperpiesia) is *diffuse*. In contrast to senile arteriosclerosis, in which the lesion greatly affects the media as well as or more than the intima, in diffuse hyperplastic sclerosis the intimal thickening (or hyperplasia) is the distinctive pathological feature. The coincident thickening of the media is explained in terms of physiological response to the persistent hypertension always found in this form of arterial disease. Further, in senile arterio-sclerosis the main incidence of the lesion is in the conducting arteries, whereas in arteriolo-sclerosis it is in the arterioles. The lesion is characterised by the following pathological features. In the terminal arterioles there is intimal thickening due to endothelial or subendothelial proliferation of cells, followed by an increase of hyaline substance and fibrous connective tissue. The process may go on to complete closure of the lumen, and in the terminal stage there is fatty degeneration of the thickened intima, so that in cross section of an arteriole the lumen appears blocked by a plug of fat. In the parent vessel from which the terminal arteriole springs, there is the same intimal thickening accompanied by an increase in thickness and number of strands which form the internal elastic lamina, at the same time an increase of fibrous tissue. There is little or no fatty change in the arteries of this size. In serial sections the marked fatty degeneration of the terminal arterioles may be seen to stop short at their offshoot from the parent vessel (Jores). It is important to note that the lesion known as diffuse hyperplastic sclerosis is recognised by the coincident presence of these particular changes in the terminal arterioles and their parent vessels, because either the lesion as described in the arterioles, or that described in the parent vessels, is found in a number of other disease states, but the two together with a distribution to be described are only found in patients who have had persistent hypertension during life. The initial hyperplasia is accompanied by hypertrophy of the media, and this may be a prominent feature and widely distributed. Hence the term **Arterial Hypermyotrophy** which has been given to it (Savill). The distribution of the lesion is diffuse in the vessels affected. In the terminal arterioles it is often moniliform, and there are varying grades of fat staining with Sudan III. In the parent trunks the initial hyperplasia is very uniform, both in the circumference of the vessel and in its length. In any organ the distribution of the lesion is partial, some vessels being more affected than others, and in the arterioles the lesion may be complete in some, whereas others escape. The organ distribution is characteristic. The lesion is always found in the kidneys or spleen, and generally in both. It is commonly found in the brain, pancreas, and suprarenals, and rarely in the liver or digestive tract; it does not occur as a complete lesion in the heart or skeletal muscle.

Essential Hypertension.—Persistent hypertension without kidney disease was first recognised in this country by Sir Clifford Allbutt, who called it hyperpiesia. Allbutt maintained that hyperpiesia pursues its

course and ends in a cerebral catastrophe or cardiac defeat, or life is terminated by intercurrent disease, but that at no stage of the disease does uræmia develop. The clinical syndrome described by Allbutt as hyperpiesia is now known as Essential Hypertension.

Essential hypertension is diagnosed in a case of persistent hypertension in which primary kidney disease (renal hypertension) and a known cause of hypertension (symptomatic hypertension) have been excluded (§ 87.) It is subdivided into benign and malignant types according to whether the kidneys are normal or are secondarily involved. The term hyperpiesia is no longer used, because it is now known that benign hypertension may (in 10 per cent. of cases) develop malignant hypertension and die of uræmia.

Benign Hypertension. *Symptoms.*—There may be none, especially in the early stage of the disease and in its benign forms. (i.) When symptoms are present, they may be general, such as might be ascribed to neurasthenia, namely, loss of energy, fatigueability, insomnia and nervous exhaustion. (ii.) Cerebral symptoms are common and varied. Headache is often occipital, but may be vertical or frontal, and is sometimes paroxysmal like migraine. (iii.) Dizziness and vertigo, or a sensation of faintness, are common symptoms. (iv.) Actual fainting attacks are less common, but are of more serious import. They vary from slight interruption of the continuity of thought on the one hand to a prolonged faint or epileptiform seizure on the other. (v.) Cardiac symptoms are common : cardiac pain, anginal attacks, palpitation, shortness of breath on exertion, or attacks of nocturnal dyspnœa. (vi.) At a later stage heart failure develops. This may take the form of congestive heart failure with auricular fibrillation or with regular rhythm, cardiac asthma or syncopal attacks.

Signs.—(i.) In established cases the blood pressure is persistently 180 mm. systolic and 90 mm. diastolic, or over; it may be much higher, even reaching 260 mm. systolic and 120 mm. diastolic. (ii.) There is left ventricular hypertrophy, recognised by an increase in the force of the cardiac impulse, an increase in the area of cardiac dulness, and a lengthening and lowered tone of the first sound at the apex. Or it is recognised by an increase in the size of the heart in a radiogram and by left axis deviation in the electrocardiogram. (iii.) The second aortic sound is accentuated. (iv.) The radial pulse is hard and resists compression. The artery is generally felt to be thickened, and it may be tortuous. (v.) The retinal arteries may be pale and contracted while the veins are somewhat full, or there may be signs of retinal arterio-sclerosis, of which irregularity of calibre of the arteries is the most important. Papillœdema is a grave sign. (vi.) The urine is normal, apart from the presence in some cases of a trace of albumin, and slight excess of granular and hyaline casts. Renal function is normal. As was pointed out by Sir Clifford Allbutt, there is no anæmia or other effect of a chronic toxæmia. In fact, in benign essential hypertension the patient is often over-weight and plethoric. The complexion is a good colour in contrast to the pale and

muddy complexion of chronic renal disease accompanied by persistent hypertension. In women more often than men the complexion may be pale and sallow in spite of the absence of kidney disease. In these patients, however, the deep colour of the lips contrasts with the pallor of the face, and indicates a good hæmoglobin content of the blood..

The *differential diagnosis* of hyperpiesia is from the various forms of urinary disease on the one hand and symptomatic hypertension on the other. *Urinary disease* is excluded by the absence of a history of kidney disease or its symptoms, a normal or practically normal urine, normal renal function and pyelography. *Symptomatic hypertension*, such as may be due to heart failure, endocrine disturbance (as at the climacteric, also in certain tumours of the suprarenal gland), and such poisons as alcohol and lead, is diagnosed by recognising the etiological factors, and the diagnosis is confirmed by response to treatment and progress of the patient.

Prognosis. The disease is essentially progressive, but there are some cases which pursue a benign course, and in these progress is very slow. Indeed the blood pressure often remains constant at a level of, say, 200 mm. systolic and 110 mm. diastolic, while only occasional symptoms appearing from time to time show progress of the disease or a failure on the part of compensatory mechanisms to make adjustments necessary to well-being. Some maintain so good a level of health that the disease must be regarded as quiescent, and indeed it is often stationary for a year or more at a time. In the most favourable event the patient may live in good health, with little limitation of his or her activities, for a period of ten years or more, and die from intercurrent disease or natural causes. In other cases, particularly those with a diastolic pressure of 130 mm. or over (in which cases the systolic pressure may be 260 mm. or over), the disease tends to be progressive and ends fatally from heart failure or coronary disease in about 60 per cent. of cases ; from apoplexy in 19 per cent. ; from renal failure in 8 per cent. ; from intercurrent disease in 12 per cent. (Bell and Clawson). It is uncommon for a high blood pressure, once established over a length of time, to return to normal. Nevertheless this may happen on occasion following a severe illness, as after a severe attack of coronary thrombosis : and after treatment of obesity. There is another type in which after the blood pressure has become established at a moderately high level, such as 210 mm. systolic and 100–110 mm. diastolic, there is a period of good health for four to ten years, when without obvious cause the disease suddenly takes on a progressive form and the terminal picture is that of malignant hypertension. In the majority of cases the disease in its general course is slowly progressive, with periods of activity alternating with periods of quiescence, and ending in a cerebral hæmorrhage or thrombosis, in heart failure or intercurrent disease.

Treatment.—There are no means known of arresting these arterial diseases, nor is there any remedy available which will directly promote the healing and repair of diseased arteries. Nevertheless a good deal can be done to improve health and prolong life of these patients. Irksome

restriction of activities is to be avoided unless essential. It is generally better for the person to continue at work provided it is within the limits of the patient's strength. Extra rest, a longer night spent in bed, or an hour spent horizontal between lunch and dinner, may be the means of keeping a man at work without overtaxing his strength. Exposure to cold should be avoided. Patients with arterial disease are more liable than others to feel the effects of chill. Though the part played by infections in causing arterio-sclerosis is doubtful, nevertheless an infection may determine complications of the disease. Thus cerebral thrombosis may supervene a week or two after an attack of tonsillitis or bronchitis in an arterio-sclerotic subject. The habits of life should be regular. Large meals and over-eating are to be avoided, and alcohol should be taken in strict moderation. Constipation must be corrected to prevent straining at stool, and habitual loose stools are weakening. Mild aperients such as rhubarb, aloes, cascara and magnesia are indicated. An occasional colonic douche is good in some cases. A mercury pill followed by a small dose of salts in the morning and taken once or twice a week may be beneficial. The activity of the skin should be promoted by attention to clothing, a daily warm bath, and in some cases a Turkish bath once a week. Some patients seem to benefit by a reduction of animal protein in their *diet*. For these, fish, chicken or meat taken only once daily may be prescribed, or trial may be made of a vegetarian diet, and continued so long as it seems to benefit the patient. The empirical restriction of meat and salt has received support from recent observations on the adverse effect of giving meat and sodium chloride to dogs with hypertension experimentally produced. In other respects the diet should be plain, varied, and easily digestible, care being taken to see that it contains an optimum of protective food substances, especially vitamins B and C. In some cases, where there are signs or symptoms of active disease, or during a progressive phase of the disease, especially when there is reason to think that the complaint is of recent origin, trial may be made of a period of intensive treatment, namely, two to four weeks' rest in bed on a strictly vegetarian diet of low calorie value, together with sedatives, and an occasional small dose of mercury by mouth. *Venesection* is indicated when there is peripheral congestion ; 15–20 fl. oz. of blood are withdrawn. This may be repeated after six months. It is not well tolerated after the age of 65, and is contra-indicated in the presence of kidney disease. *Drug* treatment is chiefly of value for symptomatic treatment. It is important to secure a peaceful sleep at night, and small doses of aspirin, bromide or phenobarbitone are given for this purpose. For heart weakness or failure digitalis should be given. Bicarbonate of soda prescribed with powdered rhubarb and compound infusion of gentian may improve gastric function. Phenobarbitone ¼ gr. is given for relief of restlessness and nervous tension. A change of environment from time to time, residence in a warmer climate during the winter months, and the provision of congenial occupation to take the place of work which has to be given up on account of failing strength, are helpful

measures. Benefit may result from the treatment of associated conditions, as for instance, by reducing a body that is over-weight, by the successful treatment of osteo-arthritis or gout, the control of carbohydrate metabolism when there is diabetes mellitus, and by the eradication of gross sepsis in infected teeth. Lastly, there is a variety of treatment which can be directed towards the control of structural and functional changes in the various organs and tissues of the body which are due to arteriosclerotic disease. Gangrene in the extremities may be delayed or prevented by intermittent venous occlusion or by sympathectomy. The circulation may be improved by such drugs as digitalis, nikethamide (coramine), cardophylin and nicotinic acid. Symptomatic treatment helps to prolong both the patient's life and activities.

When conservative treatment on the above lines fails, and in severe cases, other medicinal or surgical treatment may be advised. Potassium thiocyanate 0·1 G. given in solution t.d.s. after meals relieves headache and giddiness when other measures have failed : in some it reduces blood pressure with beneficial results. The dose varies between the wide limits of 0·8 G. and 4·2 G. weekly : the object is to secure a serum concentration of 6–10 mgm. per cent. : in no case must it be more than 12 mgm. per cent. A serum thiocyanate estimation must be done at the end of the first week and again at the end of the second week : after this it is made at longer intervals; after some months, when the maintenance dose is well established, it is made each 3 or 6 months. Some patients cannot tolerate thiocyanate. A dull red maculo-papular rash may appear in spite of well-regulated dosage with a low serum thiocyanate level. Other symptoms of intolerance or over-dosage are asthenia, extreme tiredness, malaise, pains in the limbs, loss of appetite and nausea : rarely there is hypothyroidism or even myxœdema. On the appearance of any symptom of poisoning the drug is stopped immediately and a serum thiocyanate estimation is done. Potassium thiocyanate is a symptomatic remedy and does not cure the disease, but it is on occasion so effective that patients take it for years. The value of *dorsi-lumbar sympathectomy* is well established. It is advised in severe cases of benign hypertension especially in those between the ages of 25 and 40. (It is not advised after 50 years of age.) The results are better in women than in men. It is generally contra-indicated when hypertension is complicated by heart disease, in those who have had cerebral vascular accidents and in malignant hypertension. Before advising this operation renal efficiency should be tested and intravenous pyelography carried out. When the operation fails in its objective or is only partly successful, treatment with potassium thiocyanate may be effective even when it failed before operation.

Malignant Hypertension.—This disease may suddenly declare itself after an insidious onset, as judged by symptoms, over a period of a few weeks or months.

Structural pathology.—The changes observed are those already described in diffuse hyperplastic sclerosis (§ 94). In malignant hypertension these changes are over-

shadowed and partly obliterated by fibrinoid degeneration of arterioles and acute arteriolar necrosis, and in the more rapid and severe cases hyperplasia may be negligible or absent.

Symptoms.—The disease is recognised by the presence of papillœdema with or without retinal exudates or hæmorrhages, or by a hæmorrhage elsewhere, such as hæmaturia, hæmoptysis, hæmatemesis, and so on. It is suspected when the diastolic pressure is 130 mm. Hg. or over. In addition to macroscopic or microscopic hæmaturia renal involvement is registered by albuminuria, cylindruria, and urea retention of mild degree (§ 401). At the same time, the patient's general condition deteriorates. There is malaise, loss of strength, energy and body-weight, often loss of appetite, and anæmia.

Diagnostic importance has been attached to papillœdema, with or without retinal changes, as described above. However, these changes in the fundi, which are known as hypertensive retinopathy, though generally indicative of malignant hypertension, may in some cases be due to local retinal arterial disease (angio-spasm). In such cases there may be little or no evidence of renal involvement and no general loss of health, as shown for instance by malaise, loss of energy, loss of appetite and anæmia. In such cases intensive treatment, with rest in bed, sedation, perhaps venesection, and the prescription of potassium thiocyanate, or dorsi-lumbar sympathectomy may arrest the disease for a period (within present experience) of five to ten years.

Prognosis. When malignant hypertension is clearly established with retinopathy, renal involvement and anæmia the disease generally ends fatally in six months to two years.

Treatment.—The treatment is on the same lines as that described for benign hypertension. Symptomatic treatment is generally without much effect, but potassium thiocyanate may relieve the severe headache and a course of careful treatment with rest in bed for three or four weeks should always be given a full trial.

Hypertensive Cerebral Attacks (hypertensive encephalitis) are a cerebral form of this. *Symptoms* are sudden headache, drowsiness, coma or convulsions, vomiting and albuminuria. The retinal arterioles are seen to be constricted, retinal hæmorrhages, exudates and papillœdema suddenly appear. The systolic pressure may rise rapidly during a period of a few hours by as much as 100 mm. Hg. The C.S.F. pressure is raised, and there is cerebral œdema. *Diagnosis* is from a cerebral vascular complication of benign hypertension.

Treatment.—In any case of hypertensive cerebral attack the rising blood pressure is an indication for venesection. A raised C.S.F. pressure is an indication for the removal of 10–20 c.c. of cerebro-spinal fluid. Restlessness is controlled by the injection of heroin hydrochloride gr. $\frac{1}{8}$ to gr. $\frac{1}{6}$. Sweating should be promoted and the secretion of urine stimulated.

III. **Periarteritis nodosa** (Syn. polyarteritis nodosa) is a low grade infection of the smaller arteries and arterioles which may be localised or generalised. The essential lesion is a whitish-grey nodule consisting of aggregations of polymorphonuclear cells, together with eosinophils and monocytes. The earliest change is in the adventitia. There is necrosis of the media and proliferation of the intima. Thromboses with infarction and even aneurysms are complications of this lesion.

Symptoms.—The onset may be acute or subacute. In the generalised variety, the patient feels progressively weak and ill, loses weight and later shows mental apathy. There is an irregular temperature with tachycardia. Headache and vague pains in the limbs, and sometimes polyneuritis, suggest a diagnosis of rheumatism. Chest symptoms comprise shortness of breath, cough, sputum and sometimes hæmoptysis, and these are often associated with rales or consolidation of the lungs. Abdominal pain may occur, and thromboses in the abdominal organs at times give rise to gastro-intestinal hæmorrhages or even perforation. Pericarditis or myocardial infarction may be found. The urine almost invariably contains albumen, casts and red cells, and uræmia with or without hypertension is common. A blood count shows a more or less severe degree of anæmia, with a polymorph leucocytosis and rarely an eosinophilia: blood cultures are invariably sterile. In a proportion of cases, one or more palpable skin nodules, the size of a millet seed or a pea, materially aid diagnosis: less often purpuric or vesicular skin lesions are found. The *diagnosis* may be confirmed by biopsy of a skin nodule or of voluntary muscle.

The *prognosis* used to be considered invariably fatal sooner or later, but after a period of many months, recovery can ensue. *Treatment* is symptomatic. Chemotherapy has no effect.

IV. **Chronic** and **Acute Endarteritis**, due to syphilis and other causes, is recognised by its pathological effects (cerebral softening, aneurysm and gangrene). Acute endarteritis has pathological rather than clinical significance. It is common in arteries at the base of chronic ulcers, such as the perforating ulcers of tabes, syringomyelia and diabetes, in new growths, both malignant and benign, in the terminal branches of the coronary arteries in patients dying of rheumatic carditis, in tuberculous, actinomycotic and lymphadenomatous lesions. **Syphilis** affects the arteries in two ways: (1) a proliferation of the intima of small vessels reduces their lumen and interferes with the nutrition of the parts supplied by these vessels (syphilitic endarteritis). This condition also predisposes to thrombosis in the affected vessel, and explains many cases of cerebral thrombosis. (2) A weakening of the muscular coats of the large vessels is seen typically in syphilitic mesaortitis and brought about probably by obliterative changes in the vasa vasorum. With this is commonly associated a proliferation of the intima, especially of the first part of the aorta: it may lead to extensive scarring, and often gives rise to anginal pain.

V. **Aneurysmal Dilatation of the Arteries** belongs to surgery, excepting aneurysm of the thoracic aorta (see § 80), the abdominal aorta (§ 263), and the cerebral arteries.

VI. Complications of vascular disease are:

(*a*) **Embolism**, *i.e.*, the blocking of an artery by an embolus, which may result from heart disease, especially infective endocarditis (§ 50) and mitral stenosis; or may be secondary to thrombosis.

(*b*) **Thrombosis**, the coagulation of blood in the living vessel, results either from local vascular disease or an altered blood state.

Embolism and Thrombosis are dealt with elsewhere. See, for example, §§ 570, 577, 580, Phlebitis and Localised Dropsy.

VII. **§ 95. Other forms of Disease of the Arteries.**—Of functional diseases or vasomotor derangements we know but little, although several important maladies are attributed to this cause—*e.g.*, Raynaud's disease and migraine. Functional derangement of the arteries is also manifested by a large number of symptoms, many of which are vague and evident only to the patient. On this account they are apt to be regarded by medical men as unimportant, and it is true that they are not serious in the sense of being lethal; but to the patient they are often extremely disagreeable, irksome, and often terrifying. Of such we may mention alternate flushing and pallor

("flush-storms"), dead hands, cold hands and feet, chilblains, various other erythematous conditions, blue nose, palpitation, tachycardia (§ 84), paroxysms of copious urination, acroparæsthesia, erythromelalgia, feelings of suffocation, pseudo- and true angina pectoris, feelings of tingling, itching, throbbing, and actual swelling of the limbs. (See § 575 *et seq.*)

CHAPTER VI

THE LUNGS AND PLEURÆ

OWING to the extreme vascularity of the lungs, it is not surprising that inflammation of these organs is a frequent complication of acute general diseases. Thus, inflammation of the lungs is one of the commonest accompaniments of the acute specific fevers and other infective disorders. Overcrowding in badly ventilated rooms increases the risk of infection from persons who have tubercle bacilli in their sputum.

PART A. SYMPTOMATOLOGY.

The **Cardinal Symptoms** of diseases of the lungs are **cough, breathlessness, expectoration,** and sometimes **pain in the chest** and **hæmoptysis.** The general symptoms are pyrexia, sweating, emaciation, and debility. The heart, especially on its right side, suffers sooner or later in all serious or prolonged pulmonary diseases owing to interference with the pulmonary circulation.

§ **101.** Concerning **Cough,** if it is attended by expectoration (as in 1 to 4 below), it points to definite changes either in the lungs, bronchi, or upper respiratory passages. If, without expectoration (as in 5 to 9 below), it may point to simple congestion of the throat or larynx, to the presence of pleurisy, to the early stage of some pulmonary disorder, or to some source of reflex irritation. The *Causes of Cough* are as follows:

1. The commonest form of cough is the recurring WHEEZY cough, attended by expectoration, so typical of bronchitis.

2. PAROXYSMS of coughing followed by vomiting occur in *whooping-cough* and advanced *phthisis. Bronchiectasis* may be attended by paroxysmal cough, with purulent expectoration; so also is the rupture into a bronchus of an empyema, or of a lung or liver abscess. Paroxysmal cough, usually without expectoration, may occur with enlarged bronchial glands and other *mediastinal tumours*: also during attacks of asthma. [See also 8 on page 147.]

3. The HAWKING cough of throat affections is very characteristic, and is met with in catarrhal *pharyngitis*, especially in cigarette smokers, also in chronic laryngitis. It also occurs in *nervous* subjects. A similar type of cough may be found in cases of chronic nasal catarrh associated with *infection of the accessory nasal sinuses*, especially the maxillary antrum. In the latter case a constant hawking cough, with occasional severe paroxysms, is often present, and is relieved only by drainage of the antrum.

4. The IRRITABLE cough, most marked in the early morning and on going to bed, is especially associated with *early phthisis.* There may or may not be much expectoration.

5. A NIGHT cough may be due to chronic congestion of the pharynx, which is sometimes associated with a *long uvula.*

6. The long BARKING or nervous cough of *hysteria* is very characteristic. It is unattended by expectoration.

7. The SHORT SUPPRESSED cough associated with *pleurisy* is so characteristic as to be almost diagnostic; it is unattended by expectoration unless pneumonia is present.

8. The GANDER OR BRASSY cough associated with *aneurysm* and other *mediastinal tumours* is typical, and when once heard is readily recognised.

9. The REFLEX cough, due to irritation in the area of the vagus nerve, may be caused by (i.) *gastro-intestinal* disorders, such as dyspepsia, constipation, diarrhœa, or worms in children; (ii.) *pericarditis;* (iii.) *carious teeth;* and (iv.) *ear* troubles, such as impacted wax; (v.) *abdominal* disease with irritation of the diaphragm—*e.g.*, by subphrenic or liver abscess.

The *Diagnosis* of these varieties of cough is important in practice, since they arise from, and may be seen in, affections other than those of the lungs. When a short dry cough is set up by going into the cold, it may be due to pharyngeal congestion or irritation. In simple throat affections the cough comes on in paroxysms, especially after talking. On the other hand, if such a cough comes on in a warm atmosphere, we should suspect phthisis. In chronic irritation of the larynx or trachea the cough is often worst in the early morning, when a paroxysm is induced by the effort to bring up a little glairy mucus. The face is congested, there is difficult inspiration and even vomiting.

The *Treatment* of cough depends upon the cause, but, in general terms, irritable coughs should be treated by Tr. opii camphorata, by a linctus of squills and tolu, by bromides, by heroin $\frac{1}{12}$ to $\frac{1}{6}$ gr., or codeine gr. $\frac{1}{2}$, or by various medicated lozenges, such as the B.P. morphia and ipecacuanha, or krameria and cocaine lozenge. Coughs associated with tenacious sputum, which is difficult to expectorate, should be treated, not by sedatives, but by alkaline mixtures, with or without the addition of small doses, *e.g.*, gr. 3 of potassium iodide, to loosen the sputum.

§ 102. **Breathlessness**, or dyspnœa, is another symptom of lung affections. The causes of breathlessness are dealt with in more detail in the symptomatology of cardiac disorders (§ 26). The types of breathlessness special to respiratory disorders are:

1. Breathlessness attended by SNIFFING and NASAL BUBBLING is caused by *nasal* or *naso-pharyngeal catarrh.* The obstruction in the nose or mouth in these conditions may cause considerable stertor at night-time.

2. STRIDULOUS respiration, in which the stridor attends both inspiration and expiration, is caused by obstruction in, or *pressure* upon, the trachea or larynx. It is accompanied in severe cases by drawing in of the epigastrium and lower costal cartilages during inspiration (§§ 171, 176 and 177).

3. Dyspnœa attended by considerable WHEEZING or rhonchi in the chest is characteristic of *bronchitis*, accompanied usually by emphysema.

4. CONTINUOUS dyspnœa may be a prominent symptom of gross disease in the chest, such as a large *pleural effusion*: in this the embarrassment of respiration is mainly due to the resulting displacement and pressure on the heart. The dyspnœa associated with extensive *fibrosis of the lung* from any cause, or with *collapse* following *obstruction of the bronchus*, *e.g.* by malignant disease, may be considerable: dyspnœa is often one of the chief symptoms in the last mentioned, and is often disproportionate to the physical signs of disease present. Under this heading comes the expiratory dyspnœa of *emphysema* (§ 142).

5. A rapid respiration with altered PULSE-RESPIRATION RATIO is very suggestive of *pneumonia*. Especially in children there is seen in this disease a characteristic working of the alæ nasi.

6. PAROXYSMAL dyspnœa is present in asthmatic attacks, but is often an indication of *cardiac disorder* (§ 27).

7. The SUDDEN and URGENT dyspnœa of *spontaneous pneumothorax* is a dramatic clinical phenomenon (§ 126).

§ **103. Pain in the Chest** is usually present with affections of the pleura but otherwise it is not a constant symptom in pulmonary disorders. The various causes of pain in the chest are enumerated in § 33. The following are the chief types of pain met in diseases of the lungs:

(i.) The SHARP, cutting, stitch-like pain of *pleurisy*, before the effusion separates the inflamed surfaces, is greatly aggravated by drawing a long breath. This is undoubtedly the commonest of the pulmonary causes of pain in the chest, and this symptom in *pneumonia* indicates involvement of the pleura. It must be remembered, however, that in some *sub-diaphragmatic diseases—e.g.*, of the liver, spleen, or colon—pain is also felt on deep inspiration. One of the most intense forms of pain in the chest is due to *diaphragmatic pleurisy*. It is referred along the lower costal margin, occasionally to the tip of the shoulder, and is accompanied by very shallow respirations, chiefly or entirely thoracic; sometimes there is hiccough. The pain of diaphragmatic pleurisy is occasionally abdominal, when it may cause difficulty in diagnosis, since it may suggest the presence of acute abdominal disease. (ii.) A SORENESS behind the upper part of the sternum attends the onset of *acute tracheitis* and *bronchitis*. (iii.) SUDDEN severe pain, followed by considerable pulmonary distress and general collapse, occurs with the onset of *pneumothorax*. (iv.) SUDDEN pain, attended by hæmoptysis, marks the occurrence of *embolism* of the lung or rupture of an aneurysm into the lung. (v.) *Cancer* of the lung may or may not be accompanied by pain, according to its proximity to the pleura or other sensitive structures. (vi.) *Mediastinal tumours*, including aortic aneurysm (§ 80), give rise sooner or later to pain in the chest. (vii.) Slight pain in the upper intercostal spaces is a frequent symptom in early phthisis: a similar pain is also found in some cases of lung abscess, the site varying according to the position of the abscess.

The presence of **expectoration** or **sputum** is an important sign; its physical appearance may lead to the diagnosis of certain lung diseases. It must be examined by the physician, and it is therefore described in § 111. It must be remembered that children usually swallow sputum; as also do adults with bad habits. Expectoration from the pharynx must not be mistaken for expectoration from the bronchi or lungs. The amount of coughing required to void the sputum may aid diagnosis—*e.g.*, in the early stages of bronchitis much coughing brings up a little tenacious sputum, in the later stages moderate coughing brings up much frothy muco-purulent sputum.

§ **104. Hæmoptysis** means the spitting of blood (*αἷμα*, blood; *πτύω*, I spit), but the term is confined to the expectoration of blood from the organs of respiration. A distinction should be drawn between the expectoration of blood-stained sputum, a common phenomenon in many general diseases attended by severe coughing, and the coughing up of actual free blood; a clear understanding on this point is important in diagnosis, and also in treatment.

The *fallacies* with regard to this symptom are very important. When a patient comes with a history of having "brought up blood," it may at first be difficult to determine whether the blood has come from the stomach, from the lungs, or from the upper respiratory passages (nose, throat, etc.). In the so-called hysterical hæmoptysis, small quantities of thin reddish fluid containing red corpuscles are coughed up; its source is usually the gums or the mucous membrane of the cheeks or pharynx. Although in many such cases the patient is suffering from genuine hysteria, the possibility of deliberate malingering must always be borne in mind.

The differentiation of the various forms of blood-spitting is given more fully under Hæmatemesis (§ 272), but the following points are mentioned here as being characteristic of the issue of blood from the lungs: (i.) It is not infrequently preceded and accompanied by a tickling cough (if the blood be large in quantity it may excite retching on touching the pharynx); (ii.) the patient usually continues to cough up blood for some time afterwards; (iii.) the blood has a bright red colour, is alkaline and frothy, (if very profuse, it may be darker in colour, without froth, and clots may be present); (iv.) physical signs of disease of the lungs are often, though not always, present—they may be absent in the hæmoptysis of early phthisis; (v.) the antecedent history of the patient may point to pulmonary tuberculosis or to cardiac disease, these being undoubtedly the most common causes of hæmoptysis. The above details are given for guidance; in actual practice the distinction between hæmoptysis and hæmatemesis seldom presents real difficulty if care is taken to obtain an accurate history; the descriptions of hæmoptysis given by patients themselves are remarkably constant:—"I felt something suddenly come up in my throat and it was blood."—These or similar words constitute a common statement volunteered by patients. The persistence of stained sputum for a day or two after the initial attack affords further strong

presumptive evidence of true hæmoptysis. The amount of blood expectorated at once may be slight, and the bleeding may be protracted or recurrent; or there may be copious bleeding at one time, and the attack may be fatal within a few minutes. The main causes of hæmoptysis are:

I. PHTHISIS. This is by far the commonest cause, at any rate in young adults, and it is a reasonable clinical rule that definite blood spitting in a young adult, though only to the extent of a drachm or even less, should be assumed to be due to pulmonary tuberculosis until it has been proved to originate from some other cause. The hæmoptysis of phthisis may occur either in the early or in the advanced stage of the disease; in either case it may be small or very large in amount. When the disease is advanced, and bleeding occurs from rupture of an eroded vessel, the issue may be rapidly fatal, even within a few minutes, though death seldom occurs with such suddenness as in rupture of an aortic aneurysm (*q.v.*). Tuberculosis of the lungs may be recognised by: (i.) the previous history of the patient; and (ii.) evidence of congestion, consolidation or excavation of the lung. Nevertheless, the most careful physical examination may fail to reveal any obvious signs; sputum tests and especially X-ray examination are often necessary before the presence of tuberculosis can be ruled out.

II. MITRAL STENOSIS. Here the pulmonary blood pressure is raised and the lungs are congested owing to relative stagnation of blood therein. The patient's cardiac condition is usually known before the hæmoptysis, but in some cases hæmoptysis may be the first symptom which occasions a visit to the doctor. An erroneous diagnosis of phthisis has often been made in these circumstances, even in the absence of tubercle bacilli from the sputum, and when characteristic cardiac signs are present.

III. In BRONCHIECTASIS there may be considerable hæmoptysis. As in phthisis, this may occur at any stage of the disease, but is a prominent and frequently severe symptom in the so-called "dry bronchiectasis" in which, between the attacks of hæmorrhage, the patient may be free from symptoms and physical signs, and only X-ray examination (after the introduction of iodised oil into the bronchi) reveals the cause (see § 143).

IV. In PRIMARY MALIGNANT DISEASE of the bronchus there may be hæmoptysis, seldom severe, except in the advanced stages; even then much hæmorrhage is uncommon. (Repeated small hæmorrhages in a patient of middle-age, or especially one of more advanced years, should raise the suspicion of malignant disease in some portion of the respiratory tract.) *Adenoma* of the bronchus may be the cause of recurrent hæmoptysis.

V. Various PULMONARY DISEASES other than phthisis may be attended by expectoration of blood of varying amount. In *acute tracheo-bronchitis* the sputum may contain streaks of blood from time to time; in *spirochætal bronchitis* the hæmorrhage is likely to be much more definite and pronounced. In *pneumonia* blood in the sputum is a frequent characteristic, the amount varying with the type and bacteriology of the disease. The rusty sputum which is such a diagnostic feature of the pure pneumo-

coccal lobar pneumonia differs from the expectoration of some of the acute broncho-pneumonic conditions following influenza, especially those associated with a hæmolytic streptococcus; in these the whole lung becomes sodden, and almost pure blood may be coughed up in large quantities throughout the acute stage. In *chronic bronchitis* with emphysema the sputum may at times be blood streaked. *Gangrene, abscess, sporotrichosis* and other *fungi* infections, and *hydatid* disease may cause bleeding. *Pulmonary distomatosis* is the cause of so-called endemic hæmoptysis in Japan.

VI. § **105. Pulmonary Infarction** is commonly caused by embolism of one of the branches of the pulmonary artery, but may also be due to a primary thrombosis occurring in the pulmonary vessels. Some have suggested that it may be caused by hæmorrhage into the alveolar spaces from rupture of the pulmonary blood-vessels, hence the expressions "*hæmorrhagic infarction*" and "*pulmonary apoplexy*." Pulmonary infarction complicates mitral disease, and in fact any form of chronic heart failure. It may occur in malignant endocarditis and also in association with septic venous thrombosis, *i.e.*, as a complication of an infective thrombo-phlebitis in any part of the body. It may be seen after abdominal operations, and especially major pelvic operations, and occasionally after an operation for empyema. (It is also known to occur after childbirth.) Small emboli may cause few clinical signs, beyond some pain in the chest. Where a large infarct has been formed, hæmoptysis may be considerable, and pain and respiratory distress severe. In such cases it is common to find at a later date the physical signs of localised pulmonary consolidation, and a friction rub is often audible, owing to the development of a localised dry pleurisy.

When large thrombi are dislodged from distant parts and travel to the lung, the patient may die suddenly at the moment of impaction of the clot in a main branch of the pulmonary artery. In such cases there may be no premonitory symptoms, even the existence of a clot being unsuspected. Post-mortem examination may reveal a large embolus plugging the main branches of the pulmonary artery near its bifurcation.

Treatment. Absolute rest must be insisted on. Morphia may be necessary if the patient is very restless, and oxygen if cyanosis is present. Circulatory stimulants may be required.

VII. Rupture of an ANEURYSM into the trachea or bronchus is usually followed by immediate death, the preceding hæmoptysis being of the most dramatic and appalling character, though in some cases there may be a considerable leakage going on for a day or two before the final issue (§ 80). Apart from such instances of fatal hæmoptysis, a slight degree of blood-spitting is common; hence the occurrence of occasional mild hæmoptysis, if associated with a history of sub-sternal pain, should raise suspicion as to the presence of an aneurysm.

VIII. ULCERATION in some part of the upper respiratory tract (throat, larynx, trachea, etc.). In rare circumstances bleeding from this source

may be considerable, but as a general rule hæmoptysis due to local ulceration or nævi of the upper respiratory passages is small in amount, but apt to be recurrent. The diagnosis depends upon thorough and complete investigation of the respiratory tract, and may necessitate bronchoscopic examination. It should not be too readily concluded that hæmoptysis is due to local causes in the throat; the diagnosis of varicose veins of the pharynx is seldom justified or substantiated, and patients who have been told that their blood-spitting originates from enlarged veins at the back of the throat are usually found on subsequent investigation to be suffering from phthisis or some other serious organic disease.

IX. Purpura, erythræmia, Ayerza's disease, hæmophilia, scurvy, leukæmia, and some other Blood Conditions may be attended by bleeding from the lungs. These causes are rare, but when present are usually recognised, though at first they may not be obvious. Hæmoptysis has been recorded in some of the eruptive fevers.

X. Cardio-Vascular and Renal Disease. Hæmoptysis occurs in subjects of *arterial* and *renal* disease. Hæmorrhages from bronchial arteries have been described in cases of chronic interstitial nephritis. It is certain that hæmoptysis may occur in patients with a *high systemic blood pressure*, associated with a condition of essential hypertension, in whom no serious disease of the lungs exists. Though this cannot be described as common, it is perhaps less rare than is supposed.

XI. Vicarious Menstruation as a cause of hæmoptysis has been alleged by some. It is recognised by its occurrence in association with the menstrual period, usually shortly before this is due, normal menstruation being absent or greatly diminished, and by the absence of evidence of disease in the chest. It must be insisted that vicarious menstruation, though a genuine phenomenon, is a rare cause of hæmoptysis; this diagnosis should not be an excuse for failure to carry out the most complete investigation in cases of hæmoptysis of uncertain origin.

Differential Diagnosis. Although the cause of hæmoptysis may in many instances be obvious on ordinary clinical examination, in certain cases it can only be determined after prolonged, even elaborate, investigation. First, careful inquiry into the history should be made. Next should come examination of the chest by the usual clinical methods, and bacteriological examination of sputum when present. If these measures fail to establish the presence of definite disease of the heart or lungs, X-ray examination must be insisted on; the introduction of iodised oil into the lower respiratory tract may or may not be necessary. If it is still impossible to substantiate the presence of a lesion which will account for the bleeding, the respiratory tract may require further examination by the laryngoscope and/or the bronchoscope. Not until after such complete investigation, and the exclusion of the commoner organic diseases, should it be assumed that the hæmoptysis is due to one of the rarer causes above mentioned in small print.

The *Prognosis* depends upon the cause. Hæmoptysis must be regarded as a serious symptom, at any rate in the first place, and a patient should never be informed that it is of little or no account until complete investigation has elicited not only the cause of the symptom, but the relative severity of the underlying disease. The hæmoptysis of phthisis is of importance chiefly from a diagnostic standpoint; its prognostic signifi-

cance is less obvious, and even in cases of active tuberculosis of the lungs the occurrence of hæmorrhage should not *per se* be taken as necessarily indicating a grave outlook. It may indeed be the first symptom in an early case. It must be regarded as an indication for complete and exhaustive examination by modern methods, the ultimate prognosis depending on the evidence thus acquired as to the patient's condition as a whole.

Treatment. (*a*) For *profuse hæmorrhage* immediate treatment is necessary. It is usually best to allow a semi-recumbent position with the head turned a little to one side to allow more comfortable breathing and greater freedom for coughing and expectoration of the blood. A hypodermic injection of morphine, gr. ¼, is the most efficacious remedy for immediate administration in severe cases; it quietens the patient, who is frequently alarmed and restless. Morphine is by no means always necessary, and should not be prescribed as a routine measure. Since the alveoli and bronchioles are usually full of blood, it is wise to give atropine, gr. 1/100 to gr. 1/50, in combination with morphia. The administration of an extract of blood platelets by intramuscular injection (hæmoplastin or coagulen Ciba), in combination with intravenous injection of 20 c.c. of a 10 per cent. solution of calcium gluconate, is a useful measure which appears to check and sometimes to stop the hæmorrhage in serious cases: 10 c.c. of 1·0 per cent. freshly prepared solution of Congo-red (intravenously) has often proved of value—to avoid reactions it must be passed through a fine filter. Where the hæmorrhage is due to extensive tuberculosis of the lung and if it be definitely known that it is coming from one side, collapse of the affected lung by artificial pneumothorax may be the only effective remedy. In these circumstances one must introduce a much larger amount of air (perhaps up to 1,500 c.c. altogether, or even more) than is ordinarily given in the course of pneumothorax therapy; such a procedure has obvious disadvantages, which can only be disregarded in view of the extreme urgency of the situation. (*b*) When hæmoptysis occurs in *small quantity*, calcium gluconate may be tried, the best plan being to give the drug intravenously as in the more severe cases. The oral administration of calcium salts appears to have little appreciable effect. Intramuscular injections of emetine, gr. ½ every six hours, are sometimes useful. Occasionally a large dose of liquid extract of ergot, *e.g.* ♏ 120 diluted with water, is followed by dramatic cessation of the bleeding. The hæmorrhage of congestion due to cardiac disease should not be checked, unless excessive, as it relieves the pulmonary congestion.

PART B. PHYSICAL EXAMINATION.

Examination of a patient suffering from disease referable to the organs of respiration includes: (*a*) an accurate history; (*b*) careful physical examination of the chest; (*c*) examination of the upper respiratory passages; (*d*) X-ray examination of the chest; (*e*) pathological examination of

sputum, blood, pleural fluids, etc.; (*f*) bronchoscopic examination. It is not suggested that X-ray examination and/or instrumental examination of the respiratory tract are necessary in every case, but in view of the development of modern technique and of the increasing part played by radiological and instrumental methods in the diagnosis and treatment of chest disease, it is well that emphasis should be laid on the limitations of unaided physical examination. Physical signs, although valuable as part of the total evidence, can only give information as to relatively gross structural changes. In many cases diagnosis and treatment are possible only after careful synthesis of the facts supplied by all the above-mentioned methods of examination. The history usually gives a fair guide as to the extent of examination required; time spent in obtaining an inclusive account of the symptomatology is not wasted, and often avoids unnecessary multiplication of special tests.

The physical examination of the lungs is carried out by means of Inspection, Palpation, Percussion, and Auscultation.

§ **106. Inspection.**—The inspection of the chest must be carried out in a good light, and the patient must be instructed to stand or sit erect, or, if in bed, to lie flat and evenly, and to breathe deeply. After noting the movements from the front, examine the back, then look from behind over the clavicles in order to make out the slighter distortions or inequalities of the chest. By inspection we note (1) the rate and character of the breathing; (2) the position of the apex beat (§ 39); (3) the shape and size of the chest. The chief landmarks of the chest are mentioned in § 38. Posteriorly the chest is divided into the suprascapular, scapular, and infrascapular regions. The scapular region is divided, by the scapular spine, into the infra- and supra-spinous regions. The names sufficiently indicate the positions of the various regions.

(1) *Rate and Character of the Breathing.*—The rate varies normally from 15 to 20 per minute, or one-fourth the rate of the pulse; any change in this proportion, or pulse-respiration ratio, should be observed. Notice whether the breathing is rapid, slow, shallow, or irregular. The respiration should be counted without the patient's knowledge; thus while counting the breathing, it is a good plan to feel the radial artery as if you were examining the pulse. Both sides of the chest should move equally. *Any diminution of movement of any part of the chest points to disuse of the underlying lung from disease,* whether new (pleurisy and pneumonia) or old (fibrosis or collapse). Instead of the normal concavity of the intercostal spaces there is flattening or convexity when pleural fluid is present. Drawing in of the interspaces on both sides during inspiration is indicative of some interference with the free entry of air into the lungs (inspiratory dyspnœa), as in diphtheria or other cause of obstruction of the larynx or trachea. The grunting expiration of broncho-pneumonia in children should be especially noted. It is convenient also at this stage to observe whether the alæ nasi are moving. *Cheyne-Stokes'* breathing is a peculiar rhythmical irregularity of breathing (see § 28). When movement of the

chest causes pain, as in pleurisy, or when the muscles of the chest wall are paralysed, there is abdominal breathing. When the diaphragm is out of action, as in certain abdominal conditions, there is exaggerated heaving of the thorax and noisy respiration.

(2) The *position and character of the apex beat* gives an important clue to many diseases of the lungs. Thus effusion and pneumothorax displace it to the opposite side; fibrosis or collapse draw it over to the same side; emphysema masks it.

(3) *The Shape and Size of the Chest.*—A cross-section of the *healthy* adult chest gives almost the form of an ellipse, the longer diameter being from side to side. In the child it is more circular in shape. The chest should appear symmetrical, although in reality the right side is slightly larger than the left. There should be no marked hollowing anywhere; the clavicle should form only a moderate prominence. The circumference of the chest varies with the height of the individual, but it should average about 34 to 35 inches for a man 5 feet 6 inches in height. With deep inspiration it should expand about 1½ to 2 inches or more.

The categoric association of certain alterations in the shape of the chest as a whole with particular diseases can hardly be maintained with such emphasis as is often found in the older text-books. The long narrow, so-called "*phthisical*," chest is by no means always indicative of phthisis, which may occur in a chest of normal shape. The "*rachitic*" chest, with its vertical parasternal grooves and horizontal Harrison's sulcus, is now rarely seen. The *barrel* chest is still common, with its upper ribs crowded together, the lower ribs further apart than normal and the epigastric angle unusually wide. This is traditionally attributed to emphysema, but the association between the two conditions is open to dispute. More important than the general shape and cross-section of the chest are any irregular or *asymmetrical abnormalities*, among which the student should look for *hollowing*, *prominence*, or *contraction*.

(*a*) *Localised Hollowing* or "flattening" of the infraclavicular region may indicate tuberculosis of some standing or any other disease in which fibrosis and lung contraction occur.

(*b*) *Undue Prominence* on one side of the ribs anteriorly may be due to: (i.) Scoliosis—*i.e.*, lateral curvature of the spine, the convexity of the chest being in the opposite direction. (ii.) Intrathoracic tumours, including aneurysm, effusion, abscess, or air (pneumothorax) in the chest. (iii.) If the cardiac region be prominent, it may be the result of cardiac disease in early youth, before the ribs were fully developed. (iv.) An enlarged liver or spleen, abdominal tumour, or abscess may also cause a bulging of the lower ribs on the right and left sides respectively. (v.) Subcutaneous emphysema or œdema, a localised deposit of fat or other tumour. (vi.) Localised muscular over-development, as in athletes.

(*c*) *Contraction* of an *entire side* of the chest may be due to (i.) fibrosis of the lung from whatever cause; (ii.) a previous empyema; (iii.) pulmonary collapse.

During inspection of the lungs it is convenient to note at the same time if there is any abnormal pulsation in the aortic region (see aneurysm), also to note the state of the veins in the neck, the presence of cyanosis of the face and hands, or clubbing of the fingers.

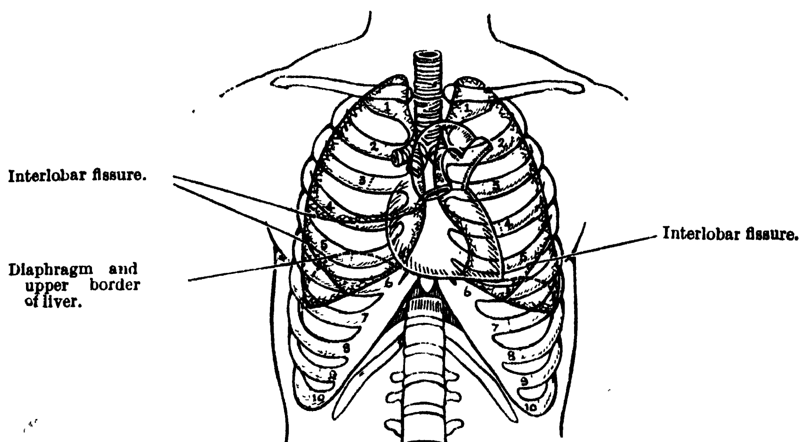
§ 107. **Palpation** is the next step in the routine examination of the lungs. The position of the apex beat should be confirmed. The amount of movement with respiration is estimated better by palpation than by inspection. This test is important in the diagnosis of consolidation at one apex, and in the detection of fluid, tumour, or other cause of deficient activity of one lung or part of a lung. By palpation *Vocal Fremitus* (V.F.), or the vibration of the voice, can be felt. It is scarcely appreciable in women or children with high-pitched voices, but is marked in the adult man. The normal V.F. is slightly greater at the right than at the left apex. Towards the bases it is less intense but almost equal. This test is of the greatest value in differentiating solid and fluid. Thus the V.F. is *increased* where there is consolidation of the lung, as in pneumonia or phthisis, whereas it is *diminished* or absent when the lung is separated from the chest wall by fluid or air, or when air is not entering the larger bronchi, as in obstruction of a bronchus.[1] Not only is the V.F. a valuable differential sign, but its degree of diminution is a useful measure of the *amount* of fluid present in cases of pleural effusion. In bronchitis the rhonchi can be felt, *rhonchial fremitus;* and in pleurisy and pericarditis *friction* may be distinctly felt by the hand. A broken rib, a pointing empyema, subcutaneous emphysema, and external tumours are made out by palpation.

§ 108. **Percussion** is, after palpation, the next step in the examination of the chest. Begin at the apex and percuss *alternate sides* at exactly corresponding points in order *to compare the healthy and unhealthy sides*, and thus work gradually downwards. Place the first or second finger *firmly* and *flat* against the chest, in a horizontal position. Then strike upon it with the tip of the second finger of the right hand. The blow should come from the wrist, not the elbow, and should be short and sharp. Except for the percussion of deep structures (*e.g.*, the dome of the liver) heavy percussion should not be employed.

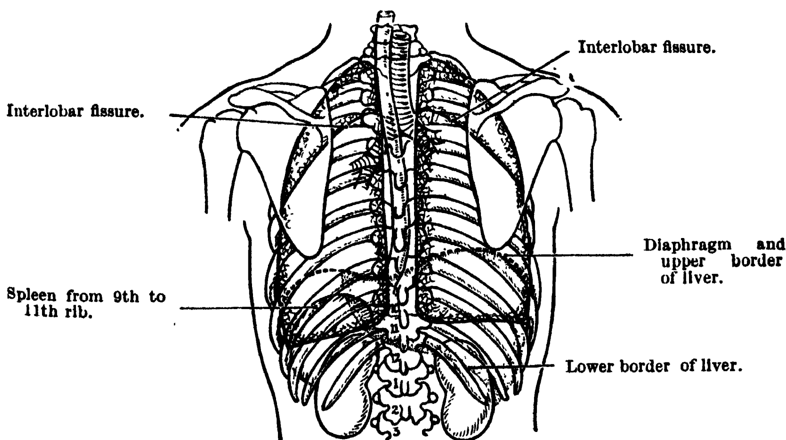
When examining the *back* of the chest (Fig. 41), the patient should be instructed to cross his arms and bend a little forward so that the scapulæ are drawn out of the way. The normal resonance of the lung extends posteriorly to the upper border of the eleventh rib on the right side, and to the lower border of the eleventh rib on the left side. On deep inspiration the resonance extends over an inch lower, and during deep expiration

[1] The absence of vocal resonance on auscultation over a pleural effusion is due to the fact that the lung, being displaced by the fluid, is usually above the level at which the chest-piece of the stethoscope is applied. It is true that water conducts sound better than air when the source of the sound and the auditory reception apparatus are both below the water level. In this case the source of the sound (viz. the larynx) is entirely outside the fluid, and the vibrations are conducted via the trachea and bronchi through the compressed lung to the chest wall.

over an inch higher. Owing to the thickness of the scapular muscles the note over the scapulæ may be markedly impaired in muscular people. For examination of the *sides* of the chest the patient should be told to put his hands on the top of his head.



(*a*) ANTERIOR VIEW OF CHEST.



(*b*) POSTERIOR VIEW OF CHEST.

FIG. 41.

The normal pulmonary note can only be learned by practice and experience, and the student should *frequently practise first on normal chests*, so as to accustom himself to the normal resonance.

The normal percussion note is resonant. It is *dull* or flat when the lung tissue is solid, as in pneumonia ; or when the chest contains fluid,

as in pleural effusion, or with a thickened pleura or a tumour. When a note is said to be dull, it means that the pitch is raised and the volume of the note diminished. Between a dull note and one that is resonant there are all stages of *impairment*. The percussion note is *hyper-resonant*, sometimes tympanitic, whenever the lung tissue is unduly open—*i.e.*, too full of air, as in emphysema, or sometimes when there is air in the pleura (pneumothorax). When one part of the lung is floating above a pleural effusion (which compresses the lower part of the lung), the percussion note is unduly resonant. This kind of resonance is called *Skodaic resonance*; and it may be very resonant and tympanitic (drum-like) in character, somewhat resembling the note normally obtained over the stomach.

Increased Resistance is another quality which can be observed in the process of percussion as above described. It is greatest over fluid, but is present also in consolidation. This sign is used especially by those whose auditory appreciation is imperfect. Subtle differences cannot be appreciated by this means.

§ **109. Auscultation.**—In auscultation there are four things to be observed: (*a*) The intensity and the quality of the respiratory murmur (R.M.); (*b*) the relative length of inspiration and expiration; (*c*) the presence of adventitious sounds within or outside the lungs; and (*d*) the voice sounds or vocal resonance (V.R.).

(*a*) The normal CHARACTER OF THE BREATH SOUNDS—*i.e.*, the vesicular or "respiratory murmur" (R.M.), should be listened to in healthy chests as often as possible. Away from the apex and larger bronchi it has a soft whiffing character. The important feature of vesicular breathing is the absence of any appreciable pause between the inspiratory and the expiratory phases. The R.M. is normally very loud in children, and when a loud R.M. is met with *in adults*, it is called "*puerile* breathing." The breath sounds audible over the right apex are more pronounced than on the left side because of the presence of the eparterial bronchus on this side (cf. p. 162, fallacies no. 9). The difference varies with the age and the build of the patient. The breath sounds are bronchial [1] when the lung is solid, as in tuberculosis, pneumonia, or collapse from any cause. In this condition the sound produced in the glottis is conveyed down the bronchi and smaller tubes direct to the ear, owing to the increased conductivity of the solid tissue. In pleural effusion bronchial breathing is sometimes heard, especially near the angle of the scapula; the effusion causes a collapse of the lung so that the bronchial quality present in the nearest bronchus is well conducted to the surface (Fig. 49). *Bronchial* breathing can be heard *normally* by listening over the upper segment of the sternum, or near the fourth dorsal vertebra at the back. It has two main features: (i.) inspiration and expiration are of approximately equal length

[1] The terms "bronchial" and "tubular" are sometimes taught as synonymous; it is more accurate to say that there are three kinds of bronchial breathing—high-pitched, or tubular; medium-pitched, or true bronchial breathing; and low-pitched, or cavernous breathing.

and character, or the expiratory phase may be obviously prolonged; (ii.) there is an appreciable interval between the inspiratory and the expiratory phases. One important thing to realise about bronchial breathing is that its recognition depends on quality rather than quantity of sound—there is usually less sound with a diseased lung; the R.M. also is weak over thickened pleura or fluid. *Cavernous* respiration is low-pitched bronchial breathing, and is heard when the sound produced in a dilated bronchus or cavity is conveyed to the surface. Cavernous respiration is normally heard over the trachea. *Amphoric* breathing is a sound like air entering a bell-jar, and is sometimes heard over pneumothorax or a very large cavity.

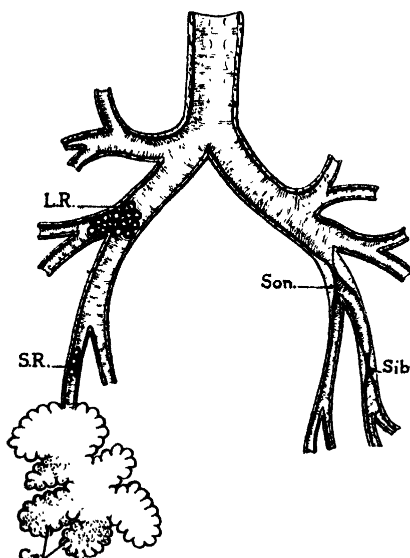


FIG. 42.—Diagram to show the production of **râles** (moist sounds) on left, by mucus in bronchial tubes, and **rhonchi** (dry sounds) on right, by narrowing of the tubes. **Moist sounds, Crepitations,** may occur in vesicles of lungs near base.

L.R. Large bubbling râles, S.R. Small râles, Cr. Fine râles (crepitations), Son. Sonorous rhonchi, Sib. Sibilant rhonchi.

(*b*) Heard through the stethoscope, the inspiratory sound is normally three times as long as the expiratory sound, which follows it without a pause. The *process* of expiration is much longer than inspiration, but through the stethoscope most of the former is unheard because the velocity of the air-current is low. *Expiration is prolonged* in any disease which involves a loss of elasticity of the lung tissue, such as emphysema, or an increase in conductivity, as in consolidation.

(*c*) The presence or absence of ADVENTITIOUS SOUNDS has next to be noted. These may be either dry or moist.

DRY SOUNDS.—(i.) *Pleural friction* is produced by the two inflamed

and roughened surfaces of the pleura rubbing together. The sound has been likened to the creaking of leather. It is generally heard both in inspiration and in expiration, is often intensified by pressure with the stethoscope and by deep breathing, and is not abolished by coughing. (ii.) Within the respiratory passages *rhonchi* may be added to the respiratory murmur. These are continuous sounds due to narrowing of the bronchial lumen, either by swelling of the mucosa, *e.g.* in bronchitis, or by spasm of the bronchial muscle, as in asthma. When low-pitched they are described as *sonorous* (produced in the larger tubes), when high-pitched as *sibilant* or *whistling* (when the smaller tubes are concerned).

MOIST SOUNDS.—These are known as *râles*, and are due to the presence of mucus or other fluid in the bronchial tubes. Râles are of three main varieties,[1] according to the size of the tubes involved and the amount of fluid present, viz. large or bubbling, medium, and fine râles. When the finest tubes are affected the alveoli do not escape, and the finest râles, often known as *crepitations*, are due to the opening up during inspiration of alveoli, the walls of which have been kept in apposition by a thin layer of moist secretion. They have been likened to the sound produced by the rustling of tissue-paper near the ear. When these added sounds are few and difficult to detect, they may become clearer when the patient draws a deep inspiration immediately after a slight cough (post-tussic râles). Crepitations sometimes resemble friction sounds, but are distinguished by being audible only during inspiration, and by being altered by coughing.

(*d*) The VOICE SOUNDS, or vocal resonance (V.R.). (i.) When the patient speaks, the vocal resonance is INCREASED (*bronchophony*) if the conductivity of the lung substance is rendered greater by consolidation, such as that produced by tuberculosis or pneumonia. If this be so great that even whispered words are distinctly conducted, it is known as *whispering pectoriloquy*. (ii.) The vocal resonance is DIMINISHED when a layer of fluid or air intervenes between the lung and the chest wall (*e.g.*, in pleural effusion and pneumothorax), or when there is a thickened pleura. Nevertheless, in a pleural effusion, at the upper level of the fluid, the higher tones of the voice sounds are sometimes conducted, and have been likened to the bleating of a goat (hence called *Ægophony*). Transference of the voice sounds depends upon patency of the bronchi; in any condition therefore in which there is gross obstruction of the bronchus or its main divisions, *e.g.*, by a growth, the vocal resonance is diminished or lost.

The COIN or BELL sound is a sign of some value. To elicit this, a large coin is laid flat on the chest and is tapped by another coin; the mouthpiece of the stethoscope is placed some distance away, but not on

[1] Various terms have been used to distinguish different types of râles, *e.g.* consonating, clicking, crepitant, and so forth. These are a matter of the personal factor of the individual auscultator, and are apt to confuse the student. The essentials of classification are contained in the comparatively simple division just described.

the same rib or over the stomach. A bell sound is pathognomonic of pneumothorax.

Clinically, all the diseases of the lungs may be conveniently divided into those with **dulness on percussion,** those in which the percussion note is **normal,** and those in which it is **hyper-resonant.** Those with **dulness** may be subdivided into two groups—those in which the dulness is due to CONSOLIDATION, and those in which it is due to FLUID. The clinical features by which solidification of the lung is distinguished from fluid in the chest are so important that they are given in tabular form.

TABLE V.—DIFFERENTIATION OF SOLID LUNG FROM FLUID IN THE CHEST.

	Consolidation of Lung.	Pleural Effusion.
INSPECTION. ..	Movement impaired. .. .. May be flattening over the part (if infraclavicular region).	Movement impaired. May be bulging (of intercostal spaces).
PALPATION. ..	V.F. INCREASED.	V.F. DIMINISHED or absent.
PERCUSSION. ..	Resonance impaired.	Absolutely dull over liquid.
AUSCULTATION.	BREATHING BRONCHIAL. .. V.R. INCREASED.	R.M. ABSENT OR WEAK. V.R. DIMINISHED.
HEART	In normal position (pneumonia), or pulled towards affected side (fibrosis or collapse).	Displaced to the opposite side.

Fallacies in Diagnosis of Diseases of the Chest.—This list includes the most important fallacies, but it is impossible to make it exhaustive.

1. When the chest wall is very thin the sounds heard on auscultation are proportionately loud. The percussion note is also more resonant, and it is consequently easy to fall into the error of supposing that emphysema is present. In children the breath sounds are always more distinct than in adults, and are, moreover, more readily conducted, so that adventitious sounds having their origin on one side may even be heard quite plainly on the other.

2. A chest wall with excess of muscular development, subcutaneous fat or œdema will give rise to error if it be not borne in mind that the sounds on auscultation and percussion are alike deadened and indistinct. The sounds heard over the scapular region are always less distinct than those heard elsewhere. When a patient does not breathe deeply, owing to enfeeblement or pain on movement of the chest, or when the chest wall is very fat, the breath sounds may be almost inaudible.

3. The presence of much hair on the chest, as it is rubbed by the stethoscope, gives rise to sounds like fine crepitations.

4. The sounds in the subcutaneous and fascial tissues round the shoulder joint often lead to mistaken diagnosis of pleurisy at the apex (scapular creak).

5. Care should also be taken to hold the chest piece firmly and flat on the skin.

6. It is well to remember that dulness on percussion does not necessarily mean that there is fluid or consolidation present. It may also be caused by thickened pleura and by a tumour. The latter may be outside the chest, but pushing up into the thorax—*e.g.*, hepatic or splenic enlargement, subdiaphragmatic abscess.

7. Tumours of the chest wall will sometimes lead to the impression that there is some difference in the size of the two sides of the thorax, and this difference may be referred to some morbid condition of the chest contents. The swelling caused by subcutaneous emphysema or blood clot, both of which may follow an accident, gives

rise to a faint crepitation which may be easily mistaken for the signs of injury to the lung beneath.

8. When one lung has been long out of action, as in fibroid phthisis, the other undergoes compensatory enlargement and encroaches on the affected side of the chest. The hypertrophied lung gives rise to sounds identical with those of emphysema.

9. The breath sounds are better heard and the percussion note is higher at the right than at the left apex, owing to the presence of the eparterial bronchus on the right side. The area over which the bronchial quality can be detected is also larger.

10. Atrophy of the muscular tissues about one shoulder leads to an apparent flattening on that side very like that seen in phthisis.

11. Dextro-cardia is very rare, but it is necessary to be on one's guard lest it be rashly supposed that the heart is displaced by effusion or by some tumour.

12. Finally, it is well to remember that the presence of lung signs usually found in association with acute disease must always be interpreted with due regard to the constitutional condition and co-existing signs of disease in other organs.

13. Distension of the abdominal organs, as in meteorism, may extend high up into the chest and simulate hyper-resonance of the lungs. This is especially probable when the lungs have been drawn up with adhesions or fibroid contraction or when one half of the diaphragm is paralysed.

14. **Hernia of the Diaphragm** is rare and often unsuspected. Usually congenital in origin, it occurs chiefly through the left half of the diaphragm, so that the stomach, small intestine, colon, omentum or spleen may become intrathoracic.

The chief *symptoms* are pain, often in the left hypochondrium or left shoulder-tip, coming on during or after a meal, with a feeling of extreme distention which is relieved by vomiting or by eructation. Other symptoms include dyspnœa, palpitation, dysphagia, and those of intestinal obstruction. The *physical signs* include hyper-resonance in the lower third of the left chest (often mistaken for a pneumothorax), cardiac displacement and cyanosis. Symptoms and signs may be entirely absent. The true state of affairs can only be diagnosed by X-ray screening, and with a barium meal or barium enema.

§ **110. Radiology of the Chest.**—X-ray examination often reveals the presence of disease in the lungs when other methods of examination give a negative result. Its value is, perhaps, best exemplified in the case of early tuberculosis, indubitable evidence of which may be furnished by a radiogram when the most careful and competent clinician has been unable to detect any abnormal physical signs in the chest. Good radiological work is essential for the diagnosis of many cases of bronchiectasis and of new growths of the lungs and bronchi. Not only in diagnosis is X-ray important; in treatment, and especially in thoracic surgery, it is a *sine qua non.* For ideal work in a difficult case the investigation should be carried out by physician, surgeon and radiologist co-operating in a team. For practical purposes the physician must often interpret his own radiograms. Examination with the fluorescent screen is an important preliminary; by this means it is possible to determine any abnormality in the movements of the diaphragm and to observe various other features which cannot be demonstrated by radiography alone. The ordinary straightforward X-ray film may suffice to show the variations from the normal which characterise the commoner forms of intrathoracic disease; many conditions cannot be adequately shown without the use of iodised oil. When iodised oil (a preparation of 40% iodine suspended in poppy-seed oil, with which it is in loose chemical combination), is introduced into the

bronchial tree, the latter is rendered opaque to X-rays and can be outlined in a radiogram (bronchogram). Detailed information is thus afforded, *e.g.*, as to the existence of cylindrical or saccular dilatation of the bronchial tubes (bronchiectasis), as to the exact size and position of cavities, sinuses, etc., in the lung, and as to bronchial obstruction due to new growths. A *Barium Meal* examination may be useful for the investigation of new growths pressing on the œsophagus, or in cases of œsophago-bronchial fistula.

Mass Miniature Radiography has materially advanced the preventive treatment of chest disease. With a 35-mm. ciné film in a specially designed apparatus, the radiologist can examine a large number of individuals in rapid succession : these miniature films are subsequently projected on a screen, and in the enlarged image it is possible to detect definite or suspicious abnormalities. Any individual revealing abnormalities in the miniature film is subsequently X-rayed on a standard apparatus with a 15″ × 12″ film, and submitted to such clinical, bacteriological and other investigations as are necessary to establish a diagnosis :—this should not be made from the miniature film alone. In very large numbers of ostensibly healthy individuals thus examined in numerous surveys, a small but definite proportion have shown lesions of the lungs, heart or mediastinum (and see § 131).

Tomography. In a special form of X-ray apparatus, recently introduced, the tube, instead of being fixed, moves across an arc during the exposure ; a corresponding synchronous movement of the cassette containing the film takes place in the opposite direction. As a result certain of the rays pass always through one point on the film, the remainder passing through different points and giving an image which is blurred or even invisible while the fixed point image is distinct. For this reason, and as the focal point of the tube can be altered so as to centre the fixed rays at different depths of the chest, it is possible, by taking a series of radiographs, accurately to delineate various details which are not adumbrated in the ordinary flat radiograph, and to obtain more exact localisation of cavities, etc., in the lung. The tomograph has a limited sphere of application, but within that sphere its value is inestimable.

§ 111. Examination of the Sputum.[1]—Much may be learned from an examination of the sputum. First, as regards its APPEARANCE. Watery sputum is expectorated in large quantity in œdema of the lungs. If the disease be confined to a moderate catarrhal process of the bronchial tubes (*e.g.*, bronchitis), the sputum is white, clear, and frothy (" mucous expectoration "). If the process be more severe and suppurative, or if the lung tissue be breaking down, then pus is present, and the sputum is yellowish (mucopurulent). In phthisis, when the lung is breaking down, the sputum is often voided in thick purulent masses like coins, hence called *nummular*. In cases of pulmonary abscesses, tuberculous cavities, and of empyema bursting into the lung, large quantities of almost *pure pus* are expectorated from time to time. Extremely fœtid expectoration is voided in gangrene of the lungs and in bronchiectasis. In *pneumonia* the sputum is very characteristic, being (i.) almost airless and extremely viscid, so that the vessel containing it may be inverted without spilling it, and (ii.) frequently tinged with blood, thus having the colour of rust. In severe cases, and in new growth of the lung, the sputum becomes thinner, frothy, and dark red, the " prune-juice " sputum. *Casts* of the bronchial tubes, which can

[1] It is important to ensure that the specimen examined is really sputum and not merely saliva or nasal secretion.



FIG. 43.—RADIOGRAM OF A NORMAL CHEST (FEMALE). The relative want of translucency in the lower zones of the lung fields is due to opacity caused by the mammary glands.

FIG. 44.—RADIOGRAM FROM A CASE OF BILATERAL BRONCHIECTASIS. The dilated bronchi on the left side have been outlined by iodised oil.



FIG. 45.—RADIOGRAM FROM A CASE OF PULMONARY TUBERCULOSIS, showing a fairly large cavity in the left upper lobe.

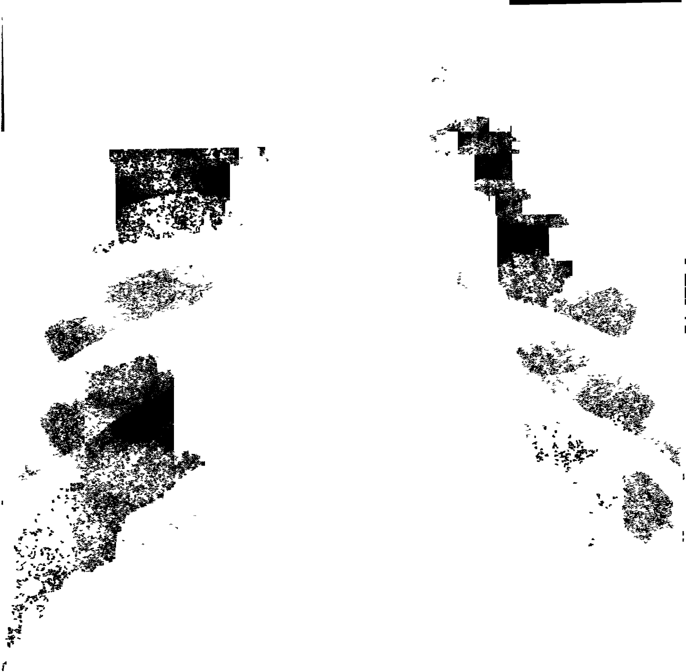


FIG. 46.—RADIOGRAM OF CHRONIC PULMONARY TUBERCULOSIS. Infiltration and fibrosis at both apices. A few areas of calcification in both upper lobes—especially on the left side.

FIG. 47.—RADIOGRAM FROM A CASE OF PRIMARY BRONCHIAL CARCINOMA, showing a dense opacity in the right upper zone due to collapse of the upper lobe from obstruction of the eparterial bronchus by growth.

FIG. 48.—RADIOGRAM FROM A CASE OF SILICOSIS in a coal-miner, showing general reticulation of both lung fields with early nodulation.

be seen by the naked eye, are expectorated in plastic bronchitis, and occasionally in bronchial pneumonia, and shreds of membrane in diphtheria. Hydatid cysts, resembling empty gooseberry-skins, are expectorated in that rare condition hydatid disease of the lungs, or when hydatid of the liver ruptures into them. In town dwellers, and those with dusty occupations, the sputum is dark, or even black, from the presence of carbonaceous and other particles. " Anchovy sauce " coloured sputum is characteristic of abscess of the liver which has burst into the lung (§ 336).

Microscopic Examination of the Sputum.—Various *bacteria* and *fungi* (*e.g.*, tubercle, pneumococcus, influenza, pyogenic cocci, anthrax, glanders, plague, spirochætes, the fungi of actinomycosis, blastomycosis, and aspergillosis) may be found in the sputum. Various *parasites* (streptothrix, echinococcus, Distoma pulmonale, etc.) are sometimes found in the sputum. Sarcinæ and Oïdium albicans come usually from the mouth. The method of detecting these is described in Chapter XXI.

In all destructive diseases of the lung, especially gangrene and abscess, fragments of pulmonary tissue are present—*i.e.*, epithelial cells and connective tissue. The most characteristic is *elastic tissue.* Elastic fibres are best revealed by taking a small portion of the sputum and boiling it with liquor potassæ, which breaks up and renders clear all the other elements, but leaves the elastic fibres unattacked. These sink to the bottom of the test-tube, and may be withdrawn by a pipette (precautions, see Urinary Deposits) for examination under the microscope. They appear as wavy, highly refractile fibres, of uniform thickness, with square-cut ends, and are typically arranged as if surrounding an air cell.

In cases of primary new-growth of the bronchus, recent improvements in technique, introduced by Dudgeon, have rendered easier the detection of carcinoma cells in the sputum and in pleural effusions.

§ 112. **Bronchoscopy** is playing an increasingly important part in the diagnosis and treatment of chest diseases: the bronchoscope is now recognised as an indispensable instrument for the complete investigation of pulmonary disorders. Thus apart from those cases in which the presence of a foreign body is known or suspected, it may give vital information in an obscure case of hæmoptysis. In bronchial neoplasms the diagnosis can often be confirmed, and a biopsy undertaken. When no intra-bronchial mass is visible, broncho-stenosis due to extrinsic pressure from a neoplasm may be recognised ; and a widened angle of bifurcation of the trachea due to a large lymph node infiltrated by secondary growth may decide between exploratory thoracotomy and palliative X-radiation. As a means of treatment, it may be possible via the bronchoscope to extract inspissated mucus plugging a bronchus in a patient with post-operative pulmonary collapse; and even to remove an innocent intrabronchial neoplasm (*e.g.*, adenoma).

PART C. DISEASES OF THE LUNGS AND PLEURÆ: THEIR DIAGNOSIS, PROGNOSIS, AND TREATMENT

§ 113. **Classification.**—For practical purposes, diseases of the lungs and pleuræ may be divided into Acute and Chronic, and each of these may be subdivided into those without dulness, those with dulness, and those with hyper-resonance.

	Acute.	Chronic.
WITHOUT DULNESS.	I. Acute Bronchitis. § 115. II. Dry Pleurisy. § 116. III. Acute Miliary Tuberculosis (Pulmonary form). § 117. IV. Whooping-cough. § 497. V. Acute Pulmonary Œdema. § 118.	I. Chronic Bronchitis (and Plastic Bronchitis). § 129.
WITH DULNESS.	I. Pleurisy with effusion. § 119 (and Empyema). § 120. II. Lobar Pneumonia. § 121. III. Broncho-Pneumonia. § 123. IV. Acute Pneumonic Phthisis. § 124. V. Acute post-operative massive collapse. § 125.	Common. I. Chronic Tuberculosis[1] (and Fibroid Phthisis). §§ 131, 133. II. Hydrothorax. § 134. III. Pulmonary Congestion (Hypostasis). § 135. IV. Fibrosis and Bronchiectasis. §§ 136, 143. V. Thickened Pleura. § 137. VI. Malignant disease of the Bronchus. §§ 81 and 138. VII. Secondary malignant disease of the Lung. § 138. VIII. Collapse of the Lung. § 139. Rare. IX. Hydatid cyst. § 140. X. Syphilitic disease. § 141. XI. Sarcoidosis. § 141.
WITH HYPER-RESONANCE.	I. Pneumothorax. § 126.	I. Emphysema. § 142.

One acute disease tends to be **Paroxysmal.**

I. Asthma. § 127.

Diseases suggested by the character of the sputum.

I. Bronchiectasis. § 143.
II. Abscess and Gangrene of the Lung. § 144.
III. Actinomycosis and other diseases due to fungi and parasites. §§ 145, 146.

§ **114.** The **Routine Procedure** here resembles in principle that used in diseases of the heart. First, *What is the patient's leading symptom?* If suffering from lung disease, his cardinal symptom will be one of those mentioned in section A: cough and breathlessness are the most common.

Secondly, follow this up with a few questions to ascertain the *history of his illness,* and especially whether *the disease be acute or chronic.* Other important points are whether the patient has been exposed to a "chill," and whether there is any "lung disease" in the family. Do not use the word "consumption"; it may frighten your patient unnecessarily.

[1] There is no dulness in the early stages of the disease.

[2] Spontaneous pneumothorax is often an acute incident in a chronic disease—tuberculosis.

Thirdly, proceed to the PHYSICAL EXAMINATION OF THE LUNGS. The routine method is as follows:

1. Ascertain whether there is any increased rate or other modification in the breathing, or any alteration in the shape of the chest (by *inspection*, and, if necessary, by measurement). Note whether any part of the chest shows decreased movement. Observe also the alæ nasi, the colour of the face and lips, and look for clubbing of the fingers.
2. Find the position of the apex beat.
3. Test the vocal fremitus by *palpation*.
4. Ascertain if there be any dulness or hyper-resonance (by *percussion*).
5. Listen to the breath and voice sounds, and then to any adventitious sounds which may be present.
6. The sputum should be inspected, and, if necessary, examined by the pathologist in further detail.
7. X-ray examination must be insisted on if necessary; in the diagnosis of many chest diseases the physician is more and more dependent on radiological evidence.

The chest should always be stripped, and it is more convenient to examine the patient in a standing or sitting posture, if he be not too ill.

If the illness developed gradually, and is of some standing, and unattended by obvious constitutional disturbance, then turn to **Chronic Pulmonary Disorders** (§ 128).

If the illness came on recently and suddenly, accompanied by fever, quickened respiration, coated tongue, and with marked malaise, then the case is one of the **Acute Pulmonary Diseases**, below.

There is one disease of the lungs, ASTHMA, which comes on in sudden acute attacks from time to time; it is **chronic**, with **acute exacerbations**.

Although it is convenient for the sake of classification to include asthma among the pulmonary diseases, since it manifests itself by grave respiratory distress and is often associated with bronchitis, it must be remembered that true spasmodic asthma is not properly speaking a disease of the lungs, but belongs to the group of so-called allergic diseases of which the characteristic respiratory syndrome is but one manifestation. See also under § 127 (Asthma) and § 521 (Allergy).

ACUTE DISEASES.

We now proceed to percuss the chest.

The acute diseases without alteration in the percussion note, *i.e.*, **without dulness**—excluding WHOOPING-COUGH, which is an infective disorder and has no physical signs in the lungs peculiar to it, and ASTHMA,[1] which is of a paroxysmal character—are: ACUTE BRONCHITIS; DRY PLEURISY; ACUTE MILIARY TUBERCULOSIS[2]; and ACUTE PULMONARY ŒDEMA.

I. *The patient complains of a* **cough,** *with frothy expectoration, and his*

[1] Bronchitis is commonly associated with asthma.

[2] In the early phase of this malady there is no alteration of the percussion note, but as the disease progresses dulness appears.

temperature is slightly elevated; there is **no alteration** *in the percussion note but on auscultating the chest, loud* RHONCHI *are heard. The disease is* ACUTE BRONCHITIS.

§ **115. Acute Bronchitis,** or inflammation of the bronchial tubes, is certainly the most common acute disease of the lungs in this climate.

Symptoms.—The disease commences gradually in the course of one or two days, with a feeling of tightness of the chest, of soreness behind the sternum, shortness of breath, frequent cough, and slight rise of temperature, 100° to 101° F. The inflammatory process lasts from ten days to three weeks, and gradually subsides. The sputum is viscid and scanty during the first few days, and then becomes thinner, muco-purulent, and more easily coughed up.

TABLE VI.—DIAGNOSIS OF COMMON ACUTE DISEASES OF THE LUNGS AND PLEURÆ.

	Percussion Note.	Auscultation.
Acute Bronchitis	Normal	R.M. and V.R. normal; Loud râles and rhonchi.
Dry Pleurisy	Normal	V.R. normal. R.M. normal, or may be diminished in intensity owing to restricted inspiratory movements due to pain; Pleural friction.
Acute Pulmonary Tuberculosis	Normal, or scattered areas of impaired note	R.M. diminished; crepitations later.
Pleurisy with effusion	Dull	R.M., V.R. and V.F. diminished; Pleural friction at early and late stage.
Lobar Pneumonia	Dull	V.R. and V.F. increased: Bronchial breathing.
Broncho-Pneumonia	Scattered areas of impaired note	Fine crepitations and scattered areas of bronchial breathing.

Physical Signs.—The percussion note is unaltered unless, as so frequently happens, emphysema be present also, in which case the chest is unduly resonant. On auscultation the vesicular murmur is obscured over the whole chest on both sides by loud rhonchi and moist râles (see Fig. 42) which are variable and altered by coughing. On palpation rhonchial fremitus can frequently be felt.

Etiology.—Bronchitis, which is of course microbic in origin, is generally attributed to: (i.) A chill; that is to say, sudden exposure to cold. (ii.) Sometimes, however, it is caused by spreading from laryngitis. (iii.) It frequently accompanies many of the specific fevers, especially measles, whooping-cough, and typhoid. It is so frequently present with the first and last as to constitute an aid to the diagnosis of those diseases. (iv.) Certain occupations which expose people to irritating vapours and small particles of dust predispose to acute bronchitis. Thus the cotton-mill hands and chemical workers frequently suffer from bronchitis. It is also common amongst cabmen, mariners and others who are exposed to all weathers. (v.) It is a common accompaniment of many other pulmonary diseases, though it may be a subordinate feature. (vi.) It is most frequent in childhood and old age. (vii.) A rare form of bronchitis, due to a fluke, is met with in the East (pp. 219 and 374, Table XIX).

The *Diagnosis* is not difficult in most cases, but *acute miliary tuberculosis* is at first apt to be regarded as acute bronchitis. The diagnosis of tuberculosis is aided by the greater elevation of the pyrexia in the former, and by the subsequent course of the disease. The "*capillary bronchitis*" of children is really a *broncho-pneumonia* (*q.v.*); the constitutional symptoms and dyspnœa are much more marked, there may or may not be some dulness, and the differentiation from simple acute bronchitis is not always easy.

The *Prognosis* is favourable in adolescence and adult life, and the disease usually clears up in one to three or four weeks, though it has a special liability to return, and ultimately to become chronic. It is dangerous in infancy and old age, where the resisting powers are feeble. It is one of the commonest causes of death in old age. If an attack of acute bronchitis does not begin to clear up in two or three weeks, pulmonary tuberculosis should be suspected, especially if the patient be young.

Treatment.—The indications are: (i.) During the first stage, to promote secretion; (ii.) during convalescence, to improve the general condition so as to enable the patient to throw off his liability to bronchitic attacks. The patient must be kept in a warm room at 65° F., without draughts but with adequate ventilation. At the onset give an aperient and a diaphoretic mixture, with perhaps a few grains of Dover's powder to soothe the pain. To promote the flow of secretion, warm alkaline drinks and expectorants such as ipecacuanha and antimony, together with liq. ammon. acet., are especially useful. A good mixture consists of: Sod. bicarb. gr. 10, Sod. chloride gr. 3, Spt. chlorof. ♏ 5, Aq. anisi ad fl. oz. 1. The dose should be followed by a *very hot* drink. When the secretion is free it is sometimes helpful to stop the antimony and administer expectorants, such as ammonium carbonate, syrup of tolu, senega, and squils (Formula 57). If the sputum is very tenacious, add potassium iodide to the expectorant mixtures. The patient must be confined to bed, and will derive great benefit from the inhalation of steam. In childhood this is best done by a bed canopy and a steam kettle beside it; in adults a steam-kettle on the fire will suffice. Linseed-meal poultices, cataplasma kaolini, or a turpentine stupe to the chest relieve the distressing tightness of the chest (see also Formulæ 30 and 68). The importance of chemotherapy and of oxygen therapy must not be forgotten (cf. § 121). During the stage of recovery tonics and cod-liver oil are called for.

II. *The patient complains of sharp* PAIN *in the chest on inspiration; he has a short dry cough, and his temperature is moderately elevated; on auscultation,* FRICTION *sounds are heard. The disease is* DRY PLEURISY.

§ **116. Dry Pleurisy** is inflammation of the pleura without effusion. In this disease there is a fibrinous exudation on the visceral and parietal layers of the pleura, and a tendency to the formation of adhesions, and to the effusion of fluid.

Symptoms.—The disease in some cases comes on quite suddenly with a stabbing pain in the chest, accompanied by a short dry cough. The

constitutional disturbance is seldom very great, and the patient may not necessarily take to his bed. The temperature may rise to 100° or 101° F., rarely higher. The most obvious symptom in this disease is pain in the chest, usually affecting one side, and characterised by being greatly increased on deep inspiration and by coughing. The pain is caused by the contact of the inflamed pleural surfaces, and is usually, though not necessarily, located over the diseased part. For the distribution of pain in diaphragmatic pleurisy, see § 103.

Physical Signs.—On inspection one side of the chest is seen to be limited in movement. Percussion reveals nothing abnormal. On auscultation, the respiratory murmur may be found to be normal, or it may be lessened, as the patient endeavours to restrain the movements of the chest on account of the pain so caused. At a very early stage a pleural rub is heard over one side, often most marked near the angle of the scapula (compare § 109). Sometimes the inflammation undergoes resolution, sometimes it goes on to effusion. As effusion takes place, the pain and pleural friction disappear.

Etiology.—(i.) It may occur as a complication of some acute infective disease, such as measles, scarlatina or influenza. (ii.) Inflammation may extend from disease of the underlying lung, such as pneumonia, tuberculosis, cancer, and infarction, or from adjacent organs, such as the liver or spleen. (iii.) Undoubtedly a large number of cases of pleurisy are tuberculous in origin, especially if recurrent; this fact should always be remembered. Acute pleurisy, with or without effusion, occurring in a young adult, should be regarded as tuberculous in origin, unless it can be definitely proved to be due to some other infective agent.

The *Diagnosis* from *fibrositis* (pleurodynia) may be difficult. Local injection of the muscles with 2 per cent. procaine solution will decide the point. In *intercostal neuralgia* there are tender points along the course of the nerve, and the pain is not aggravated by deep inspiration. Pleural friction is distinguished from the rhonchi heard in *bronchitis* by there being in nearly every case of pleurisy a distinct interval between the inspiratory and the expiratory rub. A radiogram must be taken to see if there is any other evidence of tuberculosis or other intra-pulmonary disease.

Prognosis.—It is not in itself a serious malady, and readily yields to treatment; but sometimes effusion occurs (Pleural Effusion, § 119). When this effusion becomes purulent (§ 120) the prognosis is graver. A frequent result of no great importance is thickening and adhesions of the pleura.

Treatment.—Considerable relief is derived by simply strapping the affected side of the chest, so as to limit the costal movements of respiration. For pain, the greatest relief is undoubtedly given by a linseed-meal or a kaolin poultice. Aspirin is of great service and should be used in preference to opium, though the latter may be necessary. A good combination, in cases with severe pain, is aspirin gr. 10 and Dover's powder gr. 10. Diuretics and diaphoretics are useful. In more chronic cases liniments may be helpful; infra-red radiation may be invaluable. If the con-

dition does not resolve in a few weeks, we must suspect some other cause for the mischief, such as those mentioned under pleurisy with effusion. The treatment of tuberculosis is discussed later (§ 131).

III. *The patient exhibits the signs of subacute bronchitis; but he has* SEVERE MALAISE *and a high* TEMPERATURE. *The disease is* ACUTE MILIARY TUBERCULOSIS.

§ 117. **Acute Miliary Tuberculosis** (acute phthisis, galloping consumption) is often part of a tuberculous process infecting the whole body, and is therefore sometimes described as the pulmonary form of acute general tuberculosis (see Fig. 121, a chart showing the typical course of the temperature). The type of fever exhibited varies in different cases; in some there is considerable remission in the mornings, in others the chart shows continuous pyrexia with little remission.

Symptoms.—The malady is of most insidious onset: there may be progressive weakness and emaciation. Some weeks before physical signs have appeared the thermometer may show the typical intermittent pyrexia so characteristic of tubercle—an evening elevation of 101° to 103° F., and a morning normal temperature. As the disease progresses the remissions are likely to be less, the fever being more of the continuous type. In some cases the inverse type is present, when the temperature is higher in the morning than in the evening. Night-sweats and cough are present, with muco-purulent expectoration. Dyspnœa, and sometimes cyanosis, develop out of proportion to the physical signs; the cyanosis may be extreme, and of itself is a very characteristic feature. Great weakness ensues, and in the third or fourth week the patient may develop the symptoms of the typhoid state or of meningitis.

The *Physical Signs* referable to the lungs are indefinite, or resemble at first those of bronchitis. At first there is no alteration in the percussion note, but by and by careful percussion discovers scattered patches of impaired resonance. Auscultation at first may give little help, but in a week or so it reveals rhonchi and fine râles over certain areas, which do not shift from place to place, as in bronchitis. Later on the râles are coarse and bubbling, and areas of tubular breathing may be found.

The *Diagnosis* in the first stage from bronchitis and broncho-pneumonia is extremely difficult. We have to rely upon the disproportionate emaciation and cyanosis, the character of the temperature, and the patchy distribution of the physical signs in tuberculosis. In other cases the malady is almost indistinguishable from typhoid fever except for the marked predominance of the pulmonary signs and the absence of the roseola; the Widal test is negative. In all stages the detection of tubercle bacilli in the sputum is one certain means of diagnosis, though their absence does not exclude acute pulmonary tuberculosis. X-ray examination of the lungs may reveal the characteristic "*snowstorm*" appearance of miliary tuberculosis.

Etiology.—The disease may occur at any age, but is commonest in infants, in young adults, and in those with a tuberculous family history. Acute general tuberculosis may originate from a primary focus, such as a tuberculous joint which had been considered cured. The disease may follow measles or whooping-cough in children.

Prognosis.—The disease is almost uniformly fatal in a few months, but occasionally the course is prolonged, even up to two years, and recovery has been recorded (cf. § 132*a*, chronic miliary tuberculosis). *Treatment* is almost entirely symptomatic.

IV. *The patient, a child, has* PAROXYSMS *of coughing which terminate in a* WHOOP, *and frequently in* VOMITING; *there is some fever, but the only signs in the lungs are those of a little bronchial catarrh. The disease is* WHOOPING-COUGH.

Whooping-cough (Pertussis) is an acute infectious disease, and it is described among the microbic disorders (§ 497).

V. *The patient is suddenly seized with acute dyspnœa and copious frothy sputum flows from the mouth and nose. The disease is* ACUTE PULMONARY ŒDEMA.

§ 118. **Acute Pulmonary Œdema.** *Symptoms.* The sudden onset of acute dyspnœa, with copious, often blood-stained (rose-coloured) and albuminous sputum, are characteristic. The face is pale and cyanosed, the expression is one of intense anxiety; there may be a cold sweat. The pulse is feeble, and there may be pain or a feeling of oppression in the chest. The disease usually depends on failure of the left ventricle, allowing fluid to accumulate in the lungs. It may arise in the course of heart disease, especially aortic disease, arterio-sclerosis, pregnancy, epilepsy, giant urticaria, acute infections, or renal disease. It is an occasional complication of lung operations, *e.g.*, thoracoplasty or lobectomy. The *physical signs* consist of râles and crepitations which are heard all over the chest.

Treatment.—Sometimes the disease is so rapidly fatal that no treatment is of avail. The best emergency treatment is immediate blood-letting to 20 ounces. Atropine has an almost specific action; $\frac{1}{100}$ gr. should be given hypodermically at the earliest possible opportunity, and in severe cases, with copious frothy sputum, should be repeated frequently: even $\frac{1}{50}$ gr. of the drug may be given every two hours until the full pharmacological effects are obtained. Oxygen, nikethamide B.P. (coramine) or other stimulants may be necessary. Mersalyl (2 c.c.) has been used with beneficial effects. The recurrence of attacks cannot be prevented except in those cases when the patient is able to foretell their coming, when atropine given in time may ward off or mitigate the attack. The only prophylactic treatment is that directed to the underlying disease. The disease may never recur, but some patients may relapse at variable intervals for years.

We now turn to the **Acute Diseases with Dulness on Percussion.**—I. PLEURISY WITH EFFUSION (Serous or Purulent); II. LOBAR PNEUMONIA; III. BRONCHO-PNEUMONIA; IV. ACUTE PNEUMONIC PHTHISIS; and V. ACUTE POST-OPERATIVE MASSIVE COLLAPSE.

I. *The patient has a* DRY COUGH, *with moderate fever and other constitutional symptoms. The lower part of the chest is* DULL *on one side, and over this area the* VOCAL FREMITUS *and* RESONANCE *are* DIMINISHED *or* ABSENT. *The heart is displaced towards the healthy side. The disease is* PLEURISY WITH EFFUSION.

§ 119. **Acute Pleurisy with Effusion.**—When describing acute Dry Pleurisy (§ 116) it was pointed out that the disease may undergo resolution or result in adhesions. It may also go on to effusion.

Symptoms.—There is usually a history of a more or less acute onset with pain (§ 116), but as the disease progresses, and the surfaces of the pleura are separated by fluid, pain becomes less and less marked. Occasionally the onset is insidious, and a considerable amount of fluid accumulates in the pleural cavity without any history of initial pain. The patient suffers from general malaise, and finds it difficult to lie on the sound side, because the action of the healthy lung is thereby impeded. A degree of breathlessness may be present, but even with a large amount of fluid this is not invariably a prominent feature.

Physical Signs (see Fig. 49).—(i.) Inspection may show diminished movement, fulness or bulging of the chest wall. (ii.) On palpation, the vocal fremitus is found to be diminished or absent over the fluid, and there may be bulging of the intercostal spaces. (iii.) Percussion reveals

absolute dulness over the areas of the fluid. Above the level of the fluid, if the lung be otherwise healthy, there is a hyper-resonant note (Skodaic resonance). (iv.) On auscultation over the fluid, the breath sounds are absent; the vocal resonance is greatly impaired or lost. Bronchial breathing may occur.[1] At the upper margin of the fluid posteriorly—perhaps just about the angle of the scapula—only certain overtones of the voice are transmitted, and they produce, therefore, a sound like the bleating of a goat (ægophony). (v.) When the effusion is large it causes displacement of the heart, which may be very considerable. The amount of fluid present may be estimated by the degree of cardiac displacement, and of respiratory distress.

The *diagnosis* of pleurisy in its earlier stages is referred to under Dry Pleurisy. The differentiation of the physical signs of fluid in the chest,

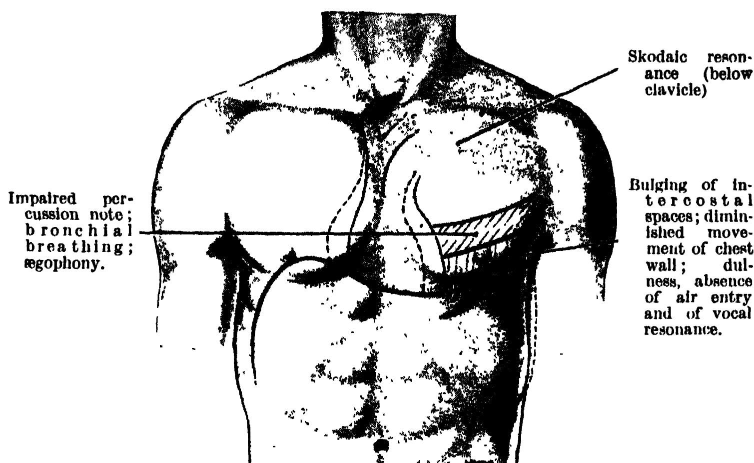


FIG. 49.—Physical signs of Pleurisy with effusion.

as compared with those of consolidation of the lung, is so important that it is given in Table V (§ 109). In case of doubt, exploratory puncture is essential to determine both the presence and the character of fluid in the pleural cavity.

Etiology.—Tuberculosis is by far the commonest cause of a sudden pleural effusion in a young adult. The fluid in such cases is commonly clear and straw-coloured: the cells are predominantly lymphocytes, though occasionally some polymorphs may be present. Young patients with a serous lymphocytic effusion should be regarded as tuberculous unless some other cause can be proved; culture on special media and/or guinea-pig inoculation of the deposit often reveals tubercle bacilli (p. 1121).

[1] High-pitched bronchial breathing is commonly heard over the compressed lung above the level of the fluid. Occasionally, however, breath sounds are still audible over a much larger area, in spite of the presence of a considerable effusion. This is especially the case in children (*e.g.*, where empyema is present).

The occurrence of a pleural effusion in a middle-aged or elderly patient should raise a suspicion of malignant disease: in such cases further investigation by bronchoscopy, etc., is indicated. In a few instances of acute tuberculous effusions the fluid is blood-stained; in the majority of cases a sanguineous pleural effusion is pathognomonic of new growth. (For other causes of serous pleural effusion, see § 134 Hydrothorax.)

Prognosis.—This depends on the cause and on the treatment available. Most tuberculous effusions will absorb if left alone. In the early stages there is often an irregular pyrexia; this usually settles in the course of a few weeks; the fluid shows signs of diminution, the vocal fremitus and resonance return and the breath sounds become more audible. After absorption, the adjacent pleural surfaces usually become adherent. If the effusion lasts a long time and re-collects after repeated aspiration, a tuberculous empyema should be suspected.

Obliterative Pleurisy.—Occasionally, after gradual absorption of a pleural effusion, the formation of multiple adhesions results in a partial or complete obliteration of the pleural cavity. This is a common sequel of effusion occurring during a course of artificial pneumothorax therapy, and arrests this treatment. In such cases, provided no uncollapsed cavities remain in the lung, an obliterative pleurisy may sometimes be an entirely beneficial end-result. The physical signs of this condition are those of thickened pleura. (See § 137.)

Treatment.—In the first place patients must be kept strictly in bed: they usually prefer to sit up, supported by pillows. No treatment other than a diagnostic puncture of the chest is necessary, but if after a few weeks' trial these measures fail, the question of aspiration should be considered. It must be emphasised that the mere presence of fluid in the pleural cavity is not an indication for its immediate removal. There is seldom any urgency, unless it is found to be purulent (empyema), or unless the amount present causes grave discomfort to the patient, who may be suffering from cardiac embarrassment. When the fluid has persisted for many weeks without showing any signs of absorption, it may then be advisable to remove part of it (*e.g.*, 10 to 20 ounces), after which the remainder often becomes absorbed in a reasonably short time. As a general rule it is inadvisable to delay paracentesis and aspiration under the following conditions: (i.) a large effusion (*e.g.*, with dulness extending upward as far as the third rib); (ii.) cardiac embarrassment, as evidenced by cyanosis, palpitation, and a rapid pulse; (iii.) respiratory embarrassment, shown by urgent dyspnœa and paroxysmal attacks of coughing; (iv.) effusion in the other pleura, or œdema of the other lung; (v.) if the fluid is not sterile.

Paracentesis thoracis.—When possible the patient should be sat up, well supported with pillows; the hand on the affected side is placed on the opposite shoulder. The usual site of puncture is the 8th space in the post-scapular line—or at a site where localised dulness is maximal. An intradermal injection of 2 per cent. procain is made at the intended site of puncture with a hypodermic syringe and needle,

The needle is withdrawn, then thrust through this now anæsthetic skin area, and on slowly through the tissues of the intercostal space until the pleura is reached; the piston of the syringe is pushed down as the needle advances. Thus the track of the needle is anæsthetised with a fine stream of procain. The exploring needle, attached to 20 c.c. glass record syringe, is pushed vertically through the anæsthetised area, gentle suction being maintained all the time. If there is fluid in the pleural cavity, it will enter the barrel as soon as the needle reaches it. Perforation of the lung is indicated if air or frothy bright red blood is sucked up into the syringe; in such a case the needle is withdrawn, the blood driven out and the process repeated, with the needle inserted in another direction.

Aspiration of a pleural effusion is best performed with a Potain's or Dieulafoy's aspirator. It is better to avoid the large trocar and cannula usually supplied with Potain's aspirator: a smaller one, such as Rivière's initial pneumothorax needle, or even the ordinary exploring needle, is usually large enough and can be fitted by means of an adapter to the tube of the aspirator. The smaller needles are easier to introduce, and the patient is spared much discomfort. Passage of the needle is facilitated by making a small incision in the skin with the point of a fine scalpel. The side tube of the needle is connected with a large glass bottle, the air in which can be exhausted by means of a pump. After withdrawing the plunger, and turning the tap to connect the pleural cavity with the interior of the bottle, the fluid from the pleural cavity is sucked into it by the partial vacuum. The aspiration can be continued until the patient is conscious of slight discomfort. If cough or pain occur, it is wiser to cease; it is unwise to remove more than 20 fl. oz. at a time unless it is accompanied by air-replacement, which obviates the cough and discomfort due to the sudden re-expansion of the lung. At the conclusion, after withdrawing the needle or cannula, cover the puncture wound with gauze soaked in collodion.

Siphonage can be used for the removal of the fluid, but is seldom so efficacious. When the condition of the underlying lung is such that collapse therapy is necessary, the fluid may be replaced by air: but this should never be the routine treatment of a pleural effusion.

I*a*. *The physical signs are those of pleurisy with* EFFUSION, *but it does not clear up in due course, and the patient continues to have* SWEATINGS, SHIVERINGS, *and an* INTERMITTENT HIGH TEMPERATURE. *The disease is probably* EMPYEMA.

§ **120. Empyema** is a collection of purulent or sero-purulent fluid within the pleural cavity. It often follows a serous effusion, but it may be purulent from the beginning. The pneumococcus is the organism most commonly found.

The *Symptoms* and *Physical Signs* are similar to those of serous effusion (*q.v.*, *supra*), with certain others in addition—viz.: (1) It may be found that the fluid *does not clear up* as a serous effusion should do, and thus the presence of pus may be suspected. (2) Whenever pus forms, either in the pleura or elsewhere, it is marked by the occurrence of sweatings, shiverings, and an intermittent pyrexia. (3) Œdema of the integument, the pointing of an abscess in an intercostal space, over the clavicle, or even in the groin, or copious expectoration of pus, may occur if an empyema is overlooked. The modern knowledge of the radiograph and the wider use of the exploring needle have made these accidents less frequent. (4) Clubbing of the fingers, especially in children, is a valuable sign which

may come on very rapidly in empyema. (5) The history commonly reveals one of the following *causes* of empyema:

(i.) Lobar pneumonia is by far the commonest cause, especially in children; (ii.) septic conditions of the pericardium, mediastinum, or respiratory tract; (iii.) tuberculosis in any form in the thorax; (iv.) the acute specific fevers; (v.) abscess of the lung, abscess of the liver or spine bursting towards the pleura, or peri-hepatic abscess resulting from appendicitis, leaking gastric or duodenal ulcer; (vi.) pyæmia; (vii.) careless paracentesis, or any wound from without, permitting the introduction of organisms.

(6) In doubtful cases a leucocyte count should always be made, since in the absence of acute lobar pneumonia more than 20,000 leucocytes per cu.mm. would strongly favour the diagnosis of empyema. (7) Diagnostic puncture is essential when pus is suspected, though there are two fallacies in this method: first, in rare cases the fluid may be too thick to come through the needle; or, again, the pus may be encysted between the lobes of the lung. In any case, a pathological examination of the material at the point of the needle will assist the diagnosis.

Prognosis.—Empyema is always serious, and may run a somewhat prolonged course of some months. Cases of pure pneumococcal empyema are much more favourable than those due to streptococci or staphylococci, either alone or with the tubercle bacillus. Operation, adequate drainage, and strict aseptic precautions, both at the operation and at the subsequent dressings, are the points in treatment which most favourably influence prognosis. If left to itself, the results vary: sometimes there is compression and destruction of the lung; sometimes, as above mentioned, the pus opens into the lung, burrows in various directions, or opens through the chest wall; or the condition may lead to pyæmia.

Treatment.—A pneumococcal empyema is usually drained as soon as the diagnosis is made. In streptococcal empyema drainage by rib-resection is not performed until the fluid is frankly purulent, because operation in the sero-purulent stage is attended by very high mortality. In the early stages, the introduction of penicillin daily into the pleural cavity (120,000 units in 10 c.c. of physiological saline) after withdrawal of the fluid is very valuable. In tuberculous cases open operation is avoided whenever possible.

The after-treatment of empyema is designed to promote expansion of the lung and is most important. The establishment of air-tight drainage at operation is very helpful. The tube leading from the wound is connected with an under-water drain. In the convalescent stage breathing exercises are of the utmost value. To obtain the best results these should be supervised by an expert, who will ensure the maximum degree of movement of the affected side, movements of the contralateral side being restricted by the masseuse.

II. *The patient has been* TAKEN ILL SUDDENLY; *the temperature is high, the dyspnœa considerable, and cyanosis is present; the expectoration*

soon becomes rusty; there are SIGNS OF CONSOLIDATION *at the base of one lung. The disease is* ACUTE LOBAR PNEUMONIA.

§ 121. **Pneumonia**—*i.e.*, inflammation of the pulmonary tissue proper, or parenchymatous inflammation—occurs in two forms. The *first* and more acute is, from its area of distribution, termed "Lobar Pneumonia." The *second* is termed "Broncho-Pneumonia," because it affects the bronchi, and spreads to the lungs; see § 123.

Acute Lobar Pneumonia commences suddenly, with well-marked constitutional symptoms, such as headache, backache, rigor, and, in children, vomiting or convulsions. The temperature during the rigor rises to 103° or 104° F., and it remains at this point for about a week (Fig. 50). The aspect of a pneumonia patient is very characteristic (§ 7). The face is flushed and cyanosed, and herpes often appears near the mouth. There is pain in the affected side due to involvement of the pleura, short cough, shallow rapid breathing, and tenacious mucoid sputum which becomes rust-coloured on the third or fourth day. The pulse-respiration ratio is 3 to 1, or 2½ to 1, instead of the normal 4 to 1. The urine is scanty and high-coloured. The patient shows more and more distress, and in a short time there may be delirium, with signs pointing to failure of the cardio-vascular system. About the *seventh* or *eighth* day the fever, as also the pulse and respiration rate, in favourable cases, terminates by crisis, falling to normal in the course of a few hours. This is accompanied by marked general improvement; the pulse-respiration ratio returns to normal, and a critical sweating or diarrhœa may occur. Pseudo-crises occasionally occur, but these are distinguished from true crises by the fact that the pulse and respiration do not return to normal. In some cases the temperature falls by lysis. The whole illness lasts about two or three weeks. If it lasts longer, *tuberculosis should be suspected* (§ 131), or more commonly some complication such as empyema (§ 120).

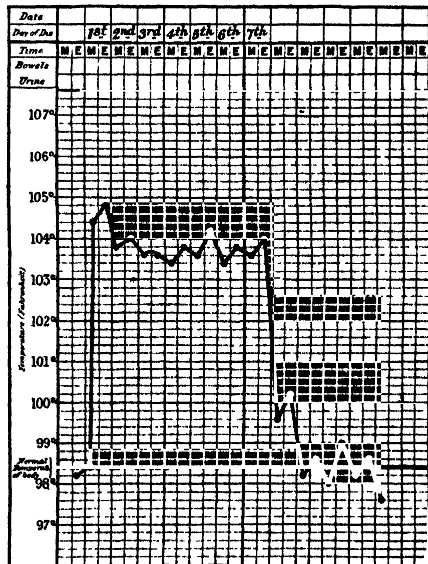


FIG. 50.—ACUTE LOBAR PNEUMONIA, showing typical crisis on the seventh day. George H., aged thirty-five, was taken ill very suddenly with shivering and acute pain in the side. (No chemotherapy was given.)

The *Physical Signs* are limited to one lung or one lobe, usually the right lower lobe. It is only in rare cases that both lungs are affected. At the onset percussion may, for the first day or two, reveal no dulness,

but, as a rule, there is elicited early in the disease slight impairment of the percussion note, which soon becomes dull. On auscultation the breath sounds are weak, and fine rustling crepitations are heard which have been compared to the rustling of hair or tissue-paper against the ears. The weak respiratory murmur is noticeable many hours before the bronchial breathing. The latter, when it does come, may be feeble; the significance, however, is the same. As the inflammatory exudation increases, the lung tissue becomes solid, and over the dull area we get all the *signs of consolidation* (§ 109). When the fever abates, coarse moist râles (redux crepitations) are heard, and the normal percussion resonance and breath sounds gradually return.

Central Pneumonia. Cases of acute pneumonia begin with the typical onset described at the beginning of this section, but physical signs of consolidation do not appear until very late (*e.g.*, a week or ten days). In other cases the fever abates suddenly, after two or three days, with a crisis similar to that of an ordinary case, the physical signs of lobar consolidation being entirely absent. Such phenomena are due to the fact that the actual lesion in the lung is deep-seated, and only spreads to the surface much later, if at all. X-ray examination reveals the condition.

Etiology.—Pneumonia occurs at all ages and in both sexes, but is commonest in adult males. It is a bacteriæmia, the specific cause being a diplococcus, the pneumococcus of Fraenkel. Debilitating influences, such as exposure, are said to predispose to the disease; but it is surprising how often strong, apparently healthy men are attacked, and these not infrequently succumb. A blow on the chest may determine an attack (traumatic pneumonia). Like other local inflammatory diseases, it may arise as a complication of a constitutional malady; the acute specific fevers in particular rendering a person vulnerable to the pneumococcus. The termination of chronic nephritis, etc., by pulmonary complications is usually a question of so-called hypostatic pneumonia. This should not be included under Lobar Pneumonia proper. When pneumonia runs an atypical course we should always bear in mind the possibility of the lung affection being only a complication of a constitutional disease such as typhoid fever.

Diagnosis.—Pneumonia is diagnosed from acute *pleurisy with effusion* (in which the lungs are often affected) by means of the data given in the table of diagnosis between consolidation of the lungs and fluid in the pleura (§ 109). *Broncho-pneumonia* runs a different course, and the signs are scattered over both lungs (see table below). The sudden onset of acute pneumonia resembles that of *scarlet fever, erysipelas,* and *small-pox,* but the absence of rusty sputum and altered pulse-respiration ratio distinguishes them. There is a pneumonic form of *acute pulmonary tuberculosis* which has to be borne in mind (§ 124), also various *aberrant forms of pneumonia* (§ 122) and acute exacerbations in bronchiectasis. Pneumonia may, especially in children, at its onset simulate *abdominal inflammation,* pain being referred to the abdomen, and lung signs being absent (§§ 238,

248). It is in such cases that a diminished respiratory murmur on one side of the chest and an increased respiratory rate are signs of great value.

Prognosis.—The case mortality of all ages combined used to vary between 20 and 40 per cent., but the advent of the sulphonamides and subsequently of penicillin treatment has reduced this to 5 per cent. or less. Instead of lasting on an average 7–8 days, treatment with these drugs causes the temperature to drop to within one degree of normal within 48 hours, with corresponding improvement in the patient's clinical condition. The prognosis is worse when the natural resistance of the patient is low or the invading organism particularly virulent: pneumonia is more serious in elderly persons, in young children, and in alcoholics, diabetics and in others suffering from debilitating conditions. Children from 3 to 10 years nearly always recover, but robust men in the prime of life often succumb, although the prognosis is generally stated to be good in healthy adults. The type III pneumococcus produces a higher mortality than any of the other types. Unfavourable features include the extensive involvement of lung (the outlook being worse when both lungs are involved), marked cyanosis, considerable delirium or a typhoid state, an unduly low temperature, marked tachycardia and particularly absence of the usual leucocytic response. Jaundice, meteorism and auricular fibrillation may occur. Complications of serious import include pneumococcal septicæmia, meningitis or endocarditis, but even these often come under control with adequate and early chemotherapy. Delayed resolution, lasting one to three months, is uncommon; empyema, abscess, and gangrene may supervene in weakly subjects. By far the commonest complication is empyema.

Treatment.—*Chemotherapy* with the sulphonamide drugs, and/or penicillin should be started immediately the diagnosis is made. In pneumococcal infections sulphadiazine, sulphamethazine or sulphamerazine are usually chosen, though in staphylococcal infections sulphathiazole and in streptococcal infections sulphapyridine are regarded by some as more effective (see Tables XXVIII, XXIX and § 515). For an adult, the usual initial dose is 4 G. followed by 1 G. 4-hourly for 48 hours, after which smaller amounts are given until the temperature has been normal for at least 48 hours. The total amount given usually varies between 40 G. and 60 G. (For doses in children see Table XXVIII.) An adequate fluid intake of at least 6 pints in 24 hours is essential: it is advisable also to give large amounts of alkali simultaneously, *e.g.*, sodium bicarbonate 6 G. initially followed by 2·5 G. every 4 hours with the sulphonamide tablets, which should be crushed before administration.

Penicillin may be used instead of, or in addition to, sulphonamide therapy: the usual dose is 20,000–30,000 units in sterile water subcutaneously every 3 hours day and night for periods up to 5 days. The necessary concentration in the blood can be maintained by giving as much as 500,000 units 8-hourly, thus avoiding disturbing the patient's sleep.

General treatment.—The administration of sulphonamides and/or

penicillin cannot be regarded as a substitute for the general measures which must be observed in all cases of pneumonia. The patient's strength must be maintained by rest in bed, good nursing, adequate sleep, and regular visits by the doctor. Fresh air is essential: patients treated near an open window have less dyspnœa and cyanosis, and do better than those treated in a vitiated atmosphere. They should be kept sufficiently warm by blankets and, if necessary, by hot bottles. The bowel should be cleared with an initial dose of calomel. The *diet* must be fluid with 2 to 3 pints of boiled milk in addition to fruit-drinks, lemonade or barley water, with glucose. Raw eggs, broths and jellies can be added later. If acute dilatation of the stomach or intestinal paresis sets in, with vomiting and abdominal distension, pituitary extract, prostigmin and lavage will often avert a fatal issue. For meteorism give turpentine fl. oz. ½ to a large enema. *Sleep* is of such paramount importance that no patient should be allowed to spend a restless night. The cause of the restlessness should be sought for and treated. For extreme restlessness with delirium, give chloral and potassium bromide, in doses of 30 to 40 grs. of each. When fever over 103° is the cause of sleeplessness, it may be reduced by tepid sponging, a measure which, next to the relief of pain and engorgement of the right heart, is the most satisfactory means of procuring sleep. Hypnotics such as paraldehyde may be given, or morphia when pain is severe. An initial dose of ¼ grain of morphia at the commencement of the disease often benefits, by ensuring sleep: in the later stages it is usually wiser to avoid morphia—it should never be given if the patient is cyanosed. Frequently *pain* is the disturbing factor. Apart from the use of morphia, this may be relieved by the local application of ice, fomentations, poultices, a blister, or leeches. Another cause of sleeplessness is *engorgement of the right heart*: in every case of pneumonia careful watch should be kept for this. If, in the early stage of the disease, the patient is blue and restless, the cardiac dulness increased considerably to the right, the liver enlarged, and the veins of the neck full, we should immediately relieve the right heart, by venesection (5 to 10 ounces) or by applying six leeches to the skin over the liver. Opium (10 gr. Dover's powder) may be used in the early stages of the disease, and is often of the greatest value, but if the right heart shows signs of engorgement, it is better to give morphia with atropine. Small doses of potassium iodide loosen tenacious sputum.

Stimulants.—The value of oxygen inhalations is well proved, especially in cases with cyanosis; they should be begun early. The most convenient method of administration is by a nasal catheter or by one of the recognised forms of mask (*e.g.*, the B.L.B. mask), at the rate of five to seven litres per minute, which is as fast a flow as can be comfortably borne. The value of the oxygen tent in severe cases, and especially in children, is beyond doubt. The modern apparatus is easy to manipulate. In order to obtain the necessary concentration (30 to 60 per cent.), oxygen is introduced at a rate of five to ten litres per minute, the patient being continually in the tent, except when nursing attention demands a temporary interruption. The

administration of oxygen by other (traditional) methods (subcutaneous injection and a wide-mouthed glass funnel attached to the tube from a gas cylinder and held in front of the patient's face) is inadequate and of no practical use. Dry oxygen is an irritant. It should therefore be moistened by being passed through warm water before reaching the patient. Nikethamide B.P. (coramine) and other vaso-motor stimulants can be used as required. Concerning alcohol, there is much difference of opinion. It is particularly indicated in alcoholic patients, for whom it should be used freely (4 to 12 ounces whisky in twenty-four hours), and especially in conditions of collapse near the crisis, when it may tide the patient over so that he is out of danger before the subsequent depressing effect of the drug becomes manifest. Pneumococcal pneumonia is an infectious disease. Preventive treatment comprises care of the mouth, teeth, and nasopharynx, and the use of pneumococcal and influenzal vaccines.

Treatment by *specific serum* has largely been replaced by chemotherapy. If toxæmia is severe before chemotherapy can be started, Felton's serum (which is polyvalent for Types I and II pneumococci) may be used (and see § 521).

§ 122. **Aberrant Acute Pneumonias.**—We have seen that in pleurisy, acute lobar pneumonia, and in other inflammatory diseases of the lungs, the course of the malady is fairly definite, and the physical signs in the lungs are characteristic. But it is important to remember that these same conditions may occur secondary to, or as part of, some general disorder. In these circumstances some of the symptoms or physical signs may be wanting or irregular, and it may not be possible to arrive at a diagnosis, except by passing in review the whole history of the case, and by making a thorough and systematic examination of all the other organs. Instances of this eccentric group of pneumonias are met with in acute glanders, tuberculosis, plague, anthrax, syphilis of the lung, distoma infection, actinomycosis, and psittacosis.

The practical outcome of this is that when a case of pneumonia, or other apparently local inflammatory condition, is *atypical* in its physical signs or its clinical history, we probably have to do with a manifestation of one of the conditions just mentioned, or some general disease, such as typhoid fever, influenza, scarlatina, pyæmia, or other general infective disorder.

The term *pneumonitis*, introduced originally in the United States of America, was intended to cover a wide range of conditions characterised by the presence of an area or areas of localised consolidation in the lung. Such conditions, although inflammatory in origin, are not examples of pneumonia in the ordinary sense of the term, and do not give any typical clinical picture with a definite clinical course. The term is a useful one to denote the nature of the underlying pathological process, but it should not be supposed that pneumonitis indicates a clearly defined clinical entity.

The so-called *primary virus pneumonia*, frequently referred to by writers in the U.S.A. as primary atypical pneumonia, comes into this group. Usually mild in type, with few physical signs in the chest, there is no leucocytosis or response to chemotherapy: in consequence the temperature may be slow in settling to normal.

III. *The illness has come on* LESS SUDDENLY *than in lobar pneumonia; there is cough, with frothy expectoration; the physical signs of* CONSOLIDATION *are* MORE PATCHY *and accompanied by signs of bronchitis. The disease is probably* BRONCHO-PNEUMONIA.

§ 123. **Acute Broncho-Pneumonia** is also an acute parenchymatous inflammation of the lungs, but it runs a very different course to that of acute lobar pneumonia. The inflammatory process occurs in small patches,

scattered unequally throughout both lungs, and it is accompanied by bronchitis: hence the name.

The *Constitutional Symptoms* usually come on more gradually in this disease. The temperature is remittent, about 100° F. in the mornings and 101° to 103° F. in the evenings, accompanied by cough, dyspnœa and muco-purulent sputum. The pulse is rapid, but the pulse-respiration ratio is not altered to anything like the extent of that in lobar pneumonia, and the face is generally pale instead of flushed. Without chemotherapy fever is likely to persist for 2 or 3 weeks or more.

Physical Signs.—When the patches of consolidation are small, there may be no dulness on percussion, but only tubular breathing; when they are of moderate size, signs of consolidation (§ 109) can be made out. The chief auscultatory signs in children consist of *intensely loud*, "consonating" râles, and rhonchi.

Etiology.—Broncho-pneumonia occurs at all ages, but is *especially frequent in young children.* The cases fall into two groups, primary and secondary. Primary broncho-pneumonia, due to the pneumococcus, arises in much the same way as lobar pneumonia. Secondary forms arise: (i.) Complicating acute infections, such as measles, whooping-cough, diphtheria, small-pox, influenza, typhoid and scarlet fevers; (ii.) complicating chronic debilitating conditions, such as chronic renal disease, chronic cardiac disease, or bed-lying, as from fracture of the femur in old people; (iii.) *aspiration* or *deglutition* (*septic*) *pneumonia*, such as occurs after operations on the tongue, mouth, or nose, in quinsy, cancer of the œsophagus communicating with the air-passages, bronchiectasis, and following hæmoptysis or the passage of food down the trachea, as in post-diphtheritic paralysis and in bulbar palsy. In operations on the throat and nose under general anæsthesia, attention must be paid to the drainage of blood and the removal of all solid particles of tissue. (iv.) A common but more chronic variety is of tuberculous origin.

Diagnosis.—Tuberculous broncho-pneumonia is discussed in another section. The pulmonary signs of *measles*, *whooping-cough*, *bronchitis* and *psittacosis* resemble broncho-pneumonia in its early stages, and it may not be easy to diagnose these several diseases until the rash of the one or the whoop of the other appears. The constitutional symptoms in acute bronchitis are much less severe. The diagnosis from *lobar pneumonia* is given in tabular form on page 185.

The clinical picture of acute pneumonia appears to have altered of late years, and the hard and fast line of demarcation between these types is to-day less easy to recognise. The very severe pneumonia seen in conjunction with influenza, and associated with a virulent streptococcal infection, is a comparatively recent phenomenon, attention having been dramatically focussed upon it by the great epidemic towards the close of the War of 1914–18.

In many cases of acute streptococcal pneumonia the onset is similar in its suddenness to that of the pure pneumococcal (lobar) type, but the subsequent picture often differs considerably from that above described under lobar pneumonia, two characteristic features being the greater intensity of the toxæmia in the very early stages and the tendency to end by lysis rather than by crisis. The toxic state of the patient, mental

TABLE VII.—DIFFERENTIATION BETWEEN A TYPICAL CASE (untreated by chemotherapy) OF

	LOBAR PNEUMONIA	*and* LOBULAR OR BRONCHO-PNEUMONIA.
Onset	Sudden, with rigors ..	Gradual, and preceded by bronchitis.
Course of Temperature	Continuous	Remittent.
Defervescence ..	Crisis usually by seventh day[1]	By lysis in two to four weeks.
Percussion	Dulness in one lung, usually the base.	Scattered patches of dulness in both lungs.
Auscultation ..	(i.) Fine crepitations .. (ii.) Consolidation signs in a day or two.	Fine crepitations and consolidation signs over dull areas, though obscured by rhonchi and bronchitic râles.
Sputum	Rusty	Frothy and muco-purulent.
Respiration ..	Pulse-respiration ratio 3 : 1 or 2½ : 1.	Less marked difference of pulse-respiration ratio.

apathy, pallor, often cyanosis, are early features, sometimes apparent before the occurrence of appreciable physical signs in the chest. Recent work on the pathology of the pneumonias, especially in children, shows that it is difficult to insist upon the exact division into the lobar and bronchial types described in most text-books ; many cases exhibit certain of the features of both, and the traditional distinction may be impossible to maintain. The important point is to differentiate the acute pneumonias according to the nature of the infecting organisms as a guide to accurate chemotherapy.

Prognosis.—Prior to the advent of drugs of the sulphonamide group the case mortality in children under five varied from 30 to 50 per cent. ; the younger the child the more fatal was the disease, and under the age of 6 months 90 per cent. of cases were lethal. The age of the patient, the virulence of the infection, and the degree of response to chemotherapy are leading factors in the prognosis. The longer the disease persists the more likely it is to have a fatal termination. Broncho-pneumonia is nearly always secondary, and a factor of importance in the prognosis is the nature of the antecedent disease. When a child weakened by a *prolonged* fever is attacked, the prognosis is very grave ; nevertheless, children often recover in apparently hopeless cases. Pulmonary fibrosis is a well-recognised sequela of broncho-pneumonia occurring in whooping-cough and measles. The aspiration and deglutition pneumonias are usually fatal.

Treatment resembles that of lobar pneumonia (*q.v.*) Children should be placed in an oxygen tent. Adults may need Dover's powder or linctus heroin if their nights are rendered sleepless. If the symptoms of cough and dyspnœa are distressing, alkaline expectorants with small doses of potassium iodide (gr. 3) will often give relief. For the reduction of excessive fever tepid sponging may be invaluable.

[1] In many epidemics lysis is more common than crisis.

§ 124. IV. **Acute Pneumonic Phthisis** (synonym: Acute Caseous Pneumonia) is not uncommon. The symptoms resemble those of pneumonia, and may start suddenly with a rapid rise of temperature and pain in the side. The temperature may continue high for a week or so. The physical signs also resemble those of pneumonia. It differs from this disease, however, in the presence of tubercle bacilli in the sputum, and the temperature, instead of falling abruptly by crisis about the seventh day, gradually becomes intermittent, and the *course of the disease* becomes indefinitely prolonged for weeks. This is followed by physical signs of breaking down in the lung, purulent expectoration, night sweats, and in some cases death in five to twelve weeks from exhaustion, hæmoptysis, or complications, such as pneumothorax (§ 126).

§ 125. V. **Acute post-operative massive collapse** of the lung is an important condition. Opinion inclines to the view that most of the so-called post-operative pneumonias are due to massive collapse. There are probably several causal factors; obstruction of a bronchus by viscid secretion is believed to be the most important. If a main bronchus is obstructed, constitutional disturbance may be severe, simulating that of pneumonia. The physical signs and radiological appearances are like those of pneumonia, but the displacement of the heart towards the affected side should make the diagnosis clear. *Treatment* consists in giving inhalations of 7 per cent. carbon dioxide and 93 per cent. oxygen several times a day, for 10 to 15 minutes at a time. An alkaline expectorant mixture containing a small dose (*e.g.*, gr. 3) of potassium iodide, thrice daily, helps to increase secretion and to liquefy any viscid mucus present in the bronchi. Removal of the obstructing plug of sputum, mucus, etc., by means of the bronchoscope, is often necessary.

We now turn to the **acute disease with hyper-resonance on percussion**—viz., Pneumothorax. Bear in mind that an acute disease may supervene upon a chronic condition accompanied by hyper-resonance—*e.g.*, when acute bronchitis supervenes on emphysema (see Table VIII, § 142).

The patient is in MARKED DISTRESS, *which has come on* SUDDENLY; *there is cyanosis, often hyper-resonance and absence of breath sounds, or faint bronchial breathing over one lung. The disease is* PNEUMOTHORAX.

§ 126. **Pneumothorax** is a term used to denote the presence of air in the pleural cavity, the air having gained admission by perforation of the pleura, either from within or from without. Effusion may form (hydropneumothorax) which, after a time, may become infected. The condition is then known as pyopneumothorax.

The *Symptoms* of the onset of the condition differ according to the condition of the lung—*i.e.*, whether it is fairly healthy or is widely diseased. (*a*) When pneumothorax occurs in the less affected of the two lungs—the other side being extensively diseased—the symptoms are very urgent, and consist of severe pain in the side, attended by great dyspnœa, shallow, quick breathing, cyanosis, and some degree of collapse, with sweating,

lividity, and a weak pulse. (*b*) In other cases, where pneumothorax comes on in a lung which is already much diseased, the onset and the physical signs may be hardly noticeable. (*c*) The occurrence of spontaneous pneumothorax in the apparently healthy, though uncommon, is not so rare as has been supposed. The onset of symptoms is sudden, often apparently a result of strenuous physical effort, *e.g.*, during a game of football, the degree of disability and distress depending on the amount of air which gets into the pleural cavity and the resulting degree of displacement of the heart and mediastinum.

The *Physical Signs* in the chest consist of: (i.) Diminished movement on the affected side; (ii.) diminished tactile vocal fremitus; (iii.) hyper-resonance on percussion; (iv.) on auscultation the respiratory murmur is reduced or absent; amphoric breathing may be heard over the lower half of the chest behind; the vocal resonance is usually diminished, but pectoriloquy and bronchophony are sometimes present; (v.) often displacement of the heart to the opposite side. The *bell sound* may be elicited on tapping the chest with two coins in one position, and listening with a stethoscope in another. When fluid is also present (hydropneumothorax), and this is usual, metallic tinkling is heard. The *succussion splash* is the most characteristic sign of hydropneumothorax—a fact which was well known to Hippocrates. It may be heard by placing one's ear against the chest whilst moving the patient's body from side to side.

Etiology.—(i.) A common cause is phthisis, when the ulcerated portion of the lung or an emphysematous bulla bursts into the pleura. (ii.) Spontaneous pneumothorax in young and apparently healthy adults is believed to be due to the rupture of an emphysematous bulla in the subpleural region of the lung. Such emphysematous bullæ may be associated with an old primary tuberculous focus, although there may be no evidence of tuberculosis in the past or subsequent history. (iii.) A fractured rib may lead to perforation of the lung. (iv.) Less common causes are gangrene of the lung, or an abscess connected with the spine or liver bursting into the pleural cavity.

Prognosis.—The prognosis in pneumothorax is often grave, but depends upon the cause. The heart and mediastinum may be pushed right over by the accumulation of air at a raised pressure in the pleural cavity: this, if unrelieved, may be fatal. The *immediate* risk depends upon the urgency of the dyspnœa and cyanosis, the state of the other lung, the patient's general health, and the cause of the condition. As regards the *cause*, the pneumothorax that results from late phthisis or gangrene of the lung is fatal; but that which occasionally complicates whooping-cough, pneumonia, early phthisis, and injury, usually results in recovery. Certain it is that the longer the patient lives after the onset of the pneumothorax, the better is the prognosis for ultimate recovery (§ 131). Death usually occurs from shock and cardiac failure, associated with gross displacement of the mediastinum, unless this can be prevented by artificial removal of air from the pleural cavity.

Treatment.—The usual remedies for shock are indicated. The patient must be kept as still as possible, preferably semi-recumbent, though sufficiently propped up to avoid increasing respiratory distress. Warmth should be applied to the extremities and stimulants may be necessary. Oxygen may aid if there is marked cyanosis. If there is severe pain, or if the patient is very restless and in great alarm, small doses of morphia may be indicated. Air must be removed from the pleural cavity when great distension is present, as indicated by marked displacement of organs, extreme pain and discomfort, but the relief is often only temporary. In these circumstances continuous aspiration of air must be carried out until the hole in the lung becomes sealed off. If the lung is seriously diseased, it may be kept collapsed (see § 131, Artificial Pneumothorax). When pus is present, it should be aspirated.

There is one disease of the lungs which belongs neither to the acute nor to the chronic category, but is **paroxysmal,** *occurring in attacks of sudden onset, usually* WITHOUT ELEVATION OF TEMPERATURE—ASTHMA.

§ **127. Asthma** is characterised by paroxysmal attacks of dyspnœa, the inspiratory effort being short, the expiratory prolonged. In severe cases there may be much cyanosis and distress. Chronic bronchitis is liable to complicate asthma, but it is important here to draw attention to the frequent error which is made by regarding exacerbations of chronic bronchitis as paroxysms of asthma.

Symptoms and Clinical History.—The leading characteristic of this disease is its paroxysmal nature. A person who is subject to asthma may be perfectly well one minute, and half an hour later may be seized with the most violent dyspnœa. An attack often commences in the early morning, the patient awakening with a feeling of tightness of the chest; he coughs and gasps for breath and wheezes, and clings to surrounding objects in order to bring into play the accessory muscles of respiration. An attack is often associated with a paroxysmal cough, and a plug of mucoid sputum is ultimately expectorated with corresponding relief. Each attack lasts from a few minutes to a few days, and then, without apparent reason, the patient rapidly recovers his normal health.

There are many curious features in connection with this malady, one of the most interesting being the tendency to skin eruptions (urticaria, prurigo and eczema), and another the fact that these eruptions may alternate with the attacks of dyspnœa. Hay fever, migraine, and even attacks of epilepsy, may alternate in the same way. The paroxysms of asthma are occasionally preceded, ushered in, or terminated by violent attacks of sneezing, or by itching; large quantities of urine may be passed as an attack subsides.

Physical Signs.—On inspection the chest is seen to be maintained in a position of inspiration, undergoing but little expansion with the short inspirations. The percussion note may be unaltered, but, after many attacks, emphysema supervenes, with consequent hyper-resonance. On auscultation the short inspiratory effort is feeble and scarcely audible;

expiration is prolonged. Loud rhonchi and often coarse râles replace the normal vesicular murmur.

Etiology.—The central fact, which alone explains all the symptoms, is a narrowing of the bronchial tubes, due to spasm of the involuntary bronchial muscles, with hyperæmia of the submucosa and swelling of the mucous membrane. Asthma is to be regarded as a manifestation of allergy (§§ 521, 609). The association of the attacks with the presence of certain animals, especially the horse and cat, has long been recognised. It is now claimed that about half the asthmatics tested intradermally with foreign proteins obtained from animals, feathers, foods, etc., show increased sensitivity to some of these substances. In certain cases, indeed, it has been possible to "desensitise" patients so that they no longer have attacks of asthma. Although this recent treatment has often proved disappointing, skin testing is of value since it may reveal a hypersensitiveness to substances which can readily be avoided. The cases which are of allergic origin usually start in early life, often before the age of 10 years. Bronchial spasm may not commence until much later in life, and may suddenly supervene during an acute attack of bronchitis: such cases often give no family or personal history of allergy, and intradermal skin tests are usually negative.

Among the *predisposing* causes we find: (i.) A family history of allergy. Careful inquiry may reveal asthma, hay-fever, urticaria, Besnier's prurigo and infantile eczema. (ii.) Asthma may occur at any *age*, but frequently makes its first appearance soon after the age of puberty. (iii.) Any previous lung disease, especially chronic bronchitis, may predispose to asthma.

Among the *exciting* causes of an attack may be mentioned: (i.) Certain atmospheric conditions which are little understood, and often appear to be most contradictory. Thus I know one patient who is free from asthma in London, but develops an attack immediately she seeks a high altitude. Another develops an attack when she enters London. Some find that the sea relieves them, others that it determines their attacks. (ii.) Reflex causes, such as derangement of the alimentary canal, dietetic indiscretions, or a large evening meal; (iii.) conditions of the nasal passages, such as sinusitis, hypertrophic rhinitis, or polypi; (iv.) dust and irritating particles; (v.) conditions as in hay fever, *e.g.*, proximity to horses, or certain plants; (vi.) emotional causes.

Diagnosis.—The diagnosis usually presents no difficulty. The paroxysmal occurrence of the disease is quite characteristic. Paroxysms of dyspnœa coming on at night occur in the course of chronic nephritis and cardiac disease, and have been loosely called asthma. The actual substances producing asthma may often be determined by performing a series of intradermal inoculations with solutions of the proteins of different substances, *e.g.*, rye, wheat, eggs, feathers, grass, etc., and thus certain definite indications for treatment may be obtained.

Prognosis.—The disease of itself rarely causes death during an attack;

it tends to produce emphysema, bronchitis, and increasing embarrassment of the right heart. Children may grow out of the disease; adults never lose it completely. The severity, frequency, and response of the attacks to treatment are our best guides to prognosis.

Treatment.—(*a*) *During the Attack.*—A subcutaneous injection of adrenalin (1 in 1,000) often aborts an attack. The earlier the drug is given, the smaller the dose; even one to two minims may abort an attack. Inhalation of an adrenalin spray with oxygen, as with the Apneu apparatus, is often efficacious. Pituitrin may be combined with adrenalin, and may be more effective than either drug given separately. Ephedrine tablets are valuable, and in certain cases remove the need for adrenalin injections. Cardophylin (a purin derivative) is a valuable antispasmodic of which 0·24 G. in 10 c.c. of sterile water may be given intravenously or 0·48 G. in 2 c.c. of sterile water by intramuscular injection. The proprietary preparation Riddobron may be effective. Benzyl-benzoate, stramonium, lobelia, belladonna and hyoscyamus may be tried. Atropin and cocaine sprays also relieve. Various inhalations are useful for the prevention or relief of an attack—*e.g.*, the vapour from a teaspoonful of turpentine and chloroform (chloroform may be pushed to anæsthesia), or the fumes of paper prepared with a strong solution of nitrate of potash, or amyl nitrite. A mixture containing equal parts of the leaves of stramonium, lobelia, black tea, and potassium nitrate is burnt in a tin plate, and the fumes inhaled; relief is thus sometimes afforded. Various other preparations, in the form of cigarettes of stramonium, potassium nitrate, and belladonna, are used, but should be discouraged, as their frequent use tends to produce chronic bronchitis. Morphia is helpful, but the risk of addiction is considerable.

(*b*) *Between the Attacks.*—The effect of locality on the disease can only be ascertained by experience. As a rule, though with many exceptions, town air and fogs are detrimental. It is better for patients to live in the upper storey than on the ground floor of a dwelling. The patient's general health must be especially considered, and anxiety, worry, and other psychological factors must receive particular attention. Reflex causes should be eliminated as far as possible: avoid large bulky meals at any time and solid meals after 2 p.m. The nose should be examined for polypi, etc., and all other sources of reflex irritation must be sought for and treated. Ephedrine, gr. ½, at bedtime, together with a simple hypnotic such as aspirin and phenacetin or chloral and bromide, or a capsule of theamine (gr. 3) will often avert nocturnal attacks. The patient should avoid the substances to which he is particularly sensitive, but specific desensitisation has on the whole been disappointing. Injections of normal horse serum (given every fourth day in doses of 4 minims increasing to 2 c.c.) may cure the asthma due to the proximity of horses. Non-specific desensitisation may be tried by injecting Armour's No. 2 peptone intramuscularly (7½ per cent. solution), or intravenously (5 per cent. solution), starting with 0·3 c.c. and increasing to 2·5 c.c., each third or fourth day.

Weekly intramuscular injections of 10 c.c. of the patient's own blood have often resulted in considerable benefit, without the occurrence of untoward reactions. When the asthma is mainly associated with acute bronchitis, the treatment is that of the primary disease. Potassium iodide is especially useful in liquefying the sputum, and if small doses (3 grains) are not sufficient, much larger doses may be employed. The antispasmodic drugs are again very useful. In some cases benefit may result from the use of an autogenous vaccine. In children, regular doses of glucose, and avoidance of undue excitement or mental strain of any kind, are advisable.

Pulmonary Acariasis.—Asthma due to infestation with mites has been described in India and Ceylon: it is characterised by marked eosinophilia in the blood.

CHRONIC DISEASES OF THE LUNGS AND PLEURÆ

128. Classification.—Chronic disorders of the lungs and pleuræ may follow an acute attack of the conditions described in the previous sections, as when chronic bronchitis and emphysema succeed attacks of acute bronchitis. But many of the chronic diseases of the lungs, such as pulmonary tuberculosis, start insidiously, and attention may not be directed to the lungs for a considerable time.

The chronic diseases, like the acute, may be classified, *for clinical purposes*, according to the results of percussion. It is convenient in actual practice, although unscientific from the point of view of classification, to make a subsidiary group in which the sputum is highly offensive or has some other characteristic feature.

(A) **Chronic Disease** in which the **Percussion Note is unaltered**:

I. Chronic bronchitis .. .. § 129

(B) **Chronic Diseases** attended by **Impaired** or **Dull Percussion Note**

(*a*) The *commoner* diseases presenting dulness, *usually in regular and defined areas either at base or apex*, are—

I. Chronic tuberculosis of the lung .. .. § 131
II. Hydrothorax .. .. § 134
III. Pulmonary congestion (hypostasis or œdema of the lungs).. § 135

(*b*) The diseases presenting dulness, *usually not* in regular and *defined areas at base and apex*, are—

COMMON.		LESS COMMON.	
IV. Pulmonary fibrosis (with or without bronchiectasis)	§ 136	VIII. Collapse of the lung	§ 139
V. Thickened pleura ..	§ 137	IX. Hydatid cysts ..	§ 140
VI. Malignant disease of the bronchus	§§ 81 and 138	X. Syphilis of the lung..	§ 141
VII. Secondary malignant disease of the lung	§ 138	XI. Sarcoidosis	§ 141*a*
		XII. Diseases due to fungi and parasites	§§ 145, 146

(C) **Chronic Diseases** attended by **Hyper-resonance :**

I. Emphysema .. .. § 142

II. Pneumothorax[1] and various other conditions in which the hyper-resonance is not the leading or constant feature (*e.g.*, Skodaic resonance) .. .. .. § 126

(D) **Diseases** suggested by the **Character of the Sputum :**

I. Bronchiectasis §§ 136 and 143

II. Abscess and Gangrene of the lung .. .. § 144

III. Actinomycosis and other diseases due to fungi §§ 145, 146

GROUP A.—The patient's symptoms point to **chronic disease of the lungs,** and on examining the chest there is **no alteration in the percussion note.**

I. *The patient has a chronic cough ; there is no elevation of temperature, and on auscultation* RHONCHI *and* RÂLES *are heard over the chest. The disease is* CHRONIC BRONCHITIS.

§ **129. Chronic Bronchitis** is a chronic inflammation of the bronchial tubes. It usually supervenes on repeated attacks of the acute disorder, but may be chronic from the beginning.

Symptoms.—A patient with chronic bronchitis and—its usual sequel —dilated right heart, often presents a typical appearance. Stout in build, with short, thick neck, of florid, slightly cyanosed complexion, short of breath, wheezy respiration, and pulsating jugular veins, he presents an aspect which can be recognised at once. The clinical history extends over many years, with alternate diminution and aggravation of the symptoms. The cough is usually present during the winter, and improves as the weather gets warmer. The constant coughing and straining to bring up the secretion results sooner or later in generalised emphysema. In later stages the cough continues all the year round, and finally an attack of bronchopneumonia, œdema of the lung, or some intercurrent malady, throws a little extra strain upon the overburdened right heart, and death ensues. There are, as a rule, no febrile or constitutional symptoms.

The *Physical Signs* vary with the amount of secretion present, the extent of the complicating emphysema (§ 142), and the degree of accompanying bronchial spasm. In cases of long duration the chest is barrel-shaped (emphysematous, § 106). It moves poorly. Rhonchial fremitus may be felt on palpation. On percussion there is never any dulness, and the note is hyper-resonant in proportion to the emphysema present. On auscultation sibilant and sonorous rhonchi and bubbling râles can be heard ; crepitations at the bases, due to œdema, may be present.

There are five recognised varieties of this disease : (i.) *Bronchitis with winter cough,* attended by slight or abundant expectoration, mucous or muco-purulent, sometimes fibrinous, sometimes containing streaks of blood. (ii.) *Dry Bronchitis* (*catarrhe-sec* of Laennec) is attended by a

[1] Pneumothorax usually comes on acutely, but it may be part of a chronic disease.

frequent cough and soreness of the chest, but little or no secretion; it is of a very obstinate character, and occurs mostly in elderly people of a gouty diathesis. (iii.) *Purulent* and/or *fœtid bronchitis* characterised by expectoration of large quantities of purulent and offensive sputum; associated with bronchial dilatation (*cf.* bronchiectasis). (iv.) *Bronchorrhœa* signifies expectoration of very large amounts of sputum, often of a thin clear nature or else thick and ropy. It is a symptom rather than a disease entity, and is often associated with bronchiectasis (*q.v.*). (v.) *Plastic Bronchitis*, described in § 130.

The *Diagnosis* of chronic bronchitis is usually easy. It is diagnosed from *chronic phthisis* partly by the absence of the tubercle bacillus from the sputum, but chiefly by the absence of radiological evidence of infiltration of the soft tissues of the lung. It is important to remember that bronchitis and emphysema may mask chronic tuberculosis. Such patients may be afebrile and bear no other clinical sign of the more important disease. As they are sources of danger to others, the sputum should always be examined for Koch's bacilli.

Etiology.—Chronic bronchitis may occur at any age, but is more common in elderly people. Sometimes, as before stated, it follows repeated attacks of acute bronchitis, but it may be chronic from the beginning. It often affects plethoric subjects, especially those of a gouty habit, and it is a recognised complication of chronic nephritis. It is a frequent sequel to cardiac valvular disease, more especially disease of the mitral orifice. It may complicate other diseases of the lungs, especially phthisis, and may follow the acute specific fevers, especially measles and whooping cough.

Prognosis.—Adults with chronic bronchitis seldom entirely recover, though they may live for a great many years; and if the heart is fairly healthy and care be taken to avoid exposure, life is not very materially shortened. The co-existence of gout, chronic nephritis and cardio-vascular degeneration make the prognosis somewhat less favourable. The condition of the lungs is not so much a guide to prognosis as the condition of the heart. This, indeed, is the point around which the prognosis centres, and the untoward symptoms which render the prognosis grave are thus referable to the heart—viz., considerable dilatation of the right heart with evidences of cardiac failure, such as great breathlessness, cyanosis, enlargement of the liver and veins of the neck, and ascites.

Treatment.—The extreme frequency of the disorder renders the treatment a matter of considerable importance. In acute exacerbations the treatment is that of acute bronchitis (§ 115). In slight cases, however, the patient can go about, but chill and exposure should be avoided. The important question of when a patient may go out must depend largely on the weather—cold and moisture, especially when in combination, are especially injurious. The choice of a suitable climate is of importance.

The chief points in treatment are: (i.) To repress an excessive cough, since this throws increased work on the right side of the heart.

For this purpose tr. opii camph. in large doses, or heroin lozenges may be used. (ii.) When the cough is dry, remedies directed to promote the secretion are given, such as ipecac., potass. iod., and alkalies. (iii.) When the sputum is too abundant, we may have to diminish secretion by such remedies as belladonna. Counter-irritants to the chest—*e.g.*, turpentine, camphor, or eucalyptus, are very popular with some. (iv.) When there is much spasm of the tubes, lobelia, iodide, ephedrine, and other remedies for asthma are to be tried. (v.) Measures to prevent dilatation and failure of the right ventricle are called for sooner or later where dyspnœa and other cardiac symptoms are present. (vi.) In suitable cases, vaccines have been found useful.

§ 130. **Plastic Bronchitis** is inflammation of the bronchi, with the formation of fibro-plastic casts, which are expectorated.

Symptoms.—The symptoms consist of (i.) violent attacks of coughing, with pain in the chest and expiratory dyspnœa, followed by (ii.) the expectoration of a fibrinous cast of a bronchus. (iii.) The patient generally suffers from chronic bronchitis, and a little hæmoptysis may follow the expulsion of a cast. (iv.) Sometimes there are no constitutional symptoms, but slight pyrexia, and in some cases even rigors may be present. Such symptoms supervening in a case of chronic bronchitis lead us to suspect the condition.

Physical Signs may be absent. If present, they are those of an obstructed bronchus —an absent or diminished respiratory murmur, accompanied possibly by impaired percussion note. Whistling rhonchi or "flapping" sounds may be heard.

Etiology.—The disease is twice as common in men as in women. It may occur at any age in subjects of chronic bronchitis.

Prognosis.—The condition is more serious than simple bronchitis. Two varieties have been described: (1) An acute form, lasting for some weeks; and (2) a chronic form, recurring at intervals for years, in the course of chronic bronchitis. Each attack may last for some weeks, and the casts may be coughed up daily. The condition occasionally leads to a fatal issue.

The *Treatment* differs but little from that of bronchitis. The removal of the membrane may be promoted by the administration of potassium iodide in order to liquefy the sputum: the inhalation of a weak solution of sodium bicarbonate atomised by means of a spray, in order to dissolve the mucin in the cast, has been advised by some. Various oils (*e.g.*, creosote oil, 1 in 40) have been injected as solvents, but the results have not been very promising.

GROUP B.—We now turn to those chronic diseases of the lungs which are accompanied by **dulness on percussion.** (*a*) The *common* diseases in which the dulness occurs, usually in regular and fairly DEFINED AREAS at base or apex, are: I. CHRONIC PULMONARY TUBERCULOSIS; II. HYDROTHORAX; III. PULMONARY CONGESTION OR ŒDEMA.

I. *The patient complains of gradual emaciation and perhaps cough; on examination of the chest* SIGNS OF CONSOLIDATION *may be found, most marked at the* APEX *of the lung; there is* INTERMITTENT PYREXIA, *and the sputum may contain tubercle bacilli. The disease is* CHRONIC PULMONARY TUBERCULOSIS (*Phthisis*).

§ 131. **Chronic Pulmonary Tuberculosis** (Phthisis). The word phthisis is objectionable because it only indicates one of the symptoms—viz., the wasting (φθίνω, I waste). For a full exposition of the pathogeny of

this disease the reader is referred to works on pathology. A brief account of the modern view may not be out of place. In civilised communities the universality of the infection by the age of puberty is generally admitted. The newly-born infant, even of a tuberculous mother, is not infected, and there is abundant evidence that children born of tuberculous parents are no more liable to the disease than other children, provided they are separated from possibility of massive infection.

Pulmonary tuberculosis is caused mainly by the human form of Koch's bacillus and is spread by direct contact; it has been shown that droplets of sputum sprayed from the mouth of a tuberculous subject may contain enormous numbers of bacilli. The first occasion of infection by tubercle bacilli, provided the dose is not large enough to cause immediate serious disease, results in a state of allergy or increased sensitiveness, after which a further dose of bacilli will call forth a type of local reaction not seen with the first inoculation (Koch's phenomenon). This reaction localises the bacilli, tending to destroy them and prevent their entrance into the body. If the dose (exogenous superinfection) is too great this reaction produces a more or less severe local lesion in which the bacilli spread. The virulence of the human form of tubercle bacilli varies little; the type of pulmonary disease produced depends on the size of the dose and the resistance of the patient. Certain diseases are known to reduce the resistance to tuberculosis; thus, after measles and whooping-cough, the disease is common in one form or another. After such maladies active disease may begin (without further infection from the exterior) from a previously healed endogenous focus (endogenous re-infection). Because of the variations in resistance, the course of the disease is very variable. It may cause death in a few weeks, or it may spread in successive areas so slowly as to cause little or no disability over a long lifetime. Such cases are dangerous sources of infection, especially to young children. They are usually without fever. The chief safeguard is to examine all specimens of sputum for tubercle bacilli.

Symptoms.—The disease is mainly a chronic one, and its onset is sometimes insidious. It is more amenable to treatment in the early stage, and since the introduction of modern methods of treatment early recognition of the disease has become of paramount importance.

(*a*) *Early Stages.*[1]—Clinically, phthisis has various modes of onset, e.g.: (i.) Progressive weakness, attended perhaps by cough; (ii.) hæmoptysis[2]; (iii.) dyspepsia; (iv.) tachycardia; (v.) pleurisy; (vi.) acute pneumonia (§ 121), bronchitis, or broncho-pneumonia. The pneumonic form may resolve almost completely, leaving a chronic lesion, or sometimes cavitation may occur with great rapidity and not long after the onset. Among the earlier *general symptoms* which should make us suspect the

[1] It must be remembered that in an appreciable number of cases active phthisis in the early stage may be an entirely symptomless disease, only recognisable by radiography (*vide infra*).

[2] Early hæmoptysis may occur before any physical signs are discoverable, except by the use of X-rays.

invasion of tuberculosis are unexplained debility, attended by languor and pallor on the one hand; or on the other hand loss of weight, with unexpected dyspepsia, or slight elevations of temperature in the evening. The temperature is an indication of the greatest importance, for *an* ACTIVE *tuberculous process is usually associated with pyrexia*, however slight. The type of this pyrexia is distinctive; it is *intermittent*, being normal or subnormal in the morning, and raised in the afternoon or at night; in rare instances this is reversed. If we have any suspicion of tuberculosis, the temperature should be taken every two hours, so that we may not miss any slight rise during the day. To avoid missing slight fever, keep the minute thermometer in place for six minutes. In the early stage the patient may not be aware of the feverishness, though occasionally he feels a chilliness in the evening, and as the disease progresses, night sweats are a characteristic feature.

The *Physical Signs* accompanying the earlier stages are necessarily somewhat vague and difficult to detect. The patient's chest should be completely stripped, and he should be taken to a room where perfect quiet prevails; and if with the above symptoms we find weak or unduly harsh breathing and prolonged expiration at one apex—especially if this is accompanied by fine crepitations—we must examine the sputum and have a radiograph taken. It is important to auscultate while the patient coughs, for râles not previously audible may thus become evident. The signs just named can often be heard best at the apex, behind, by placing the patient's hand on his opposite shoulder and listening to that part of the lung, which will thus be *uncovered by the scapula.* Fine crepitations may be heard in that situation weeks before any signs may be discovered at the apex in front. In front the earliest signs may be heard just below the clavicle. Sometimes, later on, we are led to detect phthisis by an undue loudness of the *heart* sounds at the apex of one lung. Absence of dulness, like the absence of bacilli, is not evidence of the absence of tuberculosis. The *sputum* should be repeatedly examined for tubercle bacilli, the presence of which is diagnostic. The early morning sputum is the most likely to contain the bacilli. In repeatedly negative cases bacilli may often be shown in the gastric contents or stools or on culture of the sputum. However, the absence of bacilli, even after several examinations, does not always indicate the absence of phthisis. X-ray examination by an expert is ABSOLUTELY ESSENTIAL in all cases of hæmoptysis or of cough lasting more than three weeks. It cannot be too strongly emphasised that in many cases an active spreading tuberculous granuloma in the lung is unaccompanied by any symptoms and is only recognisable by radiography.

(*b*) *Later Stages.*—The physical signs usually begin at the apex, and are generally best heard at the back, sometimes at the apex of the lower lobe. Extensive tuberculous disease may sometimes exist with but little constitutional disturbance; on the other hand, considerable disturbance of health may be present without any abnormal physical signs—depending,

partly, on the distance of the lesion from the surface of the body. It has many times been proved that a cavity can exist and bacilli be found in the sputum of a patient presenting none of the usual physical signs. X-ray and sputum examination are therefore essential in diagnosing any obscure case, especially if cough is present.

The presence or absence of A CAVITY is in the majority of cases impossible to diagnose with certainty by physical examination alone. The percussion note is usually dull, but varies with circumstances. Thus the note may be resonant when the cavity is very large, or lies very superficially. When the cavity is large and superficial, and the communicating bronchus remains patent, a characteristic note, almost tympanitic, is obtained on percussion whilst the patient keeps his mouth open. This is known as the "cracked-pot" sound (*bruit de pot felé*). The breathing is amphoric.

The *Diagnosis* of the disease is not difficult except in the really early stages. (i.) Other causes of hæmoptysis may have to be differentiated (see § 104); (ii.) other causes of debility may have to be eliminated (Chapter XVI); (iii.) when the condition begins with dyspepsia, it is very liable to be overlooked unless the physician is aware of this mode of commencement; (iv.) other causes of cough (§ 101); and (v.) various pharyngeal and laryngeal affections may have to be excluded (§ 165). In the later stages the differentiation from the other causes of percussion dulness is not difficult (see list in § 128).

Classification.—Reference has been made to (*a*) the earlier stages and (*b*) the later stages of this disease, and a brief account of the symptomatology and physical signs has been given under each heading. However, correlation of the pathological with the clinical aspects of phthisis is not so obvious as most of the older text-books have led one to suppose. The Turban-Gerhardt classification, accepted for so long as the orthodox description, was based upon the anatomical character and extent of the pulmonary lesions, with which the clinical manifestations were assumed to correspond more or less accurately. Such a division of cases is so fallacious as to be extremely misleading; therefore the Turban-Gerhardt classification should be regarded as obsolete. Inman's division of cases of adult phthisis into three main groups: (1) *ambulant afebrile,* (2) *resting afebrile—ambulant febrile,* (3) *resting febrile,* conforms more closely to the actual clinical facts. The extent of the lung lesion is in some degree important, although extensive disease may exist with but little constitutional disturbance. X-ray examination at intervals can and does reveal the development of fresh lesions in patients who are practically without symptoms, and who not only are afebrile but may even be increasing in weight. The age of the patient, the length of time during which manifest clinical disease has developed, the character of the adventitious shadows in the radiogram, etc., are also points to be taken into account in the assessment of any individual case; for their proper appreciation reference must be made to special works.

Etiology.—To find tuberculosis in a newly-born child is extremely rare, even when the mother is in the last stages of the disease. The

separation of children at birth from infected mothers has met with undoubted success in France. The mortality rate of children so removed and brought up by foster parents justifies the modern belief that there is no such thing as inheritance of tuberculosis. Both sexes are almost equally affected, and the age at which the disease usually appears clinically is between sixteen and thirty. The patient may be attacked at any time of life, although clinical signs of lung disease are rare under two years. Any condition of malnutrition may produce a predisposition to the bacillus invasion, whether it arise from deficient food, from hyperlactation, from exhausting diseases such as diabetes, or the acute specific fevers, after which an attack of phthisis is by no means infrequent. It is a curious circumstance that pregnant women offer a high resistance; a phthisical subject becoming pregnant will frequently improve until after her confinement, when an exacerbation of the disease will occur often with a fatal result. Unhealthy surroundings play an important part in the spread of tuberculosis; indoor occupations in over-crowded and ill-ventilated rooms are especially dangerous. Excessive exposure to sunlight, artificial or natural, has sometimes precipitated an acute attack. A silica-laden atmosphere, such as that of stonemasons, knife-grinders, tin and copper miners, fustian-cutters, makes tuberculosis more serious. The exact effect of silicosis on tuberculosis has not yet been fully explained. The 1911 report of the Royal Commission on Tuberculosis confirmed the view that tuberculosis in mankind was due to two types of tubercle bacillus, one of human and one of bovine origin. Pulmonary tuberculosis is for the most part due to infection by the human bacillus, which is conveyed by droplet infection or by dust containing living bacilli—hence the importance of destruction of the sputum. In children, the bovine bacillus is found chiefly in the abdomen (peritoneum or glands), bones, joints, cervical glands, and in the lungs in miliary tuberculosis when the bacillus is carried from an infected focus by the blood-stream. Recent researches in Great Britain have shown that an appreciable number of cases of pulmonary tuberculosis are due to the bovine type of bacillus (*cf.* p. 203). The bovine bacillus enters the body *via* the alimentary canal and tonsils, and causes disease by the ingestion of infected milk of tuberculous animals. It is, however, a fact that mankind is naturally resistant to the tubercle bacillus. Birch-Hirschfield found in 4,000 post-mortems of persons dying from various diseases, that in 40 per cent. the lungs showed evidences of tuberculosis which had undergone spontaneous recovery. Other investigations show even a higher figure.

Prognosis.—1. Usual *course* and *duration*. Phthisis is essentially a chronic but progressive disorder, and formerly nearly all cases applying for treatment terminated fatally. The standardised annual death-rate from tuberculosis of the respiratory system, per million persons living in England and Wales, was 1,517 in 1892 and 532 in 1938. During the war of 1939–45 it rose sharply but fell to 473 (for civilians) in 1947. Rapid cases may terminate in death in the course of three to six months. When

the disease is indolent, and the patient resistant to the bacillus, it may drag on for years. There are four chief modes of death, which in order of frequency are—(1) asthenia, (2) hæmoptysis, (3) bronchitis and heart failure, (4) the occurrence of other complications.

2. The prognosis in individual cases has been greatly improved by four main advances in treatment: (a) early diagnosis; (b) rest and open air; (c) artificial pneumothorax; (d) surgical methods of collapse-therapy. The type of disease, its rate of progress, and its distribution are all important in prognosis. The age of the patient influences the course considerably, for it is much more rapid in the young adult than in people over thirty. The hygienic surroundings of a patient, as we shall see under *Treatment*, make a considerable difference to the course of the disease. Where the patient can be properly treated with rest, open air, and good food, he has nowadays a good chance of recovery. A correct prognosis cannot always be made until the patient's reaction to treatment is observed.

3. *Untoward Symptoms.*—(i.) Undoubtedly the most important feature is the temperature. Not only is active tuberculosis evidenced by pyrexia, but the degree of fever, and still more the extent of the diurnal variations, are a fairly precise measure of the activity of the tuberculous process. (ii.) The pulse rate is also important, and, quite apart from the temperature chart, is valuable as a measure of toxæmia. (iii.) The condition of the lung itself is, of course, not without significance, but physical signs must be interpreted in the light of additional evidence furnished by the temperature and pulse charts, and especially by serial X-rays. Variations in the adventitious sounds within a comparatively short period are of greater significance than the persistence of râles which remain unchanged in character. An uncollapsed cavity, with a positive sputum, is always a potential source of danger, and augurs a poor outlook for the ultimate future, though individual patients in such circumstances can live for many years in apparently good health and without much disability. (iv.) Absence of symptoms and increase in weight are of good import, subject to the proviso already mentioned in regard to the warning frequently given by the X-rays. (v.) Hæmoptysis does not appear to bear any constant relation to prognosis, though profuse hæmorrhage weakens the patient considerably, and may be fatal; it is occasionally followed by extension of the disease.

4. *Complications.*—The presence of complications is undoubtedly bad. The commonest complications are: (1) Pleurisy is very frequent, but the adhesions may be beneficial in preventing spontaneous pneumothorax[1]; (2) tubercle may occur in other parts—the peritoneum, meninges, and especially in the intestine, giving rise to ulceration and an exhausting diarrhœa[2]; (3) the larynx may be affected, and undoubtedly this adversely

[1] On the other hand, adhesions may prevent an efficient lung collapse when this treatment is indicated.

[2] Diarrhœa may occur as part of the toxæmia without ulceration of the bowels.

influences the prognosis; (4) pneumothorax and pyopneumothorax may ensue from the bursting of a cavity into the pleura—fatal asphyxia *may* result (§ 126); (5) thrombosis of various veins is a less common complication; (6) peripheral neuritis is now a recognised occurrence, sometimes very early in the disease; (7) tuberculous pericarditis (rare).

Treatment.—The subject of treatment will be dealt with under six headings: (*a*) open air and rest; (*b*) artificial pneumothorax; (*c*) thoracic surgery; (*d*) symptomatic treatment; (*e*) other measures not widely used; and (*f*) preventive measures.

(*a*) The "*open-air*," hygienic, or sanatorium treatment of phthisis, as it is now called, is not altogether a new method, for fresh air has always been advocated as advantageous to these patients. Systematic open-air treatment was first established at Nordrach. There are now numerous sanatoria both at home and abroad. Much discussion has taken place as to whether the treatment cannot be carried out without a sanatorium. Among the well-to-do, perhaps, a sanatorium is not indispensable, but in the middle and lower classes the necessary discipline cannot be otherwise ensured. The dryer atmosphere and absence of fog found at certain mountain resorts are desirable, but by no means essential. Great heat is deleterious. Sea voyages are not recommended.

Briefly, the *advantages* gained by this method of treatment consist of: (i.) Increased medical supervision from day to day and hour to hour by the medical officer of the sanatorium; (ii.) the continuous exposure of the patient to fresh pure air, night and day, the windows never being shut and sometimes being wholly removed; (iii.) graded exercise; (iv.) nourishing and sufficient food; (v.) a suitable amount of rest during the fever stage, and a freedom from excitement; (vi.) the avoidance of mixed infections by hygienic mode of life. Cleanliness and fresh air tend to obviate pyogenic processes and infections. All possibility of the introduction of influenza and other infective disorders should be avoided by the *proper regulation of visitors* to patients. I believe that some day these latter will be subjected to the most rigorous scrutiny and inquiry before being allowed to come in contact with the consumptive patients in a sanatorium. The treatment varies at the different sanatoria. Patients who return to ordinary life, return with a working knowledge of the hygienic rules appropriate for consumptive subjects.

With regard to sanatorium treatment, important points are:

(1) Much depends on the suitability of the case, and the *earlier the stage* the better.

(2) The food must be abundant, and the cuisine appetising and attractive, but here an important caution comes in, else the patient puts on fat without influencing the disease. The food must be properly balanced and in proportion to the exercise; the patient's weight should never much exceed his previously normal weight. The protein food should be increased relatively to the farinaceous, otherwise the patient becomes plethoric and breathless. The bowels must be regulated.

(3) Evidences of benefit should be carefully looked for. They are (i.) A lowering of the temperature and a lessening of its range; (ii.) an increase in the appetite; (iii.) increase of weight *combined* with the two previous features; (iv.) improvement in radiographic appearances.

(4) In deciding the important question of rest or exercise, the great value of accurate temperature records is again seen. The system of graduated labour introduced at Frimley Sanatorium by the late Dr. Marcus Paterson [1] is being followed by many with excellent results. There were originally six grades of labour, varying from walking exercise, carrying heavy implements, to the full work of a navvy. The patient is not allowed to begin work until the temperature is stable at not over 99° F. in males, and 99·6° F. in females. If it rises after slight exercise, the patient rests until it is normal. Progressively heavier work can be performed without any rise of temperature.[2] The physical and mental effects are invigorating, and enable the patient to resume ordinary occupation after leaving the sanatorium with a healthier standpoint than after a long rest with idleness.

(5) Amusement is necessary, but it requires to be carefully regulated. The patient should not talk too much, and any excitement or heated discussion is bad. *The whole day, and, if possible, the night also, should be spent out of doors, no matter what the weather may be,* and outdoor amusement cultivated. A useful contrivance is a small revolving summer-house, the front of which is open, and can be turned away from the wind.

(6) The duration of the treatment must be sufficient, and should be continued for some time after all symptoms have disappeared. The patient may in favourable cases be able to resume a more or less ordinary life after six months' treatment, but careful medical supervision is necessary for several years.

(7) The hygiene and the locality of the building are important matters, but the reader must refer to special works for this.

(*b*) *Artificial pneumothorax* is indicated when there is extension of a one-sided lesion in spite of routine treatment. After a short time of treatment with open air and rest it is usually possible to decide whether the disease is being arrested. When, in spite of these measures, the disease still spreads, artificial pneumothorax should be attempted. To get the full benefit from this treatment a fair degree of lung collapse is needed; extensive adhesions which prevent collapse are frequently found. Artificial pneumothorax may be tried when there is cavity formation. In severe hæmoptysis the results are striking. Regular refills of air are required; the lung must be kept collapsed, and the treatment prolonged on an average for three years or more. Work may be usually resumed early in the treatment—a decided economic advantage. The sputum

[1] "Auto-inoculation in Pulmonary Tuberculosis," by Marcus Paterson, 1911. The continual auto-inoculation induced by exercise is believed to set in motion the protective mechanisms of the body.

[2] Mouth temperatures should be six minutes; rectal, three; urinary half a minute.

commonly becomes free from bacilli after a few months and therefore the patient is no longer infectious. The drawback is that this method requires special training and constant control by X-rays, and must therefore be left to the expert.

Contra-indications.—In the earlier days of collapse therapy, active disease in the contra-lateral lung was regarded as a contra-indication to the induction of an A.P. The scope of this treatment is now recognised to be much wider, and in many cases bilateral A.P. has restored to active work a patient who would otherwise have gone steadily downhill. Complications such as severe cardiac failure or renal disease are definite contra-indications. Advanced tuberculous enteritis, with an accompanying general breakdown of resistance, usually renders a patient unsuitable for pneumothorax therapy, which should in such circumstances be discouraged. Tuberculous laryngitis, formerly regarded by many as a contra-indication, is now known often to be materially improved by A.P.

Technique.—Air or any other gas is slowly absorbed from the pleural cavity; hence the injections must be repeated at intervals of a week or more. The volume injected is the amount required to maintain the correct degree of collapse without causing discomfort to the patient. Only the regular use of the radiograph enables one to judge this. For further details of technique and control of this important method of treatment, special works must be consulted.

(*c*) *Thoracic Surgery.*—With the development and improvement of surgical technique in the last few years the scope of surgery in the treatment of pulmonary tuberculosis has greatly increased. In cases in which collapse of the diseased lung by artificial pneumothorax is impossible or inadequate, extrapleural thoracoplasty may effect the desired result, and in properly selected cases may save patients who would otherwise go downhill. Temporary diaphragmatic hemi-paralysis (phrenic crush) may relax the elastic tension in the lung sufficiently to heal a minimal lesion, and even occasionally obliterates a small thin-walled cavity. Further elevation of the diaphragm can be produced by introducing air into the peritoneal cavity (pneumoperitoneum). In a small proportion of cases this procedure may obviate the need for major surgery.

(*d*) *Symptomatic Treatment.*—It will be seen that in the advanced stages there is not much hope of recovery, but even in the worst cases we can ameliorate the symptoms, and so ease the passage to the grave. (1) For the cough, tinct. opii camphorata and expectorants are not of much use. The best cough mixture is one containing liquor morphinæ, or better still, codeine in small doses with dilute sulphuric acid. Warm alkaline drinks promote expectoration. (2) Night sweats, which are often very profuse and exhausting, may be combated by belladonna and zinc oxide, especially the first named. Night sweats are seldom troublesome if there be free exposure to fresh air. (3) The diarrhœa is also very exhausting, and must be combated with catechu, opium, intestinal disinfectants, and mineral acids. (4) Pleural pains may be eased by stupes, or painting with tincture of iodine. (5) The concurrent dyspepsia must be combated in the usual way, but the vomiting is often a very troublesome symptom, and there are three kinds of vomiting which admit of three different methods of treatment. (*a*) If preceded by nausea,

it points to disorder of the stomach, and should be treated by bismuth, etc., on the usual lines. (b) If the vomiting be preceded and caused by coughing, it is a good plan to give hot drinks just before a meal, in order to encourage expectoration and get the paroxysms of coughing over before the meal is begun. (c) If neither of these is successful, vomiting may sometimes be relieved by opium; sometimes it is controlled by the will. (6) The treatment of hæmoptysis, pneumothorax, and laryngeal ulceration is dealt with elsewhere.

(e) *Other measures not widely used.* *Tuberculin* is still tried by some for pulmonary disease. The evidence of its value is unconvincing. *Gold salts* were used experimentally by Koch; the modern forms are sanocrysin, myocrisin, crisalbine, etc. Some claim good results from their use; others, after extensive trial, believe that the improvement observed can be otherwise explained. All, however, agree that chemotherapy has yet a long way to go. Apart from these serious attempts to find a remedy, there are numbers of secret sera and proprietary nostra, widely advertised, which deceive the uncritical layman and waste money which would be better spent on good food and holidays. Streptomycin is still in the experimental stage and *sub judice.*

(f) *Preventive Treatment.*—The prevention of pulmonary tuberculosis is a wide and complex problem depending for its solution upon a proper appreciation of the principles which underlie our present conception of the pathogenesis of the disease. Preventive measures come under four main categories: (1) the public health organisation, (2) the part played by the general practitioner, (3) the education of the layman, and (4) the education of the medical student.

The public authorities have made good provision for the adult and in some cases for the child in the active stages of the disease, though more might be done in the way of observation of contacts and of suspects who are not actually notified. Much has been achieved in the attempt to educate the public as to prevention of the communication of the disease from man to man and of its extension from animals to man. The proper disposal of sputum from infected patients is of great importance, the patient being instructed to spit only into some portable receptacle containing a disinfectant such as lysol, or into paper sputum cups or handkerchiefs which can be burned. Phthisical patients should not share the sleeping rooms of healthy individuals.

Bovine tuberculosis is conveyed by ingestion of the milk or products of diseased cattle. In one research it has been shown that out of 2,825 cases in which the organism had been definitely typed, 1,040 (*i.e.*, 36·8 per cent.) were cases of pulmonary tuberculosis. Of these 2·3 per cent. were proved to be bovine in origin. Lange, in a series of 40 cases of pulmonary tuberculosis, found the bovine bacillus in the sputum in 20 per cent. In the light of modern work, it is important that raw cow's milk should be pasteurised before being used as a food for infants and young children. Adequate inspection of cattle is necessary, with authority to deal with

infected meat and milk. Good educative work has been done by the tuberculosis dispensaries, but more co-operation is desirable between the dispensaries, the sanatoria, the hospitals and the medical schools.

Mass radiography of the chest, carried out by experts, has been responsible for the detection of early pulmonary lesions in apparently healthy subjects; this method of examination should play a greater part in the machinery of preventive medicine in our national life. Thus the work of examination of contacts can be improved and effective steps taken in the early diagnosis of the disease and in the tracing and removal of the source of infection.

§ 132. **Chronic miliary tuberculosis** has recently been described. The clinical and radiological features of this are similar to those of the acute form described in § 117, but the course of the disease is much more protracted, extending in some cases even to two years. In some instances recovery has been recorded, radiographs showing complete resolution of the pulmonary lesions.

§ 133. **Fibroid Phthisis** is one of the chronic forms of pulmonary tuberculosis. It may be defined as a tuberculo-fibroid disease of the lungs, occurring for the most part in elderly subjects, running a protracted course, and terminating in contraction of the lung. This disease is very apt to be confused with chronic fibrosis of the lung (§ 136).

Symptoms.—The disease is essentially one of insidious onset and long duration. The patient complains of a chronic cough for many years. Later on this may become paroxysmal, and especially troublesome in the morning. Progressive shortness of breath, clubbed fingers, slowly increasing weakness and emaciation, with little or no fever, constitute the other symptoms.

The *Physical Signs* begin and are almost always most marked at the apex. *Both lungs* are usually affected (which contrasts with interstitial pneumonia), but the signs of disease are afterwards more advanced on one side. There is impairment of the chest movement and contraction of one side of the chest with signs of consolidation of the underlying lung. The heart, trachea, and other viscera are displaced to the more affected side. Hæmoptysis sometimes occurs, and the tubercle bacillus may be discovered on careful and repeated examination of the sputum or by guinea-pig inoculation.

The *Diagnosis* from other forms of *phthisis* is made by the extremely protracted course of this disease and the age of the patient. Non-tuberculous *pulmonary fibrosis* resembles it very closely, both in its physical signs and symptoms, and the diagnosis can only be inferred (i.) from the absence of the tubercle bacillus after oft-repeated examinations, (ii.) from the more usual localisation in one lung, (iii.) and from the history.

Etiology.—Fibroid phthisis is more frequently met with at and after middle life. It may follow chronic bronchitis, broncho-pneumonia, or repeated attacks of pleurisy. In true Fibroid Phthisis the tubercle bacillus is primarily deposited in a healthy lung under the same circumstances as in chronic pulmonary tuberculosis, and then causes an indolent fibroid reaction.

Prognosis.—Its course is very indefinite and protracted. Sometimes acute tuberculosis supervenes. The chief complications are bronchiectasis, compensatory emphysema of the lungs, lardaceous disease of other organs, and cardiac failure. In general terms the prognosis depends upon the same conditions as those of pulmonary tuberculosis and the *Treatment* is conducted on the same general principles.

II. *The patient complains of breathlessness; on examining the chest,* **dulness** *is found at one or both* **bases,** *and* SIGNS OF FLUID *are detected there. The disease is* HYDROTHORAX.

§ 134. **Hydrothorax** is a chronic collection of serous fluid in the pleural cavity, differing from the effusion of pleurisy in being non-inflammatory.

Symptoms.—The general symptoms may be but little marked if the fluid is small in quantity. The onset is usually gradual. Dyspnœa is generally present, especially on exercise, but its degree depends upon the amount of fluid. As hydrothorax is always a secondary condition, the symptoms may be masked by the presence of dropsy elsewhere, and it is remarkable how often hydrothorax is overlooked on this account. In rare cases the fluid collects with great rapidity. The sudden onset of signs of fluid in the chest, accompanied by shock or collapse, in a case which has previously presented the symptoms of aneurysm, points to the occurrence of hæmorrhage into the pleural cavity (hæmothorax). The *Physical Signs* are those of fluid in the chest (*vide* §§ 109 and 119).

Diagnosis.—The disease has to be diagnosed from other disorders giving rise to dulness on percussion (§ 113). As regards *pleurisy*, hydrothorax is distinguished by the absence of pyrexia at the onset, by the absence of pain, and by the fact that the fluid occurs usually on both sides.

Etiology.—(i.) Hydrothorax may form part of the *general* dropsy of subacute nephritis, in which circumstance both pleuræ are usually involved. Here the hydrothorax is of no very great importance *per se*, but the onset of dyspnœa in nephritis should always direct our attention to the pleuræ. (ii.) Similarly, it may form part of *cardiac* dropsy, in which circumstances one pleura (the right) is often solely or chiefly affected. (iii.) Malignant disease in the chest is frequently attended by hydrothorax. In this case the fluid is often blood-stained, and sometimes, as in pleural carcinoma, may be found to contain cancer cells. (iv.) Aneurysm or other intrathoracic tumours pressing on the veins of the thorax may give rise to hydrothorax on one or both sides. In this condition also the effusion may be blood-stained.

Prognosis.—The disease is essentially chronic, the duration depending very much upon the cause. In general terms the prognosis of the condition is unfavourable. The patient should be carefully watched for the occurrence of shivering, sweating, or intermitting pyrexia, as indicative of empyema.

Treatment.—Paracentesis (§ 119) should be performed when the amount of fluid is such as to cause symptoms : in congestive heart failure it is wise to undertake this early. Tapping may be repeated indefinitely. Diuretics or circulatory stimulants are useful. For the rest, the treatment must be directed to the primary condition (see also § 119).

III. *The patient complains of breathlessness ; on examining the chest,* **dulness,** *usually slight, is found at one or both* **bases,** *and on auscultation,* FINE CREPITATIONS *are heard. The disease is* PULMONARY CONGESTION, HYPOSTASIS OR ŒDEMA.

§ 135. **Hypostasis of the Lung** (Pulmonary Congestion or Œdema) is a serous exudation into and around the air vesicles. It is synonymous

with the term "hypostatic congestion," or, as it is sometimes called, "hypostatic pneumonia." It determines the end of many serious disorders.

Symptoms.—(i.) It is never a primary condition, and therefore our attention is first directed to the symptoms of its cause. The advent of hypostatic congestion is always insidious, and it is only by careful watching that it can be detected. (ii.) A considerable amount of dyspnœa is present, which may amount to orthopnœa. (iii.) There is a frothy mucous expectoration, not infrequently tinged with blood.

The *Physical Signs* are somewhat indefinite but they are found, as is implied by the term "hypostatic," chiefly at the bases of both lungs. The percussion note is somewhat impaired, and the air entry at the bases is diminished, and is attended by abundant moist crepitations.

Diagnosis.—The condition is diagnosed from true pneumonia by the gradual onset, the indefinite signs, and the absence, for the most part, of pyrexia, and other constitutional symptoms. Any rise of temperature that may be present is due to the primary or causal condition or to the development of broncho-pneumonia in the hypostatic areas.

Etiology.—(i.) The disease is most frequently met with in elderly people, especially when bed-ridden. (ii.) Pulmonary œdema complicates various blood disorders and fevers, especially typhus and typhoid fevers. In subacute nephritis œdema of the lungs occurs as part of a generalised dropsy. (iii.) Cardiac and other diseases, leading to mechanical dropsy, produce œdema of the lungs, sometimes acutely. (iv.) Tumours pressing on the veins within the mediastinum may result in pulmonary œdema. (For acute pulmonary œdema, see § 118.)

Prognosis.—The prognosis is always grave, because pulmonary œdema indicates either considerable impediment to the circulation in the lungs, or a serious toxic condition of the blood. It frequently terminates life in circulatory disorders, and in specific fevers of the asthenic type. In pneumonia it heralds a fatal issue.

Treatment.—The indications are to relieve the cause, if possible, and to stimulate the circulation. Ammonium carbonate and other stimulating expectorants promote expectoration. The liberal administration of alcohol and other diffusible stimulants is called for. The administration of 7% CO_2 with oxygen for ten minutes, two or three times a day, is valuable. In the aged, among whom even slight disorders are apt to be attended by pulmonary œdema, it is well *to keep the patient propped up* in a semi-recumbent posture. For the same reason it is advisable, in cases of fracture and other surgical maladies in the aged, to get them up as soon as possible, even at the risk of doing harm to their surgical ailment, so as to obviate the occurrence of hypostatic congestion of the lungs. The mercurial diuretics are of particular value in those cases due to myocardial weakness: when auricular fibrillation is present, digitalis is useful (§ 62). Should acute pulmonary œdema supervene, see § 118.

GROUP B.—We now turn to the chronic diseases attended by

dulness on percussion, which (*b*) does NOT always occur in regular and DEFINED AREAS AT BASE OR APEX. The *common* diseases in this group are: IV. PULMONARY FIBROSIS; V. THICKENED PLEURA; VI. and VII. NEOPLASMS.

§ **136.** IV. **Pulmonary Fibrosis** may be localised or diffuse, according to the variety, running a protracted course, and resulting in contraction of the pulmonary tissue. It may be associated with bronchiectasis (§ 143).

An increase of the fibrous tissue of the lung may take place under the following conditions, all being chronic processes: (i.) An indolent tuberculous process. Fibrosis is one of the ordinary terminations of a tuberculous focus; but when the progress is very slow and protracted, with excessive formation of fibrous tissue, it constitutes true *fibroid phthisis* (§ 133). (ii.) The constant inhalation of dust in certain trades (*e.g.*, fustian cutters, jute workers, wool sorters, stone, knife, and other grinders, and silica workers, notably gold miners, asbestos workers, etc.) gives rise to a gradually progressive fibrosis of the lungs (pneumokoniosis). In all the diseases of this group there is a slowly progressive change in the mucous membrane of the respiratory tract, leading eventually to a diffuse fibrosis throughout the lungs. The most characteristic feature of silicosis is the formation of fibrous nodules, recognisable at a certain stage of the disease in the X-ray picture (Fig. 48), which bears a certain resemblance to that of miliary tuberculosis. There is a tendency in these cases to widespread infection with tuberculosis, which causes corresponding modifications in the clinical and radiological phenomena. (iii.) Repeated attacks of *pleurisy* may be attended by a subpleural fibrosis (thickened pleura (§ 137)), and dense bands of fibrous tissue may extend into the lung. (iv.) *Acute broncho-pneumonia*, becoming chronic, may result in a pulmonary fibrosis. The commonest cause of fibrosis in young persons is broncho-pneumonia following measles or whooping-cough. (v.) *Syphilitic disease* of the lung is rare, except as a congenital manifestation in infancy, in which circumstances the change consists of a fibroid induration of the lung. Gummata also occur. Only (i.), which is a *tuberculo-fibroid* process, should be called "fibroid phthisis." The other varieties constitute cirrhosis of the lung, and if they are invaded by the tubercle bacillus, they form a *fibro-tuberculous* process, which in its later stages may be indistinguishable from fibroid phthisis. Silicosis in particular is often accompanied by tuberculosis. In such cases the prognosis is much worse than in fibroid phthisis. (vi.) Fibroid changes in the lung may occur as a late phenomenon in some cases of Boeck's sarcoidosis (§ 141*a*).

The general *Symptoms* consist of progressive weakness and dyspnœa. There is no fever unless there is infection—a common occurrence in bronchiectasis. The *Physical Signs* may be found either at the base or the apex, though usually the former. Except in the variety due to the inhalation of silica, only *one lung* is involved as a rule, thus differing from fibroid phthisis, in which both lungs are usually affected.

VII. **Secondary Malignant Disease of the Lung** occurs as numerous, scattered, more or less circumscribed nodules, and is always secondary to cancer of the breast or abdominal organs. Sarcoma of any part of the body may give rise to secondary deposits in the lungs.

Symptoms.—The most common symptoms are increasing dyspnœa, cough, and cyanosis. Much respiratory distress occurs when a large area of lung tissue has become involved. Miliary carcinomatosis, which is usually secondary to carcinoma of other organs, especially the stomach, causes the greatest dyspnœa and cyanosis (Assmann), apart from that due to stenosis of a large bronchus. The *physical signs* are often very indefinite. When a large surface of the pleura is involved, the first sign may be an effusion into the pleura; on exploration the fluid may be found to be hæmorrhagic.

(*b*) We now turn to the less common and the rare chronic diseases attended by **dulness on percussion,** not always in regular or DEFINED AREAS AT BASE OR APEX. These are: VIII. COLLAPSE OF THE LUNG; IX. HYDATID CYST; X. SYPHILIS OF THE LUNG; XI. Some of the DISEASES DUE TO FUNGI AND PARASITES.

§ **139.** VIII. **Collapse of the Lung,** or **Atelectasis,** is a condition in which the lung tissue is in an unexpanded state. The term "atelectasis" is usually applied to lung tissue which has never properly expanded, a congenital condition, due to imperfect development. The term "collapse of the lung" is applied to lung tissue which has previously expanded, but in which the air vesicles have subsequently collapsed.

Atelectasis is a *congenital* condition. The *symptoms* occurring in the new-born child consist of cyanosis, with shallow, rapid respiration. The lower part of the chest is drawn in by each respiration. On auscultation, the respiratory murmur is found to be very faint.

The *Symptoms* of **collapse of the lung** follow and complicate those of the disease which has led to the condition; for instance, the patient may not recover so rapidly as he ought, or the breathing is more embarrassed than can be accounted for by the concurrent disease in the chest. The physical signs vary considerably with the degree of collapse. Thus:

(*a*) In *complete* collapse of a part of the lung, as, for instance, in collapse due to compression or complete obstruction of a bronchus high up, there is impairment of the percussion note, with a diminution or absence of the breath sounds and of the vocal resonance and fremitus.

(*b*) Where the collapse is only *partial* in degree—*e.g.*, where the bronchi remain patent, as occurs sometimes when the lung is compressed by pleural or pericardial effusion—there are signs resembling those of consolidation (§ 109), except that the percussion dulness is not so marked, and the breath sounds, though bronchial in character, are somewhat feeble.

(*c*) Where the collapse is *slight* and limited, the chief sign is an enfeebled respiratory murmur. During deep inspiration fine rustling crepitations are heard, due to the expansion of the collapsed vesicles.

The *Diagnosis* is made usually by the existence of a causal condition.

When this is detected, attention may then be directed to the physical signs in the lungs. It will be observed that the signs of partial collapse resemble the signs of consolidation, and those due to slight collapse resemble early pneumonia. If the collapse is extensive the heart and mediastinum are drawn over to the side of the lesion. In addition to the evidence supplied by physical examination is that given by radiography, the airless portion of the lung appearing as a more or less homogeneous opacity. In cases of localised collapse there may be little or no evidence beyond that afforded by X-ray examination. Now that routine X-ray examination has become a matter of practice, localised pulmonary collapse, without symptoms, appears to be far more frequent than was originally supposed. It is particularly common in children, despite the absence of obvious clinical manifestations of disease.

Etiology.—The causes are of four kinds: (*a*) Causes which produce *obstruction*, such as (i.) a tumour at the root of the lung (*e.g.*, aneurysm); (ii.) obstruction in the throat (*e.g.*, the larynx); (iii.) stricture of a bronchus (*e.g.*, new growth or gumma); (iv.) secretion obstructing the bronchi; (v.) foreign bodies obstructing the larynx or bronchus. (*b*) *Compression* of the lung may be produced by pleural or pericardial effusion, an enlarged heart, or tumours of the mediastinum or of the abdomen. The condition may be the result of gross spinal curvature. It may occur after abdominal operations and anæsthesia (*acute post-operative collapse*), and give rise to difficulty in diagnosis, unless the possibility of its occurrence is borne in mind (§ 125). (*c*) *Paralysis* of the intercostal muscles or diaphragm, as in diphtheria or other cause of neuritis. (*d*) *Injury* to the chest wall with or without involvement of the thoracic contents, especially by high velocity projectiles, is a fertile cause of collapse of lung either on the same or the opposite side.

In *adults* collapse is most often met with as the result of pleural effusion or tumours in the chest; in *children*, as the result of bronchitis or slight catarrhal affections of the respiratory tract.

Prognosis.—The course of the disease depends very much upon the cause. Recovery as a rule soon takes place after compression by effusion, obstruction or stricture of the bronchi, and throat affections.

The *Treatment* is unsatisfactory. It should be directed to the removal of the cause, and especially to the recovery of any concurrent pulmonary disorder. That form which yields best to treatment is met in children with bronchitis. In adults, respiratory exercises should be given.

§ 140. IX. **Hydatid** of the lung and pleura is much more common in the Argentine and Australia than in this country. Generally cysts are single and tend to involve the base of the lung, especially on the right side. Clinically they remain latent in 75 per cent. of cases till the supervention of some complication like hæmoptysis, rupture or suppuration. A history of the expectoration of "grape skins" is pathognomonic; cough is frequent. The physical signs resemble pleural effusion, but the area of dulness is localised and has a rounded outline. Apical hydatid simulates tuberculosis, but the pulse is slower and fever absent or less marked.

The *Diagnosis* of deep cysts is often impossible until X-ray reveals the characteristic spherical shadow surrounded by translucent lung tissue. Eosinophilia is

generally absent, but the intradermal skin test is almost invariably positive, the complement fixation and precipitin tests less frequently so. The sputum may contain hydatid elements such as hooklets, scolices or laminated membrane.

The *Prognosis* largely depends on the presence or absence of complications. Many cases undergo spontaneous cure by rupture into a bronchus; others may be drowned in the process. Rupture into the pulmonary artery or heart is fatal. Secondary infection may lead to pulmonary abscess or empyema. Cysts of the liver frequently coexist (§ 347).

Treatment is surgical: resection of the adjacent ribs, incision and evacuation of the cyst content with or without drainage. Aspiration is dangerous owing to the danger of flooding the bronchial tree.

§ 141. X. yphilis of the Lung.—In infants this disease may take one of two forms: (*a*) The pneumonic condition of lung, found in infants, usually still-born, is regarded as an interstitial pneumonia of syphilitic origin. (*b*) Gummata are occasionally met with in the lungs of infants who are the subjects of hereditary syphilis; still more rarely they are met with in adults. Dyspnœa is usually the only symptom. The signs are those of consolidation and collapse. In adults syphilis of the lungs may take other forms—*e.g.*, broncho-pneumonia, bronchiectasis, etc.—and may lead to extensive infiltration and breaking down, or to fibrosis. Pulmonary syphilis is rare enough to be a curiosity. For mediastinal gummata see § 81. V.

§ **141a.** XI. **Sarcoidosis** (Besnier-Boeck-Schauman Disease).—Much attention has been directed in recent years to the pulmonary manifestations of this generalised disease which may involve skin (§ 647), mucous membranes of nose or throat, parotid and lachrymal glands (§ 9), tonsils, uveal tract, lymphatic glands, lungs, bones, and internal viscera such as the spleen and alimentary tract.

IN THE CHEST the most constant feature is enlargement of the hilar glands. In addition, there is a general reticulosis which may progress to an interstitial fibrosis of the lungs with a tendency to nodulation. Occasionally, diffuse and confluent parenchymatous infiltrations are seen throughout the lung-fields.

Symptoms.—Patients may be symptomless, the condition being discovered by routine X-ray examination. The most frequent symptom is dyspnœa: there may be slight constitutional disturbance, *e.g.*, pyrexia, lassitude, and anorexia, with occasional cough. If the condition progresses to an extensive fibrosis, dyspnœa is likely to become more severe, and the patient may even be cyanosed. The radiological picture is that of a diffuse reticulosis with or without interstitial fibroid changes, and with marked enlargement of the hilar glands.

Etiology.—Typical sarcoid lesions consist of collections of pale staining epitheloid cells among which giant-cells occur. The main distinction between these lesions and tubercles is the absence of caseation and of tubercle bacilli. The three chief hypotheses as to the pathogenesis of these lesions are that they are (*a*) due to tuberculosis of an atypical and anergic form, since the Mantoux reaction is negative; (*b*) a non-specific tissue response called forth by a variety of pathogenic organisms such as the tubercle and lepra bacilli; (*c*) a manifestation of a systematised disease of the reticulo-endothelial system, similar to lymphadenoma and of unknown etiology.

The diagnosis is from miliary tuberculosis, silicosis and other forms of industrial lung disease, and secondary carcinomatosis. In an X-ray the enlargement of the hilar glands is the most constant and reliable feature. Schaumann noted a relative monocytosis: eosinophilia has been reported by others: an increase in the serum globulin has also been noted. The diagnosis has been established in some cases by liver biopsy.

Prognosis.—Usually this is favourable, the disease pursuing a chronic course and tending to recovery. Death has been recorded from heart failure supervening on progressive pulmonary fibrosis: in some cases, there is a terminal development of active tuberculosis.

Treatment.—The disease is uninfluenced by any treatment.

XII. Diseases due to Fungi and Parasites are recognised on examination of the sputum, and are referred to in §§ 145 and 146.

Group C.—Chronic Diseases attended by **Hyper-resonance** on percussion: I. In quite nine out of ten cases of hyper-resonance it exists on both sides, and is due to Emphysema. Other conditions which give rise to it are: II. Pneumothorax (§ 126); III. Skodaic Resonance above the level of an effusion (§ 108 and Fig. 49). The diagnosis is given in the form of a table (Table VIII).

Table VIII.—Causes of Hyper-resonance.

Cause.	Hyper-resonance.	Auscultation.	Other Diagnostic Features.
I. **Emphysema.**	Bilateral and universal.	R.M. distinct but weak and expn. much prolonged; signs of bronchitis, if present.	Barrel-shaped chest, cardiac dulness obscured.
II. **Pneumothorax.** An acute condition.	Hyper-resonance always unilateral, though it may extend beyond middle line.	Absence of R.M. and V.F. over affected area; sometimes amphoric breathing. Bell sound.	Organs displaced.
III. **Skodaic Resonance**—*i.e.*, the high-pitched note above a large *pleuritic effusion*, when the lung is otherwise healthy.	Unilateral; level may shift with position of patient.	Loud R.M.; V.F. felt over affected area.	History of pleurisy; signs of fluid in lower part of chest.

I. *The patient has complained of breathlessness for some years. There is* **hyper-resonance** *on* **both** *sides of the chest. The disease is* Emphysema.

§ 142. **Emphysema** is a chronic non-febrile disease of the lungs in which the air vesicles become hyper-distended, the walls separating each vesicle become atrophied, inelastic and ruptured. As a result the aërating surface is greatly diminished, and the lungs are deficient in their elastic recoil.

Symptoms.—(1) The onset of the disease is imperceptible, and generally supervenes gradually after repeated attacks of bronchitis, the patient becoming more and more breathless after each attack. (2) This breathlessness is practically the only symptom, and it differs from all other kinds of breathlessness in this, that the chest remains *permanently in the inspiratory position*—in other words, owing to the inelastic state of the lungs and the shape of the chest, the patient finds it more difficult to expire than to inspire. A certain degree of cyanosis is usual. (3) Symptoms of bronchitis are *nearly always present.*

The *Physical Signs*, expressed *briefly*, are a barrel-shaped chest, hyper-resonance, and prolonged expiration. The shape of the chest is special to emphysema (*cf.* § 106).[1] The chest assumes permanently the shape of

[1] According to Cabot (*Physical Diagnosis*, 10th Ed., p. 309, London, 1930, Baillière, Tindall and Cox), the condition of "Barrel-chest," though characterised by a definite clinical syndrome, hereinafter described, is not necessarily associated with macroscopic emphysema, and may be due to abnormal conditions of the chest wall.

a healthy chest in a position of deep inspiration. The antero-posterior diameter is considerably increased. The hyper-resonance is always bilateral, and it obscures the dulness of the neighbouring organs—namely, the heart, the liver, and the spleen. These last two organs are also displaced downwards. The apex beat may not be palpable, but epigastric pulsation is usually felt. On auscultation, the respiratory murmur is modified; the inspiratory sound, which is full, is followed by a pause, and then by a prolonged expiratory sound. There are no adventitious sounds proper to emphysema, but, as just mentioned, bronchitis (*q.v.*) nearly always accompanies it. The heart sounds, especially at the base, may not be heard, or only with difficulty. Well-established emphysema interferes considerably with the pulmonary circulation, on account of the ruptured alveoli, and consequently the right side of the heart in course of time becomes dilated.

Variety.—In old people there is sometimes hyper-resonance with weak breath sounds, but no enlarged barrel chest; this is called *Atrophic* Emphysema, and is due to the giving way of degenerate air vesicles.

Etiology.—(i.) The condition occurs usually in elderly subjects. Both sexes are affected, but it is much commoner in males owing to the prevalence of bronchitis and asthma in them. (ii.) Heredity is said to play no part in the disease, but undoubtedly a hereditary tendency can frequently be traced. (iii.) The disease is frequently associated with senile degeneration, chronic nephritis, and cardio-vascular changes. (iv.) Bronchitis is the most frequent of the exciting causes, owing to the prolonged coughing and straining to get up phlegm, and owing also to the blocking of certain tubes with thickened secretion, which prevents the access of air to some alveoli, and unduly distends others. (v.) Asthma is also a potent exciting cause, owing to the constant strain on the elastic tissue of the lungs.

Prognosis.—Patients may live with emphysema to a good old age, and provided it is only moderate in degree it does not necessarily shorten life, though it predisposes to, and adds to, the seriousness of other pulmonary disorders. The gravity of any particular case is best measured by the extent of cardiac involvement (*q.v.*).

Treatment.—The indications are: (i.) To relieve the accompanying bronchitis (see § 129); (ii.) to improve the cardiac condition. The diet is of considerable importance in advanced cases, for any distension of the stomach greatly adds to the respiratory distress. It is a good rule never to let patients take a solid meal later than two o'clock in the day; otherwise their nights become considerably disturbed by the breathlessness. Ephedrine in repeated small doses (*e.g.*, gr. ½) often relieves the respiratory distress.

GROUP D.—There are three chronic pulmonary conditions in which the percussion note varies considerably in different cases, but the **character of the sputum** suggests their presence—viz.: I. BRONCHIECTASIS AND FŒTID BRONCHITIS; II. GANGRENE AND ABSCESS OF THE LUNG

(*vide* § 144). In Abscess the sputum is not invariably offensive. III. ACTINOMYCOSIS and other diseases due to fungi and parasites affecting the lung can usually be diagnosed only by examination of the sputum.

§ **143. I. Bronchiectasis** is a cylindrical or saccular dilatation of the bronchial tubes. In order clearly to understand the significance of this it must be remembered that bronchiectatic dilatation is a pathological process, usually accompanying or resulting from some other morbid condition, and not strictly speaking a disease *sui generis* (see § 136).

Pathogenesis.—Traditional teaching has hitherto attributed the development of bronchiectasis to two main factors—(1) weakening of the bronchial wall by infective processes, with consequent loss of elasticity, and (2) increased intra-bronchial pressure due to excessive coughing. This view is still held by most teachers, but of late years increasing attention has been given to the association between bronchiectasis and pulmonary collapse, and many cases of so-called atelectatic bronchiectasis have been demonstrated in which bronchography has shown the presence of dilated bronchi within a collapsed lobe. So frequent is this association that atelectasis is now admitted to be one of the factors concerned in the pathogenesis of bronchiectasis. According to certain recent observations, partly clinical, partly experimental, collapse of the lung is regarded as an invariable antecedent of bronchial dilatation, the traditional theory of infection as a primary cause being considered no longer tenable.

According to this view, which is not yet generally accepted, bronchiectasis, cylindrical or saccular, is in itself a symptomless condition, only demonstrable by bronchography. If and when the factor of infection supervenes, symptoms arise, and eventually there appears the typical clinical picture, familiar in text-books. The following description applies to cases in which infection of the respiratory tract has occurred : we have as yet no accurate knowledge of the incidence of infection in pure atelectatic bronchiectasis.

Symptoms.—Although bronchiectasis may exist for long without sputum formation, sooner or later this occurs ; in characteristic cases there are bouts of coughing at intervals varying from a few days to a few hours, accompanied by expectoration of a large quantity of sputum. This is purulent owing to secondary infection, and sooner or later may become extremely fœtid and offensive owing to stagnation and to the presence of saprophytic and/or anærobic organisms. The cough and expectoration are often started by some change of posture, and are therefore frequent when the patient wakens in the morning. In early cases, even with little sputum, the characteristic odour of the breath may be detected, but this may be absent. In advanced cases the total amoun of sputum expectorated during 24 hours may be considerable (up to 20 ounces or more). In such circumstances the fœtor is usually pronounced, but this depends on the bacteriological factors present. Constitutional disturbance is frequent, and in bad cases there may be considerable pyrexia, sweating and loss of flesh. Often the sputum is found on standing to have become divided into three more or less distinct layers, the upper one frothy and muco-purulent, the middle more fluid, and the lower one chiefly pus, in which may be found " Dittrich's plugs," which consist of the debris of leucocytes, fat, and epithelial cells ; crystals of fatty acids and numerous organisms may also be present. Hæmoptysis,

occasionally an early symptom, may occur at any stage, and in advanced cases may be considerable, often causing a diagnosis of phthisis.

Dry (hæmorrhagic) bronchiectasis (Syn.: Silent Bronchiectasis). In this form of the disease, which is now recognised as a clinical entity, infection has not occurred, and consequently the clinical picture above described does not appear. The condition is characterised by hæmoptysis of varying severity occurring at intervals; between these attacks the patient exhibits neither symptoms nor abnormal physical signs. This variety of bronchiectasis, which is one of the possible causes of sudden hæmoptysis, is only demonstrable by bronchography.

Polycystic disease of the lung (Syn.: Congenital Cystic Disease of the Lung), resembles bronchiectasis or, rather, bronchiolectasis, with which it has been identified by some authors, though others regard it as etiologically distinct. The chief *symptoms* are cough, shortness of breath and recurrent hæmoptysis. The X-ray appearances are characteristic and give rise to the so-called "soap-bubble" lung.

Solitary cysts of the lung are frequently recognised on X-ray examination. Usually clinical symptoms are absent, but hæmorrhage and infection may arise in the cysts.

Physical Signs.—These necessarily vary according to the degree of structural damage. When sufficient to cause definite physical signs, we meet chiefly those of fibrosis and retraction of the lung, with or without cavitation; co-existent are the signs of accompanying bronchitis. Cyanosis is common. Clubbing of the fingers is almost always present, and varies in extent according to the degree of infection and toxæmia.

Diagnosis.—Chronic cough, aggravated by movement or change of posture, and accompanied by the typical offensive sputum, is usually sufficient to distinguish the presence of bronchiectasis, especially when accompanied by the physical signs of fibrosis and excavation. In earlier cases, where there is little structural damage to the lung tissue, and little infection, the diagnosis may only be established by radiology; without the aid of iodised oil (*vide* Fig. 44), even an X-ray film may fail to show appearances which are pathognomonic. It is sometimes difficult to distinguish bronchiectasis from abscess of the lung, especially as the two conditions are frequently associated and are often part of one pathological process (see § 144).

Etiology.—Fibrosis and bronchiectasis are often said to occur after the broncho-pneumonia of measles and whooping-cough. There is good reason to believe, in the light of modern observations, that previous illnesses which have been hitherto regarded as attacks of pneumonia have in reality been attacks of massive collapse of the lung. Where there have been several alleged pneumonias, it is likely that a condition of atelectatic bronchiectasis has occurred upon which periodic infections of the respiratory tract have supervened. A foreign body or neoplasm, a tumour, aneurysm, or syphilitic stricture may cause blockage of a bronchus, with consequent pulmonary collapse, and bronchiectasis. In these cases infection frequently follows with the clinical picture already described.

Prognosis.—The condition is a very serious one, and is often incurable. The patient may live for many years. The prognosis is much worse

in bilateral cases, and in cases associated with extensive disease of the lungs or pleura.

The *Complications* which may occur are fatal hæmorrhage, gangrene of the lung, broncho-pneumonia, cerebral abscess, and pyæmia.

Treatment.—Until a few years ago the treatment of bronchiectasis was purely medical, the early attempts at radical surgery being attended by a very high mortality. The development of thoracic surgery in the last few years has resulted in such improvement in operative technique as to make lobectomy the treatment of choice in properly selected cases, *i.e.*, where the disease is unilateral and localised in one lobe. For extensive bronchiectasis involving more than one lobe of a lung, total pneumonectomy has given satisfactory results. Thoracoplasty is a possible alternative to pneumonectomy, and is a less severe procedure, but it does not offer so good a prospect of radical cure. Postural drainage and bronchoscopic aspiration are important pre-operative measures when toxæmia is a marked feature ; in any case a preliminary period of rest for a few weeks is desirable before a major operation is undertaken. When operative measures are contra-indicated, or when the patient will not submit to surgery, medical treatment can relieve the distress occasioned by large quantities of offensive sputum, and can build up the general health. Penicillin, administered by the inhalation method or by subcutaneous injection, may be of real help in combating toxæmia due to penicillin-sensitive organisms. The usual stimulant expectorants may be given at intervals to assist expectoration, and postural drainage should be carried out systematically. Liberal inhalations of turpentine, coal tar or creosote should be given on account of their antiseptic and deodorant effects. The most valuable form of treatment is the creosote vapour bath. Patients (whose eyes are protected by closely fitting goggles) are placed in a more or less air-tight room in which crude creosote is volatilised by placing it in a shallow iron dish which is supported on a stout ring and heated by a Bunsen burner. Terebene or creosote (refined) may be given by mouth in capsules (3 to 5 minims of the oil) three times a day. A sedative cough linctus may have to be given if sleep is much disturbed by cough. General tonics such as arsenic, nux vomica, cod-liver oil, etc., are indicated for cases with debility, anorexia, etc., and open-air treatment should be carried out when possible.

§ **144.** II. **Abscess** and **Gangrene of the Lung.** *Symptoms.*—The clinical history is often indefinite, though (i.) cough is usually an early and prominent symptom. (ii.) The sudden expectoration of a considerable quantity of offensive sputum, followed by a sudden drop in the temperature of a patient previously febrile, suggests the presence of an abscess which has discharged into the bronchus. (iii.) Pain is a variable symptom ; it is seldom severe. (iv.) Clubbing of the fingers develops rapidly in many cases.

The *physical signs* are usually those of consolidation, but vary according to the stage of the disease and the extent to which the abscess has drained by rupturing into the bronchus. There is usually little or no displacement of the heart and mediastinum.

X-ray findings vary. In the earlier stages, before there is extensive breaking down of the lung, the skiagram may show only a dense homogeneous opacity. If a definite cavity has formed, it may be possible to see a fluid level, which remains horizontal in spite of changes in the position of the patient. This phenomenon is practically diagnostic of abscess.

Etiology.—Between these two forms of intrathoracic suppuration it is hardly possible to draw a sharp line of distinction; both result from invasion of the lung tissue by pathogenic organisms, and the consequent damage varies in character and extent according to (*a*) the nature of the invading organisms and (*b*) the virulence-resistance ratio of the individual. Another factor which determines the character of the inflammatory changes in the lung is the blood supply of the particular part affected. In the case of rapid and extensive necrosis of the lung parenchyma, for which the term gangrene should really be reserved, the effects of an unusually virulent toxin are probably aided by the vascular occlusion which accompanies the process. A gangrenous necrosis frequently precedes abscess formation; on the other hand, a lung abscess may arise insidiously, without evidence of previous acute inflammation. For practical purposes it is convenient to consider abscess and gangrene of the lung under one heading.

The distribution of lung abscess may be single or multiple. Single abscesses are more often found in the right lung than in the left, and in the lower more often than in the upper lobe. If abscess formation in the lung is a result of embolism following septic infection in other parts of the body, the lesions are likely to be multiple. Two main causes of intrapulmonary suppuration (abscess and gangrene) are: (i.) aspiration of infective material into the lower respiratory tract; (ii.) embolism. There are two schools of thought, each favouring one of the above causes; probably both play a part. The development of a lung abscess often follows operations upon the mouth, nose and upper respiratory tract, when it is probably due to inhalation of blood, mucus, or portions of soft tissue into the lower respiratory passages. (iii.) In other cases abscess formation follows the occurrence of a pulmonary infarct. Sometimes it complicates pneumonia. Often there is no history of previous acute illness; the patient comes with an obvious abscess of the lung, for which no definite cause can be found, the symptoms having gradually and insidiously increased for many weeks. (iv.) An underlying new growth is a possible cause. (v.) A liver abscess (especially the " tropical abscess "), or a suppurating hydatid may rupture into the lung. Any of the well-known pathogenic organisms may be present; streptococci, staphylococci, pneumococci, and various saprophytic organisms. Anærobic organisms also should always be sought for, especially in cases of acute and rapid gangrenous necrosis of the lung.

The *Prognosis* is serious, but with early diagnosis and skilled surgical intervention the outlook is by no means hopeless.

Treatment. As above mentioned, an abscess frequently ruptures into

a bronchus, leading to spontaneous cure ; if there is free drainage of pus in this way, there is seldom immediate need for surgical intervention. If, however, drainage is inadequate, or if no pus has been coughed up, and the patient still shows evidence of toxæmia, surgical drainage by a two stage operation must be instituted. Artificial pneumothorax has been advocated, but carries with it a grave risk of rupture of the abscess into the pleural cavity ; where drainage through a bronchus is in progress it might conceivably be useful. In practice the actual results of pneumothorax treatment of lung abscess have been almost always unsatisfactory, and frequently disastrous. As in other infections of the respiratory organs, the value of penicillin in addition to any necessary surgical measures is becoming increasingly apparent.

§ 145. III. **Actinomycosis** may affect the pleura or the lung, imitating the signs of empyema, pneumonia (§ 121), phthisis, or bronchiectasis. In the absence of cutaneous or other lesions it is rarely diagnosed except by an examination of the sputum, when the little yellow pellets containing the ray fungus are visible. The streptothrix may by cultured anærobically. The disease is usually fatal.

§ 146. IV. **Paragonimiasis**, caused by *Paragonimus westermani*, the common lung fluke of Japan and China, gives rise to pulmonary symptoms, including cough and hæmoptysis. The physical signs may suggest bronchiectasis, broncho-pneumonia or pleurisy, the diagnosis being made by finding operculated eggs in the rusty-brown sputum. Diarrhœa and Jacksonian epilepsy due to involvement of the intestine and brain respectively may occur. No specific drug treatment is known.

ASPERGILLOSIS. The fungus *Aspergillus fumigatus* may cause signs resembling tuberculosis. The disease affects pigeon-feeders, who chew the seeds containing the fungus. It may undergo spontaneous resolution.

BLASTOMYCOSIS and SPOROTRICHOSIS may affect the lungs. Cutaneous and other lesions are usually present in addition (§ 648). ASCARIS infections may be hard to diagnose in early stages.

PSITTACOSIS, ANTHRAX, PLAGUE, GLANDERS and DISTOMA may affect the lungs, and can be recognised only by the sputum and concurrent signs. Psittacosis (§ 506) often resembles pneumonia.

Various OTHER FUNGI have been identified in association with broncho-pulmonary inflammations, *e.g., monilia, geotrichum, coccidioides,* etc. These conditions are mostly seen in Eastern countries. The presence of *mites* has also been identified with asthmatic and other respiratory symptoms.

CHAPTER VII

THE UPPER RESPIRATORY PASSAGES AND THE THYROID GLAND

THE throat may be the seat of the same morbid processes as affect other mucous structures, such as catarrh, ulceration, or new growths. Moreover, in the throat several important constitutional diseases, such as diphtheria, scarlet fever and syphilis, have important local manifestations. These facts have long been known, but it is now recognised that the throat, and especially the tonsils—organs whose functions are still imperfectly known—may constitute the portal of entry of certain microbic conditions. It is probable that the organisms causing influenza, rheumatism, malignant endocarditis, and other infective conditions, may thus enter the general systemic circulation.

This chapter deals with the symptoms referable to the **pharynx** (§ 151), the **larynx** (§ 164), the **nasal cavities** (§ 178), and the **thyroid gland** (§ 184).

THE THROAT

§ 151. **Symptomatology.**—" The throat " may be said to consist of the fauces, tonsils, palate, pharynx, and larynx, and we are here concerned with the investigation of these structures. The symptoms indicating disease of these parts are principally two—namely, SORE THROAT and HOARSENESS. The examination of the mouth and tongue is described under Disorders of Digestive Tract (Chapter VIII).

(*a*) SORE THROAT is indicative mainly of disease of the *pharynx*, tonsils, and adjacent structures. If the patient complains of " sore throat," turn to § 153.

(*b*) HOARSENESS AND OTHER ALTERATIONS OF THE VOICE are indicative of some affection of the *larynx* (§ 164). If NASAL INTONATION or NASAL DISCHARGE be present, turn to § 178.

There are also several minor symptoms which arise in conjunction with these, such as a dryness accompanied by tickling sensations, or an excessive secretion, which leads to " hawking " and " coughing." Thus it happens that we may be consulted for what the patient believes to be pulmonary disease, when in reality the lungs are perfectly healthy. Dyspnœa and dysphagia may also be produced by local conditions of the throat and larynx. " Globus," a paroxysmal sensation as of a ball in, or constriction of, the throat is a symptom of hysteria.

§ 152. **Clinical Investigation.**—The anatomy and relations of the throat are indicated in Fig. 51; the various parts may be investigated by (*a*) direct, and (*b*) indirect (*i.e.*, laryngoscopic) examination.

(*a*) For the DIRECT EXAMINATION of the fauces and neighbouring

structures all that is necessary is a good light and a spatula or spoon to depress the tongue. If direct light is not available—as for instance, when the patient is in bed—a head mirror can be used (*vide infra*). The patient should be instructed *not to strain*, and to "*breathe quietly in and out.*" The posterior wall may be seen by directing the patient to say "Ha—ah," by which procedure the soft palate is raised. Note should be made of the colour of the mucous membrane, the presence of exudation or ulceration, of granulations in the pharynx, of mucous patches (syphilis), bulging of the pharyngeal walls; also of paralysis or weakness of the tongue, palate

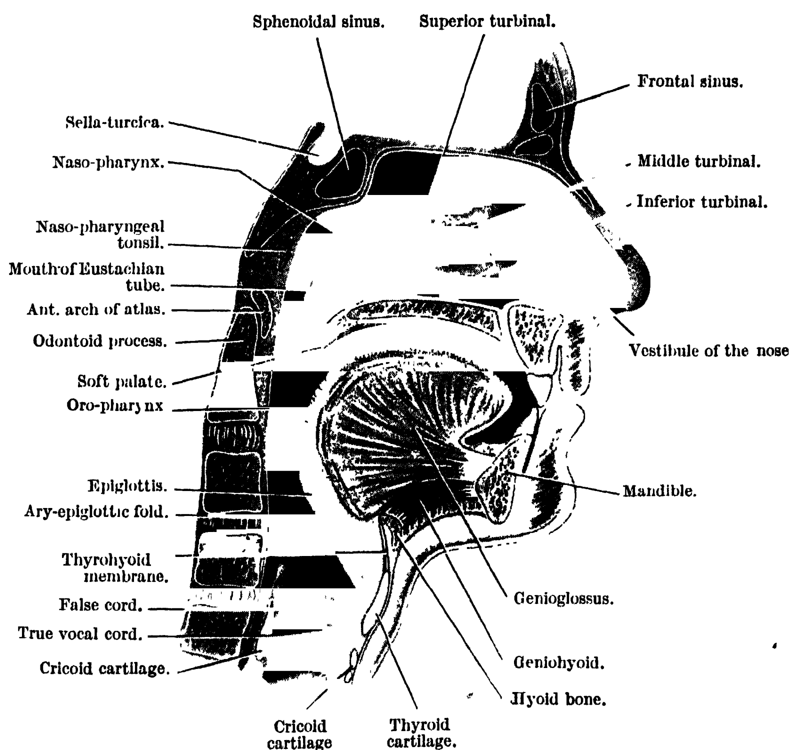


FIG. 51.—ANATOMY OF MOUTH, NOSE AND THROAT.

or pharynx. The size and length of the uvula should be observed; a long uvula may be the cause of a chronic cough and of symptoms such as the sensation of a foreign body and constant hemming and hawking. When a patient complains of cough coming on, or getting worse at night or when he lies down, *elongated uvula* should be suspected. It does not follow that such a uvula may appear too long at the time of inspection. The symptoms frequently ascribed to an elongated or "relaxed" uvula are often due to pharyngeal or post-nasal catarrh which, indeed, may be the cause of the elongation. Temporary congestion from various causes,

e.g., much talking, produces undue elongation and nocturnal cough. Treatment should be directed to any catarrh or sepsis in the upper air-passages, with, locally, the use of astringent paints and lozenges. When conservative treatment has failed, part of the uvula may be amputated, but this is rarely done nowadays.

(*b*) The INDIRECT OR LARYNGOSCOPIC EXAMINATION of the throat is described in § 164.

§ 153. Classification, Diagnosis, Prognosis, and Treatment.—Sore Throat is a symptom common to nearly all diseases of the throat. Mentioned in order of frequency, the diseases which give rise to sore throat are as follows (*laryngeal affections being excluded for the present;* see § 164):

TABLE IX.

Commoner Causes.	*Rarer Causes.*
I. Pharyngitis, including several acute and chronic varieties.	VI. Retro-pharyngeal abscess.
II. Tonsillitis (acute parenchymatous, acute follicular, quinsy, Vincent's angina, and more rarely agranulocytic angina, glandular fever and acute leukæmia). Chronic tonsillitis.	VII. Phlegmonous sore throat and acute œdema.
III. Scarlet fever.	VIII. Cancer, and other new growths.
IV. Diphtheria.	IX. Tuberculosis.
V. Syphilis.	X. Other acute specific fevers.

§ 154. I. Acute Pharyngitis is an inflammation of the mucous membrane of the pharynx and soft palate, and to a certain extent of the tonsils also. It may be so mild as to cause only slight discomfort in swallowing, dryness of the throat, tickling and hawking, and in such mild cases there is only a moderate congestion of the parts. But in more severe cases constitutional symptoms are more pronounced, and locally there may be œdema and marked congestion. The temperature in such cases varies from 100° to 104° F. The disease rarely lasts more than a few days, ending generally in resolution, although sometimes it passes into a chronic condition.

(*a*) **Chronic Catarrhal Pharyngitis** presents the same symptoms as the acute variety, in a milder degree, and extending over a longer period of time. It is often known as Relaxed or Relapsing Sore Throat, on account of the chronic congestion of the parts and the consequent predisposition to the repeated occurrence of subacute attacks. It forms one variety of clergyman's or school-teacher's sore throat.

(*b*) **Granular (Follicular) Pharyngitis** is a *chronic* condition, the local symptoms of which resemble the foregoing, with the addition of visible granulations on the pharyngeal walls due to the grouping of masses of lymphoid cells round the openings of the ducts of the mucous glands. The ducts may become obstructed and dilated, and later discharge yellow cheesy material, when the name **follicular pharyngitis** is given to the

condition. Anyone subject to this common condition, although apparently in good health, is liable to repeated attacks of sore throat whenever the weather is damp or his health a little below par. There is excessive mucous secretion, which collects in the throat, especially in the morning, and leads to chronic cough and hawking. When the disease has lasted some time, the throat becomes dry from atrophy of the glands (Pharyngitis sicca). With all forms of pharyngitis it is essential to make sure that there is no dental or nasal infection keeping up the condition.

(c) **Adenoids** may be regarded as a form of chronic pharyngitis limited to the naso-pharynx, but often associated with lymphoid granulations in the oro-pharynx. The lymphoid granulations may fill a large part of the naso-pharynx, occurring as a large grooved cushion or pedunculated growth, which, on examination, can be seen and felt behind the soft palate. This condition is common in childhood. The child *breathes with the mouth open*, and thus acquires a characteristic vacancy of expression. The intellect is often below the average. The voice has a dull or nasal twang, and there are snoring and disturbed sleep. The nares are narrowed, and the palate may be high from the negative pressure in the nose, the diminished air tension in the nose not counterbalancing the normal air tension on the buccal aspect of the hard palate. Pigeon-breast may follow. The condition is a pregnant cause of middle-ear catarrh and subsequent deafness. Adenoids in the naso-pharynx are usually accompanied by chronic enlargement of the tonsils. The disease often runs in families.

The *Causes of pharyngitis* vary somewhat in the different forms, although the several causes are largely interchangeable. (1) In certain persons exposure to cold and damp is immediately followed by an attack of pharyngitis, but this probably acts only as a predisposing cause. (2) Unhygienic surroundings, such as crowded conditions, bad ventilation, and work in the presence of dust or irritating vapours, undoubtedly predispose. (3) Bad health predisposes especially to granular pharyngitis, so much so that the throat in some persons constitutes a veritable barometer of the state of their health. (4) The gouty, rheumatic and tuberculous diatheses. (5) Chronic pharyngitis is often secondary to carious teeth, pyorrhœa or septic tonsils. (6) Nasal obstruction from any cause predisposes by inducing mouth breathing; nasal discharge, due to rhinitis or sinusitis, infects the pharynx and causes congestion by the constant efforts to get rid of it. (7) Wrong methods of production of the voice (clergyman's and school-teacher's sore throat), excessive smoking, the constant use of alcohol, spiced or hot foods. (8) The bristle of a toothbrush, or a fish-bone impacted in the pharynx, is a not infrequent though unsuspected cause. (9) Chronic pharyngitis is often seen in people who live too well. The excessive secretion and the perpetual hawking direct the attention of the patient and of his medical adviser to the throat, larynx, or lungs; but the cure cannot be accomplished until dietetic and other measures are directed to the relief of the portal congestion.

(10) Pharyngitis, especially in its chronic forms, is often associated with anæmia (see the Plummer-Vinson syndrome, § 227). (11) The pharyngitis of influenza is slow to go, and is accompanied by a very irritating cough.

Prognosis.—Pharyngitis is one of the most frequent and troublesome of the minor ailments. The milder varieties of acute pharyngitis last only a few days, but the more severe forms may last many weeks, and be followed by considerable debility. All the chronic forms have a great *tendency to relapse.*

Treatment.—The indications are to relieve the local inflammation, to improve the general condition, and to prevent relapse. For the *acute forms,* most of the remedies mentioned under Tonsillitis are available. Penicillin lozenges and the sulphonamides are most useful. In all subacute and chronic forms, smoking, alcohol, and other causes of local irritation must be avoided. Excessive secretion may be removed by a gargle of bicarbonate of soda. For the "relaxed throat" a gargle consisting of a wine-glassful of water, to which a pinch of salt has been added, may be used; so, also, are gargles of alum, potassium chlorate, and ammonium chloride (Formulæ 15 to 19). Mandl's paint is a good application, and carbolic acid employed as a spray, gargle, or lozenge, is of value. A good spray is that of menthol (1 in 50 of paroleine). Later, astringent paints should be used—*e.g.*, nitrate of silver (4 per cent.) or equal parts of solution of iodine (B.P.) and the glycerine of alum. Codeine in ½ gr. doses is the best remedy for the irritating cough.

The most efficient treatment for the granular forms of pharyngitis, where gargles are of little use, is painting with silver nitrate (8 or 16 per cent.), tannin (1 in 8), or with liquor ferri perchloridi, or iodine in glycerine. The galvano-cautery may be applied to the individual granulations. For a permanent and radical cure, when adenoids are present, these must be thoroughly removed under general anæsthesia. Nasal obstruction, if present, must also be relieved. Any sepsis in the nose, tonsils or teeth must be adequately treated. Especially in the granular varieties, the general health is often more important than the local condition, and many a relapsing and granular pharyngitis can be cured by Blaud's pills. The rheumatic or gouty diathesis, dyspepsia or constipation, especially if associated with portal congestion, should be appropriately treated.

§ 155. **II. Tonsillitis,** or inflammation of the tonsil, is met with clinically in acute and chronic forms.

(*a*) **Acute Parenchymatous Tonsillitis.**—The whole substance of each tonsil is inflamed and appears red and swollen.

(*b*) **Acute Follicular Tonsillitis.**—The inflammation is more superficial and the crypts especially are involved, becoming filled with fibrin, leucocytes, bacteria, etc. The tonsils are not so swollen as in (*a*), but their surface is studded with yellow dots which may be wiped off without bleeding.

The *symptoms* of both varieties are the same. A sore throat is complained of and pain on swallowing may be severe. At the onset there

may be a slight rigor and the temperature varies between 100° F. and 104° F. General malaise, headache and pain in the limbs occur. In children the general symptoms are often more marked than the local. The tongue is furred and the breath offensive. The cervical glands on both sides are enlarged and tender.

The *Diagnosis* of both these forms of tonsillitis from scarlet fever and diphtheria is sometimes a matter of considerable difficulty, but one of great importance. It is given in the form of a table (X, p. 228).

Etiology.—Even in healthy individuals many micro-organisms may be found in the tonsils, *e.g.*, streptococcus, staphylococcus, pneumococcus, influenzal organisms and micrococcus catarrhalis. In tonsillitis one or more of these may be the exciting cause. (1) Any general cause of ill-health predisposes. (2) Unhygienic conditions, such as bad ventilation and overcrowding. (3) The tonsils become acutely inflamed in scarlet fever, in diphtheria, often in influenza and "colds," and in so large a proportion of cases of rheumatic fever that they are regarded as the portal of entrance of the organism of that disease. (4) Many cases arise by "droplet infection" in schools and hospitals. (5) Fish-bones and bristles of a toothbrush sometimes give rise to one-sided tonsillitis.

(*c*) **Quinsy or Peritonsillar Abscess.**—In this condition, which occurs usually after tonsillitis but occasionally primarily, an abscess forms just outside the capsule of the tonsil, as a rule only on one side. Severe pain is felt in the throat and swallowing may be almost impossible. The pain radiates to the ear and down the neck. The temperature may be high (103° F.); the patient looks toxic and speech is thick and muffled. Trismus often makes examination difficult. The anterior pillar of the fauces and the soft palate on the affected side are very red and œdematous and bulge forwards, while the tonsil is pushed inwards. Much sticky mucus is present. The cervical glands on the corresponding side of the neck are enlarged and tender.

(*d*) An uncommon form of acute tonsillitis is known as **Vincent's Angina.** It is often mistaken for diphtheria; it can occur during convalescence from diphtheria, and *vice versa.* As a rule only one tonsil is affected, occasionally both. It is characterised by one or more patches of exudation, often presenting a necrotic appearance, on the tonsil or adjacent anterior pillar, and sometimes encroaching on the palate. Later a deep ragged excavation may form. The pellicle is not easily detachable, and leaves a shallow ulcerated surface, the healing of which may be somewhat tedious. It is attended by some pyrexia and constitutional disturbance, usually slight. There is characteristic fœtor of the breath. A smear from the affected surface contains a large fusiform bacillus which stains with the ordinary aniline dyes, but will not grow on ordinary culture media, and a delicate mobile spirochæte. Both these organisms may be found occasionally in ordinary ulcerative stomatitis, in carious teeth, and in some cases of septic scarlet fever. Salvarsan powder may be applied locally or the anti-syphilitic arsenical preparations may be injected. Penicillin lozenges and systemic penicillin given together are effective. Nicotinic acid (100 mgm. per diem) seems to be beneficial.

(*e*) **Agranulocytic Angina** (Agranulocytosis, malignant neutropenia) is uncommon and has only recently been recognised. Women are much more susceptible than men. It occurs as a rule about middle age. In *acute cases* there is first soreness of the throat,

and malaise with pyrexia. The disease rapidly progresses and there is necrotic ulceration of the tonsils, fauces, buccal mucous membrane, and sometimes the vagina and any part of the intestinal tract, but especially the rectum. In the absence of polymorph cells in the blood, any invading micro-organism produces widespread local invasion of tissues as well as septicæmia. Prostration is marked and the patient commonly dies in a few days. A *chronic type* may also occur with recurrent mild attacks of sore throat, malaise and fever, often occurring at the menstrual periods. The blood picture is characteristic. The total white count is very low (it may be only a few hundred) and the polymorphonuclear granulocytes (neutrophils, eosinophils and basophils) are much reduced in number (absolutely and relatively), and may be absent. This is due to their non-formation by the bone marrow. Sternal bone marrow biopsy shows either that no cells of the granular series are formed at all, or that myeloblasts and myelocytes are present in large numbers but are apparently unable to mature to form polymorphs. Certain drugs cause this condition, particularly amidopyrine and allied compounds, especially when associated with a barbiturate; also arsphenamines, thiouracil, dinitrophenol, the sulphonamide group and occasionally compounds of heavy metals.

Treatment.—(1) Elimination of possible causes, *e.g.*, drugs; (2) treatment of local lesions with mouth washes (*e.g.* hydrogen peroxide) and sprays; (3) treatment of agranulocytosis by stimulating the formation of granulocytes by the use of nuclein derivatives, *e.g.*, pentnucleotide, which is injected daily intramuscularly in doses of 20–40 c.c. in divided doses until the white count rises. In certain cases intravenous pyridoxine 100–200 mgm. daily is helpful. (4) Penicillin injections are useful to prevent bacterial invasion.

(*f*) **Glandular Fever** causes (i.) a sore throat, with general reddening of the fauces and tonsils, enlargement of lymphatic glands and often some enlargement of the spleen. (ii.) In the anginose variety, after 1–2 weeks of malaise and fever, the throat becomes sore and a membrane indistinguishable from that of diphtheria forms on one or both tonsils. There is much peritonsillar œdema and cervical adenitis; the axillary glands and the spleen may enlarge. Diphtheria bacilli cannot be cultured from the membrane, and a rash with the typical blood changes (§ 499) helps to confirm the diagnosis.

(*g*) **Acute leukæmia** may cause a membranous form of acute tonsillitis as one of its earlier manifestations (§ 543).

Chronic Tonsillitis. (*a*) In *adults* it may follow repeated attacks of acute tonsillitis or it may develop insidiously. In both types there may be little surface evidence of deep infection of the crypts; the tonsils may be enlarged or small and buried; but there is usually redness of the anterior pillars of the fauces and some enlargement of the cervical glands. In a crypt infection of the insidious type the patient may be unaware of any throat trouble; he may have a chronic toxæmia, leading to arthritis, neuritis, nephritis and diminished vitality. (*b*) In *children*, the condition is indicated by local and general symptoms. *Local:* running nose, muco-purulent rhinitis, postnasal discharge, fœtid breath, enlarged cervical glands, unilateral or bilateral otorrhœa, associated with a history of frequent colds, sore throats and other infections, particularly exanthemata. On *examination:* the tonsils may be large or small and buried; the crypts often contain yellow debris or liquid pus. *General:* Pale, puny children, suffering from nerve, respiratory or alimentary disorders, restlessness, night terrors, etc.

Chronic Enlargement of Tonsils is generally associated with enlarged

adenoids and is regarded as a chronic hyperplasia. In children we find superimposed the symptoms due to adenoid enlargement (§ 154), and a chokiness at night when lying down, sometimes when swallowing. This hypertrophic type occurs especially in the presence of dental sepsis, or persistent mouth breathing, and also in the " catarrhal diathesis." In this latter condition the children are over-weight, subject to recurrent catarrh, and have a bright malar flush which gives the appearance of good health. Sudden death, described post-mortem as Status Lymphaticus (§ 37), rarely occurs in this type of child.

Course and Prognosis of Tonsillitis.—Acute tonsillitis, without complications, is a frequent, sometimes troublesome, but rarely fatal, disease. Sometimes the patient continues at work; at other times he is totally incapacitated. Chronic tonsillitis renders the patient liable to repeated attacks of acute tonsillitis and coryza and is a common source of recurrent pharyngitis, leading to otitis media and deafness. Enlarged tonsils in children occasionally resolve during adolescence; the mental and physical development of children who have chronic enlargement of the tonsils is sometimes impeded. The development of the child is more likely to be hindered by the presence of adenoids, which interfere with the respiration.

Treatment of Tonsillitis.—The indications are (*a*) to reduce local inflammation; (*b*) to reduce pyrexia; and (*c*) in chronic tonsillitis to prevent relapse and improve the general health.

(*a*) Penicillin lozenges should be kept in the mouth and gently sucked. A spray of anethaine (4 per cent.) relieves the pain. Cold or hot compresses externally, steam inhalations, warm gargles of potassium chlorate, sodium bicarbonate, aspirin, and carbolic acid in glycerine (1 in 40) or formalin (2 per cent.) relieve the congestion (Formulæ 15 to 19). Frequent very hot saline gargles are as useful as anything. In subacute cases the tonsils may be painted with Mandl's paint.

(*b*) To reduce the pyrexia a brisk saline purge should be given at the onset. Give copious drinks of warm fluid; then sodium salicylate, as in rheumatism, or liquor ferri perchloridi. The sulphonamides are often helpful in infections with hæmolytic streptococci, *B. Friedlander* and pneumococcal infections (see Tables XXVIII, XXIX, § 515). Penicillin injections are most useful when the organisms are susceptible. A quinsy should be opened by inserting a fine-pointed pair of sinus forceps into the abscess, and slightly opening the blades. Enter at the point of maximum swelling and softening. A guarded scalpel, with plaster wound round the blade to within half an inch of the point, may be used instead of the forceps. No anæsthetic should be used, other than cocaine locally, lest the cough reflex be abolished and pus inhaled.

(*c*) In chronic tonsillitis the most useful remedies are iron, cod-liver oil, tonic treatment, vitamin preparations and change of air. Salicylic acid and guaiacum are used in the relapsing form. Chronic enlargement may be diminished by painting the throat with glycerine of tannic acid, or other astringents (*vide supra*); but in most cases the

question of enucleation of the tonsils arises sooner or later, or their destruction by diathermy in cases where removal is contra-indicated. Recently in America repeated small doses of deep X-ray therapy have been used, with good effect, for children with infected tonsils and adenoids.

§ **156. III.** In **Scarlet Fever** (§ 477) the tonsil is generally the chief seat of inflammation in the throat. Both scarlet fever and acute tonsillitis start more or less suddenly, with constitutional symptoms, and thus the diagnosis is often difficult. There are four distinguishing features of scarlet fever—viz.: (i.) The diffuse *scarlet* colour of the soft palate and pharynx, with complete immunity of the larynx; (ii.) sudden onset of the illness with high fever and often vomiting; (iii.) on the second day the rash; and (iv.) about the third day the "strawberry" tongue (see Table X and § 477).

TABLE X.

Tonsillitis.	*Scarlet Fever.*	*Diphtheria.*
	(*a*) LOCAL SIGNS.	
Swelling and redness chiefly confined to one or both tonsils. In the follicular form, tonsils covered with sticky mucus, with numerous small, separate yellow spots of secretion on one or both, which are easily removable. Nothing on soft palate.	Diffuse *bright* redness of throat and palate generally. The tonsils swollen, and may be covered with mucus and *sometimes* with multiple yellow points. Nothing on soft palate in ordinary cases.	Ashy-grey patch or patches on tonsils, uvula, *and soft palate* (latter situation is pathognomonic); patches *larger* than in follicular tonsillitis. Patches consist of membrane surrounded by red areolæ; difficult to remove, leaving raw surface. Characteristic smell. Klebs-Löffler bacillus found in membrane. Sometimes a muco-purulent, acrid *nasal discharge*. Comparative absence of pain.
	(*b*) GENERAL SYMPTOMS.	
(i.) Onset moderately sudden, with moderate fever. (ii.) Temperature may be very high, but local symptoms are usually more troublesome than general symptoms.	(i.) Onset with fever and usually vomiting. (ii.) Temperature may be high. Local symptoms a subordinate feature. (iii.) Rash on first or second day. (iv.) Strawberry and cream tongue about third day.	(i.) Onset insidious. Early and marked enlargement of cervical glands. (ii.) Temperature not so high at first, and may remain low during whole course. (iii.) Paralytic sequelæ sometimes.

§ **157. IV.** The sore throat of **Diphtheria** (§ 494) may be recognised at once if there be an ashen-grey patch of exudation *upon the soft palate.*

When this is absent, and the membrane is on one or both tonsils, there may be difficulty in diagnosing between diphtheria and follicular tonsillitis or Vincent's angina. In diphtheria the large size and the colour of the patches (grey with surrounding red areolæ), the raised, sharply defined margin, the difficulty of removing them, and the raw bleeding surface left, enable us to come to a conclusion. The membrane may become blackish with a very offensive odour, and hæmorrhages may occur. There may be considerable swelling of the tissues of the fauces and of the neck (" bull-neck "). The onset is more insidious, the pyrexia less marked, but the prostration is greater in diphtheria. A muco-purulent or hæmorrhagic discharge from the nose is characteristic of diphtheria. Albuminuria is frequent with acute tonsillitis as well as with diphtheria. When other diagnostic features are absent, the presence of *one* large patch on a tonsil, instead of several small patches, is in favour of diphtheria. A swab will reveal the presence of the bacillus. Vincent's angina usually affects only one tonsil (§ 155).

§ 158. V. **Syphilitic Sore Throat** is very characteristic. This and the other *secondary* manifestations of syphilis develop about 3–6 weeks after the appearance of the chancre, but they may appear much later. Symptoms may be slight but there is usually some pain and dryness in the throat and sometimes marked pain on swallowing. The symptoms last for some weeks. (1) Syphilitic *erythema* is the most constant change. Dusky red patches, isolated or symmetrical, appear on the soft palate, tonsil or pharynx. The whole throat may be involved. (2) *Mucous patches* (snail-tracks) appear later as grey-white, translucent or milky areas of variable size, surrounded by a narrow red areola. They are seen on the uvula, the pillars of the fauces, the tonsil and the soft palate and tend to be symmetrical. (3) All the lymphoid tissue in the throat enlarges. *Primary chancre* of the tonsil does occur, though rarely. Symptoms are slight, and it is characterised by great enlargement of the tonsil and the glands on the corresponding side of the neck. Spirochætes may be recovered from the small ulcer or erosion usually present.

Tertiary syphilitic ulcers may produce sore throat, their favourite position being the soft and hard palate, the tongue, the fauces and tonsil, and the posterior pharyngeal wall. They are usually preceded by gummatous swellings. (1) The ulcers are deep, with ragged floor, sharply cut edges, and covered with thick yellow-grey secretion. (2) They are progressive, and in course of time will destroy the hard palate or any other parts they invade. (3) They leave characteristic stellate cicatrices, which are indisputable evidence of the disorder.

The less frequent causes of Sore Throat are—RETRO-PHARYNGEAL ABSCESS, PHLEGMONOUS SORE THROAT, NEOPLASTIC *and* TUBERCULOUS ULCERATIONS, *and* ACUTE SPECIFIC FEVERS.

§ 159. VI. **Retro-pharyngeal Abscess** is an abscess situated in the areolar tissue between the pharynx and the spine. It may develop insidiously, or the onset may be comparatively sudden. It is known by

(1) the rigidity of the head, with difficulty of swallowing, alteration of the voice and inspiratory stridor; (2) evidence of swelling in the posterior pharyngeal wall on inspection and palpation, by which means it is diagnosed from other causes of dyspnœa in children.

Etiology.—Acute cases are met with mostly in the very young and are due to the formation of an abscess in the retro-pharyngeal lymphatic glands following an acute infection in the nose or throat. They are usually met with in feeble and undernourished children.

Treatment.—The acute abscess should be opened at once, through the mouth, the child being held with the head down and no anæsthetic being given.

Chronic retropharyngeal abscess is much less common and is almost always due to tuberculous disease of the bodies of the cervical vertebræ. The abscess tends to point in the posterior triangle, where it should be opened.

§ 160. VII. Phlegmonous Sore Throat—*i.e.*, Acute Septic Inflammation of the Pharynx and Larynx—and ANGINA LUDOVICI (when the inflammation is chiefly external, in the neck).—This very severe disease may start *inside* the throat, with symptoms of sudden pain, accompanied by considerable swelling, leading to severe dyspnœa, stridor, aphonia, and complete dysphagia in a few hours. There is much œdema around the fauces, followed by a brawny infiltration of the skin of the neck, spreading from under the jaw to the tongue and larynx. In some cases there is hæmorrhagic necrosis of the tonsils and surrounding parts, suggesting agranulocytic angina or diphtheria gravis. Sometimes the infiltration starts *externally*, and rapidly invades the internal structures. There is great constitutional disturbance, and a temperature of 102° to 105° F., but unless pus forms, rigors and delirium are generally absent. Pus formation is further indicated by widely and irregularly intermittent pyrexia. Mild cases begin with a stiffness and pain in the tissues around the jaw, and if recovery is to take place, the symptoms go no farther. But in many cases, and especially in alcoholic and debilitated subjects, the disease rapidly progresses, and death takes place in twelve to forty-eight hours from heart failure, coma, or asphyxia from œdema of the larynx. Suppurative forms are very fatal. Among the recognised complications are pneumonia, pericarditis, pleurisy, and meningitis. There is a more chronic form in which induration is in excess of pus formation; this may continue indefinitely until the pus is found and drained.

Etiology.—The condition, happily, is rare, and the causes consequently obscure. (1) It sometimes arises in association with scarlet fever, erysipelas, and small-pox (in former times being a common cause of death in this disease), or other acute specific fevers. (2) Dental suppuration or an alveolar abscess often forms the source from which rapid infiltration starts. (3) It may arise in people apparently in good health, and has then been attributed to the entrance of infection by the tonsils, or through the socket of an extracted tooth.

Treatment.—The indications are to control the inflammation, and to keep up the strength of the heart. A course of penicillin injections and one of the sulphonamides in full doses must be given. The general lines indicated in the treatment of acute tonsillitis should be followed. Use hot or cold applications to the neck. Remove carious teeth or stumps. Free and early incisions should be made if there is pus formation, and the practitioner should be at hand to perform tracheotomy if the dyspnœa be increasing. Stimulants must be liberally administered.

ACUTE ŒDEMA of the throat may be part of the above disease when the œdema is secondary to septic infection; or it may be part of a general dropsy or giant urticaria. It is dangerous, as it may spread to the larynx and cause death by suffocation (§ 167).

§ 161. VIII. CARCINOMA commonly affects the pharynx, more often in men than

in women. No part of the pharynx is immune, but most frequently the pyriform sinus, the ary-epiglottic fold or the epiglottis and base of the tongue and tonsil are involved. The main complaint is of soreness, and, later, of difficulty in swallowing. Metastases in lymphatic glands occur comparatively early, and frequently patients come for treatment when the condition is inoperable. SARCOMA is rare. The diagnostic features are more or less the same as those mentioned for the tongue (§ 216). Diathermy, deep X-ray therapy, radium and occasionally surgical removal are all employed with increasingly satisfactory results.

§ 162. IX. TUBERCULOUS ULCERS of the pharynx occur as secondary lesions. (1) They resemble syphilitic ulcers, but there is pallor of the mucous membrane, and a characteristic " worm-eaten " appearance of the pharyngeal wall : pain is usually severe. (2) *Their course is not nearly so rapidly progressive.* (3) It may be possible to obtain the tubercle bacillus from the scrapings ; and (4) there are usually other manifestations of tubercle, especially in the lungs. *Lupus* is uncommon. For treatment, see Tuberculosis of the Larynx (§ 172).

§ 163. X. ACUTE SPECIFIC FEVERS other than those mentioned above, such as typhoid, give rise to inflammation and ulceration of the throat. In variola, for example, the pustules often form upon the palate, fauces, and buccal mucous membrane, leaving superficial circular ulcers. An examination of the throat is often useful as an aid to the diagnosis between measles, scarlet fever, and small-pox. The first named always affects the *larynx,* rarely the pharynx ; scarlet fever always affects the *pharynx,* and very rarely the larynx ; whereas small-pox affects them *both about equally.* Patches of *Lichen planus* may be found on the palate even before the disease occurs on the skin, and the eruption of varicella may be found in that situation. Other patches may be due to *thrush, herpes* or *pemphigus.*

THE LARYNX

§ 164. Symptoms and Clinical Investigation.—It will be remembered that the two cardinal SYMPTOMS of diseases of the throat (used in its widest sense) were (*a*) Sore Throat, and (*b*) Alterations of the Voice. Both of these may be present in disorders of the larynx, but it is the latter especially which indicates derangements of the organ of voice. Diseases of the larynx are also sometimes indicated by Cough, Hawking, Dysphagia, Dyspnœa, and actual Pain. But in some cases all of these may be absent ; there may, indeed, be pronounced disease of the larynx (*e.g.*, paralysis or papilloma) without any *subjective* symptoms.

The CLINICAL INVESTIGATION of the larynx (laryngoscopy) is a procedure of considerable technical nicety, and requires practice. The necessary appliances are a good steady light, a *reflecting mirror* mounted on a band or a spectacle frame for the operator's forehead, and a small circular *throat-mirror* mounted on a handle at an angle of 135°. The light should be placed on a level with, and a little behind, the patient's left ear. The operator takes his seat directly opposite ; and it is advisable that his seat should be a little higher than that of the patient. Having directed the patient to open his mouth and " breathe quickly in and out," the first step is to adjust the *reflecting mirror* in order thoroughly to illuminate the back of the pharynx. The focal length of the head-mirror is generally 8 to 14 inches, and this should represent the distance of the

mirror from the patient's pharynx. Having warmed the throat-mirror over a small flame to prevent condensation from the breath, ask the patient to protrude the tongue: then hold the tongue gently, with the left hand, in the corner of a towel or in a piece of gauze. Take care not to hurt the under surface of the tongue against the teeth of the lower jaw. Then test the warmth of the throat-mirror against your cheek or the back of your hand, and, having pushed the patient's head a little backwards by pressing your right thumb against the upper teeth, introduce the mirror with the right hand, *taking care to avoid touching the top of the tongue.* Push the mirror obliquely upwards against the soft palate just over its junction with the uvula (Fig. 51, § 152). A good view of the vocal cords should be obtained by slightly lowering or raising the handle. An electrically illuminated laryngoscope may be used. In children and persons with very sensitive throats it is sometimes advisable to render the pharynx less sensitive before laryngoscopy, either by a spray of, or painting with, a 5 per cent. solution of cocaine hydrochloride, or by the administration of a few doses of bromide during the preceding twenty-four hours.

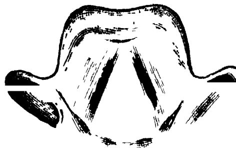


FIG. 52.—Quiet Inspiration.

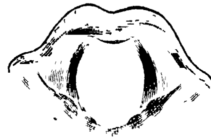


FIG. 53.—Forced Inspiration.

In normal conditions the *epiglottis*, which is in reality anterior, appears at the *upper part of the mirror.* The *vocal cords*, which are of a pearly white colour, are close together at their anterior or epiglottic ends; and at their posterior ends are widely divergent during quiet respiration. Posteriorly they appear to terminate in two prominent knobs seen at the lower edge of the mirror, which mark the position of the *arytenoid cartilages* (Figs. 52 and 53). The *ary-epiglottic folds* stretch on each side from the arytenoids to the sides of the epiglottis. To the outer side of the cords appear the ventricular bands or false cords of mucous membrane. With a little practice, and under favourable circumstances, the bifurcation of the trachea may be seen.

DIRECT LARYNGOSCOPY, with or without anæsthesia (general or local), allows of careful examination of the larynx and has almost entirely replaced indirect laryngoscopy for the performance of intralaryngeal operations. Direct laryngoscopy is also used to expose the larynx preliminary to the introduction of the bronchoscope. By means of the *bronchoscope* (§ 112) the interior of the bronchi may be directly examined. When a foreign body has entered the air-passages, the patient should immediately be X-rayed, then examined by one who is expert in bronchoscopy.

In LARYNGOSCOPY there are FOUR POINTS to be investigated:

(*a*) The presence of *congestion* or *pallor* of the vocal cords and the parts around. Congestion of the vocal cords is an evidence of LARYNGITIS, sometimes of ulceration or new growth.

(*b*) The presence of *ulceration*. Ulceration occurring under middle age is often due either to SYPHILIS or TUBERCLE; after middle life it is not infrequently MALIGNANT.

(*c*) The presence of a *nodule* or *new growth*—most frequently a PAPILLOMA.

(*d*) Whether the vocal cords move normally or not.

§ 165. **Classification.** It has been mentioned that there may be no *subjective symptoms* with disease of the larynx, and therefore it is well to adopt as a basis of classification the *physical signs* discovered by laryngoscopy. However, when symptoms are present there is always some ALTERATION OF THE VOICE (except, perhaps, bilateral abductor paralysis, in which there may be dyspnœa and stridor without alteration of the voice). The principal diseases giving rise to such alterations (*i.e.*, the **causes of alterations of the voice**) may be grouped as follows:

TABLE XI.

I. LARYNGITIS—
- (*a*) *Acute Laryngitis*, including also—
  - Œdema of the larynx.
  - Foreign Bodies in the Larynx or Trachea.
- (*b*) *Chronic Laryngitis*, including also—
  - Perichondritis, and
  - Congenital Laryngeal Stridor.

II. ULCERATION of the Larynx—
- (*a*) Tuberculous Ulceration.
- (*b*) Syphilitic Ulceration,
- (*c*) Malignant Ulceration.

III. NEW GROWTHS—
- (*a*) Benign,
- (*b*) Malignant.

IV. PARALYSIS of the Vocal Cords—
- (*a*) Organic,
- (*b*) Functional.

V. SPASM of the Vocal Cords—
- Laryngismus Stridulus (§ 177).

VI. Diseases of the PHARYNX (§ 153); VII. Diseases of the NOSE (§ 178); VIII. Some severe PULMONARY affections; and IX. Certain NEUROSES also cause alterations in the voice.

I. *The patient complains of huskiness or loss of voice, a comparatively dry cough, soreness on swallowing, and there are local signs of congestion of the vocal cords. The disease is* LARYNGITIS, *of which two varieties are met with,* ACUTE *and* CHRONIC.

§ 166. **Acute Laryngitis** comes on somewhat rapidly, and usually runs its course in a week. As a rule it is not a serious affection, but in children it may be alarming. In children a slight laryngitis coming on suddenly is a frequent cause of what mothers describe as "croup." The child wakes up suddenly at night with loud inspiratory stridor followed by an attack of coughing. This symptom is technically known as *laryngitis*

stridulosa, and is not to be confused with laryngismus stridulus (see § 177). Simple laryngitis is differentiated from membranous croup (laryngeal diphtheria) by the perfect general health of the child and the sudden onset in simple laryngitis.

Etiology.—The chief cause of acute laryngitis is exposure to cold—especially when combined with overuse and wrong production of the voice (*e.g.*, actors, music-hall artists, etc.). It is frequently a part of the "common cold." Diphtheria or measles may start in the larynx. Persons who suffer from chronic laryngitis (*q.v.*) or nasal obstruction are predisposed to attacks. A foreign body in the larynx or trachea is a cause of irritation which may produce symptoms resembling laryngitis.

Prognosis.—The affection is troublesome and apt to recur. When occurring during the course of the specific fevers, the prognosis is less favourable, because œdema of the larynx may supervene.

Treatment.—All use of the voice must be forbidden. The patient must be kept in a warm, moist atmosphere, and should use warm inhalations (such as tr. benzoin co. ℳ 60 to the pint of boiling water, and see also Formula 110). Warm compresses or fomentations should be applied externally, and warm mucilaginous and alkaline drinks should be freely taken. The most efficacious medicine is one containing small doses of potassium iodide. See also formula in § 115. For laryngitis stridulosa, apply hot sponges to the throat, and give tinct. ipecac. in teaspoonful doses, with warm water, every ten minutes until emesis ensues. If much swelling is present, spraying with cocaine and adrenalin is valuable. In more severe cases one of the sulphonamides or subcutaneous penicillin should be given.

§ 167. **Œdema of the larynx**, or œdematous laryngitis, is a clinical phenomenon, not a definite disease. It is often called œdema glottidis, but the œdema is not of the glottis; it occurs above and below the cords affecting the epiglottis and submucous tissue of the larynx. The onset is usually sudden, and attended by considerable dyspnœa, dysphagia, and inspiratory stridor. The diagnosis is usually simple, on account of the swelling which can be seen and felt on palpation at the back of the tongue. If this be absent, some difficulty may be experienced, but the sudden onset of laryngeal dyspnœa should bring the disease to our minds. It may arise either as a primary or as a secondary affection. As a primary disease it may come on as part of an acute septic inflammation of the throat, or it may be part of angioneurotic œdema (§ 609) (see Acute Œdema of the Tongue (§ 215). It may occur as a *secondary* condition in association with (1) one of the various causes of acute or chronic laryngitis; (2) a general anasarca; (3) injury of the glottis by boiling or caustic liquids, etc. Its rapid onset is the chief source of danger, but if the patient does not shortly succumb to asphyxia, recovery generally takes place in a few days.

The *Treatment* consists in the use of ice internally and externally. In severe cases, if a 20 per cent. cocaine spray or a local application of adrenaline with ephedrine fail, scarification of the epiglottis must be resorted to; and if this be unsuccessful, tracheotomy must be performed without delay. In infective cases penicillin and one of the sulphonamides are called for.

§ 168. **The Inhalation of a Foreign Body** will give rise to varying symptoms depending on its size and nature. A *large* foreign body will be arrested *in the larynx* and death from asphyxia will rapidly ensue unless it is removed or immediate tracheotomy

is carried out. A *small* foreign body is likely to pass into the trachea or one of the lower bronchi (usually the right).

Symptoms.—If a small foreign body is arrested in the larynx it will produce hoarseness or loss of voice and possibly some degree of dyspnœa. With a foreign body in the bronchus the symptoms differ markedly, depending on the nature of the material. There is usually some cough on inhalation, followed by a quiescent period. Later, cough will reappear and with it expectoration and possibly dyspnœa. *Non-vegetable foreign bodies* (pins, beads, etc.) on inhalation into the bronchus cause a cough of short duration and then may produce no symptoms for a considerable time—sometimes years: sooner or later, however, the cough returns. The bronchial obstruction produces collapse below the foreign body, and the collapsed lung becomes infected, giving rise to expectoration and, later, hæmoptysis: a lung abscess or bronchiectasis will eventually result. Unexplained attacks of cough and fever with unilateral chest disease should make one suspect the presence of a foreign body. X-ray examination and investigation by the introduction of iodised oil B.P. (lipiodol) and, if necessary, bronchoscopy should be carried out. *Vegetable foreign bodies* (peanuts, orange and apple-pips, etc.) in the trachea or a bronchus soon give rise to acute tracheo-bronchitis: an asthmatic type of wheeze may be heard at the patient's open mouth. Obstructive emphysema of the lung tissue below the foreign body may be produced, followed later by atelectasis with expectoration, dyspnœa, and pyrexia. X-ray examination will show no foreign body, but may show the emphysema or atelectasis. In cases where there is a reasonable suspicion of a foreign body, bronchoscopy should be carried out. In untreated cases lung abscess and eventually death will result.

Treatment.—The foreign body should be removed by direct laryngoscopy or bronchoscopy.

§ 169. **Chronic Laryngitis** is troublesome on account of the perpetual hoarseness and liability to acute laryngitis. Its causes are (1) repeated acute attacks; (2) excessive speaking, singing, teaching, overuse with faulty production of the voice (actors, clergymen, teachers, etc.); (3) masons and others exposed to dusty air; (4) nasal obstruction and mouth-breathing; (5) tubercle, syphilis, and new growths, evidences of which should always be sought for in cases of intractable laryngitis. These usually go on to ulceration (p. 236). (6) Spread of inflammation from adjacent parts. Many cases of chronic laryngitis are associated with a granular condition of the pharynx. Nasal sinusitis is a common cause, often overlooked. (7) Rheumatic and gouty diatheses predispose.

Treatment.—The indications are to avoid the cause and to relieve the local congestion. The removal of the cause is most important, and often most difficult to accomplish, for the living of many of these patients depends upon the daily excessive use of the voice. Much may be done to prevent and relieve the condition by proper voice-production and breathing exercises. This affection is extremely common among teachers, owing chiefly to faulty voice-production, and they ought to be specially trained to obviate this defect. The avoidance of tobacco and alcohol will aid, and residence in a dry climate will often accomplish a speedy cure. Locally, painting with strong astringent remedies, such as zinc chloride (1 in 16) or silver nitrate (1 in 24 or 1 in 16), are useful. These strong applications should not be made more than twice a week; weaker solutions can be applied more frequently. The patient himself may use sprays of alum (1 per cent.), zinc sulphate (½ per cent.), menthol (1 per

cent. in paroleine), or argyrol (10 per cent.), two or three times daily, or inhalations of turpentine, creosote, iodine, menthol, etc., for fifteen minutes three times a day.

§ 170. **Perichondritis** is uncommon and is an inflammation of the perichondrium of the laryngeal cartilages. If considerable, it may lead to necrosis of the cartilages and abscess of the larynx. The differential features, besides loss of voice or hoarseness, are dull aching pain and acute tenderness. These may be accompanied by swelling in the neck. As regards its *Etiology*, apart from traumatism, it is rarely a primary malady. It more often occurs secondary to syphilitic or tuberculous laryngitis or to malignant disease, especially after treatment by radium or deep X-ray. Syphilis is its commonest cause. It may also follow typhoid and other specific fevers.

Prognosis and Treatment.—It is a serious affection, for even in the mild forms the voice is rarely restored. Stenosis of the larynx may result. If there be much swelling the dyspnœa is very marked, and the patient may die from pneumonia or gangrene of the lungs, or, in the suppurating forms, from pyæmia. Abscess and fistula may follow. Tracheotomy may be required: large doses of penicillin should be injected as early as possible.

§ 171. **Congenital Laryngeal Stridor** is a rare form of laryngeal stridor commencing at or soon after birth and generally passing off by the age of two years. It is due to a congenital malformation of the vestibule of the larynx, the epiglottis being folded on itself and the ary-epiglottic folds thus being approximated. Stridor is marked on inspiration, slight or absent on expiration. It is worse when the child is startled or excited, and may be absent during sleep. Cyanosis is rare and although there may be retraction of the thorax and abdomen the child is usually in good health. The cry and voice are normal. As a rule no treatment is required; small doses of chloral or potassium bromide help to quiet a restive child and to lessen attacks.

II. **Ulcerations** *of the larynx are met with chiefly in* TUBERCULOSIS *and* SYPHILIS *and, in persons past middle life,* MALIGNANT DISEASE. *The simple erosions present in* CATARRHAL LARYNGITIS *hardly amount to ulceration. Ulceration is also found in the later stages of* LUPUS *and* LEPROSY, *usually when cutaneous lesions are present.*

§ 172. (*a*) **Tuberculous Laryngitis** should always be suspected when a patient complains of constant hoarseness. This form of laryngitis is recognised by (1) the general pallor of the mucous membrane, accompanied by a thickening or swelling most marked over the arytenoids or the aryteno-epiglottic folds; (2) the occurrence of irregular, slowly growing ulcers, usually bilateral; and (3) the history or presence of pulmonary tuberculosis.

The *Prognosis* is always grave; it is not so very long since recovery was practically unknown. The course of the affection depends more upon the condition of the lungs (§ 131) than that of the larynx.

The *Treatment* at first is largely constitutional. Absolute rest from speech, a warm, dry climate, and sanatorium treatment, are essential (§ 131). Creosote in doses of 1 to 5 minims is recommended. Locally, menthol, one part to five of olive oil, used as paint, or an insufflation of menthol (1 in 7) in equal parts of iodoform and boracic acid, is valuable. In certain cases the application of the galvano-cautery is useful. For the pain, which may be severe enough to cause dysphagia, orthocaine B.P. (orthoform), or benzocaine B.P. (anæsthesin), gr. 3–5, may be inhaled

into the larynx from a Leduc's tube; or the larynx may be sprayed with 10 per cent. cocaine. Alcohol has been injected into the superior laryngeal nerve with excellent results.

§ 173. (*b*) **Chronic Syphilitic Laryngitis.**—The laryngitis accompanying secondary syphilis may resemble simple catarrh, with the addition of whitish patches (§ 158). But that which occurs in the later stages nearly always takes the form of ulceration. The intensity of hyperæmia, the irritability, and the profuseness of the purulent discharge are features of syphilitic ulceration. It is distinguished from tuberculous ulceration by (1) the bright red colour of the mucous membrane; (2) the presence of a deep, *rapidly growing ulcer*, with bright yellow surface, regular edges, often undermined, sometimes unilateral. (3) A history of syphilis and a positive blood Wassermann test.

Prognosis and Treatment.—This form of laryngitis is twice as rapid as, and far more destructive than, the preceding, and is liable to involve the cartilages (*vide* Perichondritis). Even when arrested considerable stenosis may result. The usual constitutional treatment must be carried out. Where neoarsphenamine cannot be given, full doses (60 to 100 grains) of potassium iodide must be taken.

(*c*) **Malignant Disease** and (usually in other countries) **Leprosy** give rise to ulceration of the larynx (see below).

III. **New Growths.**—*The diagnosis between* **benign** *and* **malignant** *growths often presents difficulty.* SYPHILIS *and* TUBERCLE *may very closely simulate new growths, especially malignant ones. The history of the case and a general examination are helpful.*

§ 174. (*a*) **Benign New Growths** are usually papillomata, fibromata or hæmangiomata. These are almost always unilateral and are pedunculated rather than sessile. They occur as a rule in children or young adults, whilst malignant disease is rarely seen before the age of forty. If the growth is on the vocal cord or prevents the cords meeting properly, hoarseness will result; otherwise there may be no symptoms. Papillomata may be multiple and may cause stridor, especially in children. A form of chronic laryngitis is what is known as **singer's nodes.** These often affect the under surface of the vocal cord, and hence may be overlooked for a long time. They are distinguished from other nodules by their involvement of both sides symmetrically. Projections on the cords at the junction of the *anterior with the middle third* are probably Singer's Warts; those situated at the *posterior* ends of the cords are probably pachydermia laryngis. In the latter case there is often a nipple on one cord which fits into a crater on the other. **Pachydermia Laryngis** is a localised chronic laryngitis (§ 169), usually most marked over the vocal processes. **Leprosy** may affect the larynx. Benign growths often cause but little inconvenience. They are generally removable, without ultimate damage, by snares or cutting forceps.

§ 175. (*b*) **Malignant Growths** of the larynx occur chiefly in men. They may be divided into two groups, (1) *extrinsic*, growing on the epiglottis, arytenoids, ary-epiglottic folds, pyriform sinuses and the pharyngeal surface of the cricoid, and (2) *intrinsic*, arising from the vocal cords, the ventricle and false cords, the interarytenoid region and the subglottic area. The *extrinsic* variety starts as a thickening of the mucous membrane, which may resemble a benign growth, or may be greyish-white, or have a ragged surface. It rapidly passes on to ulceration, with soreness or pain and perhaps hæmorrhage; secondary enlargement of the glands follows. Death ensues unless early treatment is instituted. Laryngectomy or radiation therapy are available. *Intrinsic* cancer, on the other hand, is of slow growth, and low malignancy. It usually starts in the vocal cord, and causes a persistent huskiness. Every case of persistent hoarseness occurring in men over middle age should be sent to a laryngologist for examination. The diagnosis is often difficult, but a one-sided sessile lesion in a patient, especially if he is a man over forty, should raise suspicion, and the

case should be watched carefully. If the disease is malignant, ulceration will probably appear and the growth will spread along the cord. The movement of the affected cord will sooner or later be impaired. In many cases it is necessary to remove a piece of the neoplasm for microscopical examination. The operation of laryngo-fissure affords 80 per cent. of cures in these cases if seen early. X-ray and radium therapy, particularly teleradium treatment, afford excellent results in many cases.

IV. **Paralysis of the Vocal Cords** *can only be detected by inspecting carefully both the* POSITION *and the* MOBILITY *of the cords during* (i.) *rest,* (ii.) *phonation and* (iii.) *deep inspiration.*

§ 176. **Paralysis of the Vocal Cords.**—The larynx is supplied by two nerves, the superior laryngeal and the recurrent laryngeal branches of the vagus. The former supplies the crico-thyroid or tensor muscle and the mucous membrane of the larynx, while the recurrent laryngeal supplies all the other muscles. In progressive lesions of the recurrent nerve the abductors are paralysed first, and later on the adductors.

The Signs of Laryngeal Paralysis.—It is very rarely that a single muscle is paralysed; the paralysis nearly always affects a physiological group of muscles—*i.e.*, the glottis-openers (abductor paralysis) or glottis-closers (adductor paralysis) on one or both sides. Paralysis is often accompanied by more or less catarrh, which modifies the appearance somewhat, but the evidences of laryngeal paralysis depend upon the position and mobility of the cords during phonation and respiration. The symptoms are given in Table XII.



FIG. 54.—MODERATE ADDUCTION.—The appearance seen during REST.

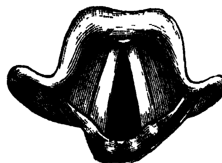


FIG. 55.—CADAVERIC POSITION of cords.

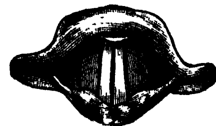


FIG. 56.—Typical position during PHONATION of high notes.

Normally, during rest the cords are midway between open and closed (Fig. 54); during phonation they are approximated so that practically no space is left between them (Fig. 56); during deep inspiration they are widely opened (Fig. 53).

When the cords are normal during phonation, but do not move out on inspiration, there is bilateral paralysis of the glottis-openers—*bilateral abductor paralysis* (Fig. 57). If both cords move during phonation, but one of them fails to move out fully during inspiration, there is *unilateral abductor paralysis* (Fig. 58).

When the cords neither move to the middle line with attempted phonation, nor move as far outwards as normal during deep inspiration, but remain midway between the two in the cadaveric position (Fig. 55), there is *total bilateral paralysis of* adductors and abductors (Fig. 59).

If during phonation and inspiration one cord remains immobile, there is *total unilateral paralysis.*

If there is aphonia, and on laryngoscopic examination the cords do not meet properly during attempted phonation, although they move outwards with inspiration there is *bilateral adductor paralysis* (Figs. 60 and 61).

The *Etiology* of laryngeal paralyses differs considerably in the various forms. They may arise from **organic** or **functional** conditions, but each is so characteristic that it can be readily identified. *Abductor paralysis,* whether unilateral or bilateral, is always organic in origin. If the left vocal cord cannot be abducted, it is almost certainly due to pressure on the left recurrent laryngeal, and this is frequently due to a mediastinal neoplasm. *Adductor paralysis* is always bilateral and functional in origin.

TABLE XII.—LARYNGEAL PARALYSES.

(From Gowers, slightly modified.)

Lesion.	*Symptoms.*	*Signs.*
Bilateral abductor (opener) paralysis.	Voice little changed; cough normal; inspiration difficult and long, and attended with loud stridor.	Both cords near together; not separated during inspiration, but even drawn nearer together.
Unilateral abductor (opener) paralysis.	Symptoms inconclusive; little affection of voice or cough. Brassy cough sometimes.	One cord near the middle line not moving during inspiration, the other normal.
Bilateral complete paralysis.	Weak voice; no cough; stridor only on deep inspiration.	Both cords moderately abducted and motionless (*i.e.*, the cadaveric position).
Unilateral complete paralysis.	Voice low-pitched and hoarse; no cough; stridor absent or slight whilst breathing.	One cord moderately abducted and motionless, the other moving freely, and even beyond the middle line in phonation.
Bilateral adductor (closer) paralysis.	No voice; normal cough; no stridor or dyspnœa.	Cords normal in position, and moving normally during respiration, but not brought together on an attempt at phonation.

(*a*) BILATERAL ABDUCTOR PARALYSIS (Fig. 57) may be due to—

(i.) The earlier stages of *pressure* upon both recurrent laryngeal nerves, as by mediastinal tumour, or œsophageal carcinoma.

(ii.) *Central Causes*, as in lesions affecting the medulla or base of the brain, bulbar paralysis, disseminated sclerosis, syringobulbia, thrombosis, tumours, tabes dorsalis, chronic forms of meningitis (especially syphilitic pachymeningitis), etc.

(iii.) *Peripheral* causes (rare), such as neuritis from toxins (diphtheria, alcoholism, influenza), certain drugs (*e.g.*, lead, arsenic). Myasthenia gravis may produce the same lesion.

(*b*) UNILATERAL ABDUCTOR PARALYSIS (Fig. 58) is due to the same causes acting on one side only. Thus, if on the *left side*, it is commonly due to aortic aneurysm, mediastinal tumour, or cancer of the œsophagus or bronchus: if on the *right side*, the commonest cause is cancer of the œsophagus, and (rarely) a thickened pleura. Pressure upon the vagus in the neck, as by an enlarged thyroid, or cervical glands, may affect one or both sides. (Very occasionally ankylosis of the crico-arytenoid joint is a cause: this may result from rheumatism, tuberculosis, trauma and other causes.)

(*c*) TOTAL (AB- and ADDUCTOR) BILATERAL PARALYSIS (Fig. 59) is always of organic origin. It may arise from any of the causes mentioned under Bilateral Abductor Paralysis, but is most frequently of *central* origin. It occurs later in the disease than abductor paralysis, the abductor fibres in the nerve being the first to be affected.

(*d*) TOTAL (AB- and ADDUCTOR) UNILATERAL PARALYSIS is due to the same causes as mentioned under unilateral abductor paralysis—*i.e.*, usually involvement of the recurrent laryngeal nerve. This condition, however, occurs at a later stage in the case, unilateral abductor paralysis being a feature of the earlier stage. Total paralysis is sometimes called "recurrent paralysis," because it is due to paralysis of the recurrent laryngeal nerve.

(*e*) BILATERAL ADDUCTOR PARALYSIS (Figs. 60 and 61) is always *functional* (viz., unconnected with *gross lesions*): (1) hysterical; (2) simple catarrh, or over-use of the voice; (3) general weakness, as in anæmia. But the first of these is by far the most common.

Diagnosis.—Careful investigation of the chest and œsophagus should be made in all cases and the other cranial nerves should be examined.

Prognosis.—Laryngeal paralysis is generally only a minor element in the case. When occurring alone, however, the prognosis in adductor paralysis is good, because it is always of functional origin. In all forms the prognosis depends upon whether the cause is removable or not. Sometimes paralysis arising from syphilis is remediable if treated early.

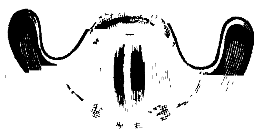


FIG. 57.—BILATERAL ABDUCTOR PARALYSIS.—The patient is able to oppose the cords during phonation, but *the cords do not move outwards during deep inspiration* (as in Figs. 52 and 53).

The same appearance as the above is sometimes produced by acute laryngeal catarrh, but the cords would be pink instead of white.

Treatment.—Hysterical paralysis should be treated on lines laid down in § 888. Strong faradisation or static electricity to the larynx is sometimes indicated, the patient being instructed to call out loudly. In organic paralyses the prognosis depends upon the cause. Syphilitic remedies should receive a fair trial. In organic cases, if dyspnœa be severe, tracheotomy must be performed.

ILLUSTRATIONS OF LARYNGEAL PARALYSIS.—It should be remembered, in studying these illustrations, that to test the motor power of the vocal cords it is necessary to make the patient INSPIRE deeply to OPEN the cords, then to PHONATE, so as to CLOSE the cords, for a given position of the cords conveys no information unless it is first known which of these acts the patient is performing.

In laryngeal paralysis it is very important to decide whether a functional or organic cause is in operation, and the following hints should be remembered:

1. Glottis-closer (adductor) paralysis is functional; glottis-Opener (abductor) paralysis Organic.
2. Bilateral paralysis is often functional; One-sided paralysis is Organic.
3. Left Abductor (glottis-opener) paralysis suggests Aneurysm.

PATIENT'S RIGHT

PATIENT'S LEFT

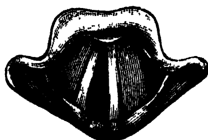


FIG. 58.—LEFT ABDUCTOR, or glottis-opener, paralysis.—DURING INSPIRATION the *left cord* remains fixed, instead of moving outwards as does the right cord. This occurs in early paralysis of the recurrent laryngeal nerve of ORGANIC ORIGIN—*e.g.*, aneurysm.

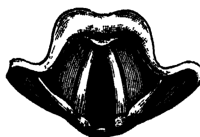


FIG. 59.—TOTAL BILATERAL paralysis.—DURING INSPIRATION and DURING PHONATION *both cords* are immobile, and remain in what is practically the cadaveric position. Always of ORGANIC origin, and frequently central.

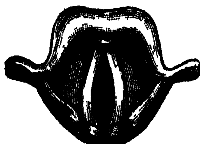


FIG. 60.

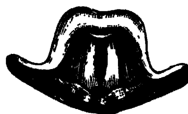


FIG. 61.

FIGS. 60 and 61.—PARTIAL BILATERAL ADDUCTOR, or glottis-closer, paralysis.—It is the condition commonly met with in hysterical or FUNCTIONAL aphonia. DURING PHONATION the cords close anteriorly and posteriorly, but leave an elliptical space between them. Two muscles help to close the glottis—the crico-thyroid in front, and the arytenoideus behind. If the CRICO-THYROID is mainly affected, the condition depicted in Fig. 60 is seen, and it is met with in functional aphonia and exhaustion. The ARYTENOIDEUS closes the posterior angle, and when this is paralysed the posterior angle remains open (Fig. 61). Both of these forms are met with in acute and chronic laryngitis, and are generally independent of any actual nerve lesion, excepting perhaps peripheral neuritis and some rare cases due to a local lesion affecting the recurrent laryngeal nerve of both sides.

V. Spasm of the Laryngeal Muscles, *and consequent* Inspiratory Dyspnœa, *is not a very common occurrence, except in the form of Laryngismus Stridulus, a disease almost confined to childhood. It may arise when a foreign body passes into the larynx, and may occasionally occur in adults who are the subjects of acute laryngitis. Inspiratory dyspnœa may also arise in Bilateral Abductor Paralysis.*

§ 177. **Laryngismus Stridulus or Nervous Croup.**—Syn.: Spasmus glottidis, spasmodic croup, child-crowing, is a form of paroxysmal inspiratory dyspnœa. This condition occurs in young children and it is considered to be due either to a spasmodic affection of the nervous system or to the indrawing by a more than usually deep breath, of unusually soft and yielding laryngeal tissues, so that the glottis is obstructed. The whole attack lasts from a few seconds to a minute or two. The child may become cyanosed or the spasms may spread to other muscles and give rise to general convulsions. Occasionally it terminates fatally. The attack usually comes on at night and starts with a few crowing inspirations followed by a period of apnœa. The attacks tend to recur, and their severity may increase at each recurrence. On the other hand, if the attacks are slight, they may gradually disappear as the child grows older. In the intervals the child is free from cough or hoarseness, and the larynx appears healthy.

Etiology.—It is a manifestation of infantile tetany (§ 778), and may be associated with infantile convulsions. It is practically confined to children of from four months to two years old, and is twice as common in boys. It is more frequent in the spring-time, and it is often hereditary. In older subjects laryngeal spasm and inspiratory dyspnœa occur sometimes in tabes dorsalis, when it forms the laryngeal crisis of that disease. Its rarer causes are epilepsy, hysteria, tetany, parathyroid deficiency, chorea, reflex irritation of the vagus or its recurrent laryngeal branch from mediastinal growths, a growth or foreign body in the larynx.

The *Diagnosis* is not difficult, though it is well to bear in mind the possibility of a foreign body in the throat, larynx, or trachea. There are, however, three pathological conditions to which the term " croup " is loosely applied and which are also characterised by a paroxysmal inspiratory dyspnœa.

1. *Laryngismus stridulus* is the non-inflammatory nervous affection described above. It is recognised by the absence of cough, hoarseness and other symptoms referable to the larynx in the intervals between the attacks. There is often a history of similar attacks.

2. *Catarrhal Laryngitis* (laryngitis stridulosa, false croup) is often associated with attacks of dyspnœa, coming on usually at night in children under ten who are suffering from cough and hoarseness during the day. It may last for an hour or so. It is due to the collection of thick secretion, to the relatively small size of the child's larynx, and to the readiness with which swelling occurs (§ 166). In addition, the nervous system of a child is more unstable.

3. *Membranous Croup*, or laryngeal diphtheria.—This is true diphtheria, and is attended by the constitutional and other symptoms of that disease. A non-diphtheritic membranous croup may occur. A severe injury (*e.g.*, drinking out of a boiling kettle) may certainly result in a membranous or " diphtheritic " inflammation of the mucous membrane.

Treatment of Laryngismus Stridulus.—(*a*) *For the Attacks.*—In severe cases cold water may be dashed in the face, or the patient plunged into a hot bath, or alternately hot and cold, or cold water douches applied. Inhalation of chloroform or ether relieves it promptly. Artificial respiration may revive, even after apparent death. In the rare cases in which the spasm is prolonged and continuous, tracheotomy may be necessary. Mild cases require no treatment except rest and warmth. (*b*) *For the Intervals.*—The patient should be kept very quiet, and any stimuli conducive to an attack should be avoided. Reflex causes of irritation should be sought for in the gums (*e.g.*, teething), the alimentary canal (*e.g.*, worms or gastric disorder), the lungs and elsewhere (*vide* causes). The general treatment of rickets should be adopted, and it is worth bearing in mind that children taken into the country very often cease to have these attacks. Calcium salts, vitamin D_2, bromides and chloral in small doses, or injections of parathormone allay the irritability of the nervous system, on which the condition mainly depends.

VI. and VII. **Diseases of the Pharynx** (*ante*) **and of the Nose** (*post*) *are generally attended by a certain amount of hoarseness and alteration of the voice.* Nasal disorders give to the voice a characteristic nasal twang.

THE NASAL CAVITIES

§ **178. Symptoms and Physical Examination.**—Diseases of the nose will be considered under three cardinal SYMPTOMS : *Inodorous discharge* from the nose (Rhinorrhœa) ; *foul discharge* from the nose (Ozæna) ; *mouth-breathing* and snoring (Obstruction of one or both Nostrils). *Bleeding* from the nose also occurs in some nasal disorders, but it is *not* a cardinal symptom. It is perhaps more generally associated with some constitutional or general derangement. *Sneezing, tickling* in the nose and *sniffing* may also be present ; the quality of the *voice* may be altered, particularly in nasal obstruction ; and the sense of *smell* is always disturbed to some extent. In some instances, headache, vertigo, and other nervous derangements are met in association with disorders of the nose, especially when the free transit of air through the nasal passages is interfered with, and the air pressure within the tympanum disturbed. Various *constitutional symptoms* may result from septic conditions of the nose or the adjacent sinuses, and not infrequently a patient suffers from general toxæmia for a long time before our attention is directed to the true source of his troubles.

Clinical Investigation.—Rhinoscopy or examination of the nose may be effected through the anterior nares (anterior rhinoscopy), and the posterior nares (posterior rhinoscopy); and by digital examination posteriorly.

ANTERIOR RHINOSCOPY.—First examine the anterior nares for any obvious disorder, such as fissures, ulcers, scars from ulcers, any narrowing of the nares, or a deviation of the septum; secondly, introduce a speculum (Fig. 62), using either a direct light or one reflected from a mirror on the forehead, as in laryngoscopy. In this way an examination of the inferior turbinate bone can be made, to see if it be hypertrophied. The inferior or middle meatus should be examined for polypi or alteration in the mucous membrane. If, as frequently happens, the anterior part of the inferior turbinate is hypertrophied, and hides the view, this may be reduced by swabbing out with a cotton-wool pledget soaked in a 10 per cent. solution of cocaine.



FIG. 62.—NASAL SPECULUM.

POSTERIOR RHINOSCOPY is effected by depressing the tongue with a spatula and introducing a warmed postnasal mirror (like a very small laryngeal mirror) which should be placed facing upwards, below and behind the posterior edge of the soft palate. It is important to avoid touching either the dorsum of the tongue or the posterior wall of the pharynx. The patient should be instructed to breathe gently all the while through the nose. This depresses the soft palate and widens the field of observation. By moving the mirror slightly in different directions we are able to examine the posterior nares and turbinate bones, the inner end of the Eustachian tube for any swelling, and Luschka's tonsil (*cf.* Fig. 51). The pharyngeal or Luschka's tonsil is a mass of lymphoid tissue on the pharyngeal roof and posterior wall above and between the Eustachian tubes; when in a condition of hyperplasia it forms the cushion-like growth of post-nasal adenoids (§ 154).

Information may also be derived by passing the finger behind the soft palate; for this it is generally necessary to spray the pharynx with anethaine (4 per cent.). In young children, POSTERIOR RHINOSCOPY is often difficult. A DIGITAL EXAMINATION may be effected by introducing the forefinger behind the soft palate and guiding it along the wall up to the roof of the pharynx. If skilfully done, this causes nothing more than an unpleasant surprise and is not resented by the little patient.

TRANSILLUMINATION of the antra is a useful aid to diagnosis.—A bright light is placed in the mouth and with the room dark a crescent of light appears over the lower eyelids. With an infected antrum, the normal crescent will be absent on that side. Similarly the light may be placed on the floor of the frontal sinus. X-RAY EXAMINATION OF THE SINUSES is extremely helpful; disease in any sinus causes an opacity.

Our *first* inquiries concerning any given case of suspected disease of the nose should be relative to the LEADING SYMPTOM, especially whether there be any nasal discharge, and whether it is inodorous or foul smelling.

We cannot depend upon the patient's statement on this point, because often the disease which causes a foul discharge may blunt the sense of smell. *Secondly,* we must investigate the HISTORY, and whether any of the other symptoms above mentioned were present. *Thirdly,* we proceed to the PHYSICAL EXAMINATION. Test whether the patient can breathe freely through each nostril separately; then examine the anterior and the posterior nares.

Classification.—Diseases of the nose, like those of the throat, are best classified by the PHYSICAL SIGNS met with on examination—viz., **nasal discharge, nasal obstruction, epistaxis**—and their **causes.**

(*a*) ACUTE INODOROUS DISCHARGES (Acute Rhinorrhœa)—the causes of which are—

I. Acute Rhinitis; II. Acute Sinusitis; III. Hay Fever; IV. Spasmodic Rhinorrhœa; V. Diphtheria, and other fevers; VI. Syphilis (snuffles); VII. Glanders; VIII. Myiasis.

(*b*) CHRONIC INODOROUS DISCHARGES (Chronic Rhinorrhœa)—the causes of which are—

I. Chronic Simple Rhinitis; II. Chronic Hypertrophic Rhinitis; III. Post-nasal Catarrh; IV. Chronic Sinusitis and Polypi; V. Cerebro-spinal Rhinorrhœa.

(*c*) CHRONIC OFFENSIVE DISCHARGES (Ozæna), which have for causes—

I. Chronic Sinusitis; II. Atrophic Rhinitis; III. New growths and foreign body; IV. Ulcerations and Bone Disease—Syphilis, Tuberculosis and Lupus.

(*d*) NASAL OBSTRUCTION (Snoring and mouth-breathing)—the causes of which are—

I. Adenoids; II. Polypi; III. Deviated Septum; IV. Hypertrophy of Turbinate; V. Foreign body and neoplasms; VI. Hæmatoma and abscess of the septum.

(*e*) EPISTAXIS, the causes of which may be Local or General.

§ 179. **Acute** (or recurrent) **Inodorous Discharge from the Nose (Rhinorrhœa).**—*The patient complains of an* ACUTE ODOURLESS DISCHARGE FROM THE NOSE, *which should be confirmed as the disease may have blunted the sense of smell. The commonest causes at any age are* ACUTE CORYZA *and* SINUSITIS. *Apart from this,* CONGENITAL SYPHILIS *should be suspected in infancy;* DIPHTHERIA *in childhood.*

I. **Acute Rhinitis** may be set up by *irritation* of any kind, as by the vapour or dust of some trade, or by any injury. For instance, a profuse discharge from one nostril in a child should make us suspect his having inserted a pea, marble, or other *foreign body,* although the history may be wanting. But its commonest cause is a "cold." In **Acute Coryza,** "catarrh," or "cold in the head," there is profuse muco-purulent discharge attended by sneezing, running from the eyes, and febrile symptoms with frontal headache, extending over a few days. It is usually attributed to "a chill"; but it frequently prevails in an epidemic form, and is of infective origin due to a droplet infection (§ 523). It is predisposed to by cold and damp weather, by adenoids, septic tonsils, sinus trouble and other causes

of chronic rhinitis. It is not serious, but causes much discomfort and, if repeated or prolonged, may lead to middle-ear catarrh, or to bronchitis.

Treatment.—Prophylactic treatment consists in accustoming the skin to changes of temperature, in teaching every child to breathe correctly through the nose, in maintaining the general health during cold and damp weather, and in instructing the public that a cold is a contagious disease and that it is therefore the duty of everyone who has a cold to take precautions not to infect others. Sneezing and coughing during the early stages of a "cold" project the infecting micro-organisms many feet into the surrounding air; therefore a handkerchief must always be used. If a delicate member of the household catches the cold, it may become a serious illness. Hence, whenever possible, those infected with a simple cold should stay in bed: Dover's powder may be given. Locally, inhalations, ointments and sprays aid cure and act as prophylactics for those exposed to infection. Anticatarrhal local remedies are ephedrine, amphetamine or menthol, camphor and borax, in paroleine. During the warm weather susceptible persons should be examined for sources of sepsis, such as adenoids, tonsils, sinuses, and have these dealt with. Vaccines, especially autogenous (because different organisms affect different individuals), are excellent both for prophylaxis and for cure, *when given in appropriate doses.* The doses usually recommended are too large for susceptible or delicate persons, and often precipitate a cold. When a susceptible person is threatened with a cold, $\frac{1}{40}$ to $\frac{1}{10}$ of the usual dose will often abort the malady.

II. **Acute Sinusitis** commonly occurs as a result of an attack of Acute Rhinitis, or during the course of influenza. (For other causes, see § 181, I.) The symptoms are nasal discharge and obstruction. In the case of the *maxillary* sinus pain may be felt in the cheek; with *frontal* sinusitis, severe frontal headache is felt. With *ethmoiditis* there is pain behind the eyes; occasionally orbital cellulitis with proptosis arises and severe complications may ensue. With *sphenoidal* sinusitis the characteristic headache is occipital or vertical; see § 181, I, and § 696, IV.

Treatment should aim at favouring drainage from the affected sinus. The application of cocaine and adrenalin, argyrol 10 per cent. or protargol 10 per cent., or the use of ephedrine hydrochloride (1 per cent. in normal saline) in a spray is often extremely useful. Inhalations with menthol or tinct. benzoin co. are helpful. In severe cases the sulphonamides and penicillin should be given. Short-wave diathermy is of value. In the case of the maxillary sinus, puncture and lavage may be necessary, and if the condition fails to clear up, an operation to establish drainage may be called for.

III. **Hay Fever** is a condition of allergy, affecting the nasal mucous membrane and conjunctivæ, due to hypersensitivity to grass pollen. It comes on fairly regularly in April, May, June or August of each year, depending on the particular pollens to which the patient is sensitive (in this country usually to timothy grass).

Symptoms may start between the age of 4 and 20 years, tend to recur each year, but usually die away by middle life. (i.) There is intense irritation of the eyes, nose and back of the throat; (ii.) considerable paroxysmal sneezing occurs, often with (iii.) periodic profuse watery discharge from the nose and eyes; (iv.) headache, mental depression and exhaustion may be present; (v.) Hay asthma, with bronchial spasm, may occur in the worst cases. Symptoms are always aggravated on hot windy days and markedly relieved in cool and wet weather.

Physical signs.—There is marked nasal obstruction, with a general swelling of the mucous membrane of the nose, which is pale and often "water-logged."

Etiology.—The condition is due to an allergic state, often with an inherited disposition to other allergic manifestations. It is always aggravated by contact with the particular allergens, and those affected cannot go near a hayfield in summer months without developing the disease. It is *diagnosed* from simple coryza by its seasonal occurrence, the symptoms and by the appearance of the mucous membrane.

Treatment.—The first indication is to avoid the cause: in severe cases residence by the sea, or at an altitude may be necessary. A course of desensitising injections of a vaccine composed of the pollens to which the patient is most sensitive is best given before the hay-fever season commences: it should be repeated each year and in many cases gradually produces permanent desensitisation. Capsules of benadryl (25–50 mgm. t.i.d.) are often of great help, but occasionally produce mental depression and other symptoms. Argyrol (10 per cent.) is a useful application, and zinc ionisation of the nasal mucosa, the application of the electro-cautery or CO_2 inhalations often help. For the conjunctivitis give dark glasses and use estevin drops: or eye-drops containing ephedrine hydroch. gr. 1, dextrose gr. 5, liq. adrenal. ♏ 10 in N-saline ad. ♏ 100. Ephedrine internally or used as a spray usually cuts short an attack.

IV. **Paroxysmal or Spasmodic Rhinorrhœa** is an allergic disorder; it produces the same symptoms as Hay Fever, but may occur at any time of the year. Various causes are responsible for it in different patients—house dust, animal emanations, face powder containing orris root, etc., and sensitivity to certain articles of diet are the commonest known causes. *Treatment* is on similar lines to that of Hay Fever. Many cases respond to treatment by calcium gluconate (gr. 60 t.i.d., a.c.) and parathyroid (gr. $\frac{1}{10}$ b.d.). Desensitisation may be successful: cauterisation of the nasal mucosa is useful. Benadryl or antistin may give relief.

V. DIPHTHERIA.—There is nasal discharge, often blood-stained, with excoriation of the nostrils and upper lip. Nasal obstruction is present but constitutional symptoms are slight; the condition is often present for some weeks before advice is sought. A greyish-white membrane is seen on the septum and inferior turbinals; diphtheria bacilli are found in the membrane and can be cultured. (See § 921.)

VI. "**The Snuffles.**"—In infants a few weeks old, congenital syphilis is usually attended by profuse nasal catarrh, known familiarly as the "Snuffles." Syphilitic snuffles is obvious in the presence of a purulent rhinitis and other associated symptoms. Respiration is noisy and the discharge is usually blood-stained. (See § 181. IV.*a.*)

VII. GLANDERS.—The copious discharge of viscid semi-purulent matter from the nostrils is one of the earliest symptoms of Farcy, or Chronic Glanders (§ 491).

VIII. **Myiasis** is chiefly met with in tropical countries. It is due to the presence of maggots. The eggs from which they hatch are laid by a fly on the nasal mucous membrane, usually while the patient is asleep. Inhalation or local application of pure chloroform is the usual remedy, but insufflations of calomel are also successful.

§ 180. **In Chronic Nasal Discharges** *it is still more difficult to draw the line between odorous and inodorous discharges, since many of the conditions, though odourless at the outset, become offensive later on, and it is generally necessary to pass in review all the conditions mentioned in this section and* § 181. The following are the chief causes of INODOROUS DISCHARGE:

I. **Chronic Rhinitis** is a chronic inflammatory condition of the mucous membrane of the nose, attended by increased secretion, and usually by thickening. It occurs in three forms: (*a*) SIMPLE; (*b*) HYPERTROPHIC (*infra*); (*c*) ATROPHIC (§ 181). The first two give rise to an *inodorous*, but the ATROPHIC to an *odorous* discharge.

CHRONIC SIMPLE RHINITIS is a chronic, congested, and sometimes, later on, hypertrophied state of the mucous lining of the nose, with a

continuous mucous or muco-purulent discharge. There is generally some nasal obstruction, giving rise to altered voice and snoring.

Etiology.—(i.) It is *predisposed to* by cardiac and pulmonary disease, alcoholism, and the tuberculous diathesis. It may be *determined* by (ii.) recurrent attacks of neglected coryza; (iii.) injury caused by an unsuspected foreign body, in which case the condition is generally confined to one side; or (iv.) constant irritation of dust and noxious vapours—*e.g.*, in masons, fustian-cutters. (v.) Oversmoking. (vi.) It is often associated with adenoids, enlarged tonsils, a deflected septum, and other causes of obstruction in the nose. (vii.) Obscure antral or sinus trouble.

Prognosis.—The disease is chronic, and requires prolonged treatment. The chief fear is that middle-ear catarrh may result from the extension of the inflammation up the Eustachian tube. Even apart from this, it is very important to treat these cases in children, because the condition interferes with the respiratory functions of the body.

Treatment.—In the early stages alkaline washes—sod. chloride, gr. 10., sod. bicarb., gr. 10, and borax, gr. 5, or carbolic acid, gr. 3 to fl. oz. 1—should be sniffed up or given by the nasal douche. This is followed later on by a spray of menthol and eucalyptol (gr. x. to fl. oz. i. of aquol or paroleine), or argyrol (10 per cent.). Constitutional treatment is necessary, by means of tonics, cod-liver oil and other sources of vitamin A. Alcohol should be avoided, and a high and dry climate should be sought. In some cases, treatment by short-wave diathermy or the cautery is helpful.

II. **Chronic Hypertrophic Rhinitis** is a special form distinguished from the preceding by the fact that there is considerable hyperplasia of the nasal mucous membrane, especially over the inferior turbinate bone at its anterior and posterior ends. It presents the same symptoms as the preceding, but in a greater degree. Even in slight cases it is apt to be accompanied by headache and mental depression. It is frequently associated with adenoids. The *Prognosis* is on the whole less favourable. The *Treatment* is much the same, but more active measures are indicated—sometimes surgery and sometimes cauterisation.

III. **Post-nasal Catarrh** is usually due to some definite cause in the nose, or to pharyngitis. Occasionally a localised catarrhal inflammation of the naso-pharynx is responsible. *Treatment* should be directed to the primary cause if this can be found; otherwise follow on the lines advised for Chronic Rhinitis.

IV. **Chronic Sinusitis** and **Nasal Polypi** often produce an inodorous muco-purulent discharge. The conditions are dealt with later, § 181, I, and § 182, II.

V. **Cerebro-spinal Rhinorrhœa** is a continual dripping of a watery, clear fluid (cerebro-spinal fluid) from the nose, due to the formation after injury or disease of a communication between the nasal cavity and the sub-arachnoid space. The fluid passes through the cribriform plate of the ethmoid. Its nature is at once recognised by the fact that it reduces Fehling's solution. The flow sometimes ceases spontaneously. Some cases have been successfully treated by applying to the nasal mucosa

irritants which cause swelling and occlusion of the fistula. By the insertion of a fascial graft the deficiency may be closed.

§ **181. Ozæna** or a **Chronic Offensive Discharge** *from the nose may occur in the later stages of* MANY OF THE CONDITIONS *mentioned in the preceding section. But the chief causes of foul discharge from the nose are as follows: the commonest and foulest occurring in* ATROPHIC RHINITIS *in the young;* SYPHILITIC DISEASE *in middle life; and* CANCER *in the aged.*

Foreign bodies (*which have already been referred to*) *may cause one-sided ozœna, and are described under Nasal Obstruction* (§ 182), *which is their leading symptom. It is here necessary to give some detailed account of*—Chronic Sinusitis; Atrophic Rhinitis; and Ulcerations and Bone disease.

I. **Chronic Sinusitis** may occur in any or all of the accessory nasal sinuses. It is usually due to an extension of infection from the nasal cavities. Chronic maxillary sinusitis is the commonest form; sinus infections may be overlooked for months or years.

Symptoms. (i.) The most constant and cardinal symptom is discharge from one nostril, which is occasionally foul smelling. (ii.) Sometimes discharge is not noticed and nasal obstruction is the main complaint. (iii.) Pain may be localised over the area of the involved sinus, or may be referred to the various parts of the skull, particularly when acute attacks of sinusitis supervene (§ 179). (iv.) Various constitutional symptoms, due to septic absorption, are associated with sinus disease. Lassitude, headache, occasional elevations of temperature, and numerous nervous and vasomotor symptoms are amongst the commonest. They generally have a periodic or paroxysmal character. Facial neuralgia may result from sinus disease. (v.) If overlooked or neglected, sinusitis may excite middle-ear catarrh (with tinnitus, deafness, etc.), recurrent nasal catarrh, and nasal polypi: cases of chronic sinusitis are often associated with asthma, recurrent bronchitis and bronchiectasis.

Physical Signs.—(i.) Pus is seen in the nose, draining from the affected sinus. It is seen in the middle meatus under the middle turbinate when it comes from the maxillary antrum, or from the frontal or anterior ethmoidal sinuses: and flows over the middle turbinate and down to the pharynx when it is derived from the posterior ethmoidal cells and sphenoidal sinuses. (ii.) Transillumination (by putting a bright light in the mouth) shows an absence of the characteristic crescent of light through the lower eyelid, when one antrum is diseased. (iii.) X-ray examination shows an opacity in the affected sinuses, and sometimes a fluid level is seen in a diseased antrum. (iv.) Diagnostic puncture and lavage will further confirm the presence of pus.

Etiology.—Acute Rhinitis or "cold in the head" is probably the most frequent cause. Influenza is responsible for many cases. It may arise in the course of any of the acute specific fevers, and after injury or operation on the nose. Infection of the maxillary sinus frequently follows dental disease or the extraction of teeth.

Prognosis.—Chronic sinusitis is intractable, but very rarely fatal.

Treatment.—Relief is given by inhalations of 25 per cent. menthol in spirit, 10 drops to a pint of boiling water, by ephedrine in a spray or drops or by alkaline douches. Operation may be needed to establish drainage, before a cure is possible.

II. **Atrophic Rhinitis,** also known as idiopathic or true ozæna, is characterised by (i.) a thick, foul discharge, which is sometimes profuse, sometimes scanty; (ii.) the nasal cavities are often large, and the bridge of the nose broad and sometimes depressed. The mucous membrane is thin, pale, and covered with crusts, hard, adherent, and decomposing. Sometimes it is unilateral—*e.g.*, in cases of deviated septum. A certain amount of chronic pharyngitis is usually present. (iii.) The breath has a foul odour, which is not detected by the patient, as the sense of smell is blunted. It is *Diagnosed* from the other causes of ozæna by the absence of ulceration, the presence of atrophied mucous membranes, and wide cavities.

Etiology.—(i.) It is commoner in the young and in women. It usually starts before sixteen years of age. (ii.) Unilateral atrophic rhinitis is mostly due to some local cause, such as deviated septum or sinus disease, the narrower side being healthy. (iii.) The exciting causes of bilateral atrophic rhinitis are obscure. It is much less common than it was some years ago, and its disappearance seems to have occurred with the general improvement in the nation's health. (iv.) In some cases it follows too extensive operative interference.

Prognosis.—Prolonged treatment is necessary, and even this is not very hopeful if the disease be advanced. The disorder is generally most marked at about twenty years of age; it becomes less troublesome at middle age, and, as it gradually disappears with advancing years, we may presume that it tends slowly to spontaneous cure.

Treatment.—Alkaline and antiseptic douches and sprays are indicated, as in § 180. To stimulate the mucous membrane, nasal tampons of cotton wool, soaked in 25 per cent. glucose in pure glycerine, are used. These are useful in unilateral rhinitis, as they ensure respiration through the narrower cavity. The instillation of œstrin into the nose is said to help. Constitutional treatment is also advisable. Vaccines assist certain cases. Various operations such as cartilage grafting have been devised to narrow the nose, and while they do not cure, considerable improvement may result.

III. **Neoplasms and Polypi** (§ 182, II), and **Impacted Foreign Body** (§ 179, I, and § 182, V), are referred to elsewhere.

IV. **Ulcerations and Bone Disease** attacking the nose are mostly of traumatic or syphilitic origin. Neoplasms in the later stages ulcerate, but in the earlier stages give rise to discharge and Nasal Obstruction.

(*a*) **Simple perforation** of the nasal septum causes a small perforation in the front of the cartilage: it is probably due to repeated small traumata.

(*b*) **Syphilitic Rhinitis.**—In the early stages of syphilitic infection we may get an acute catarrh with superficial ulceration, which is the condition found in children with congenital syphilis, known as "snuffles." In the later stages gummata form in various situations, which *rapidly involve the bone* and other parts; the discharge then becomes very foul. The ulcers have the same characters as those affecting the throat (*q.v.*). There is a positive Wassermann reaction.

(*c*) **Tuberculosis** of the nose is extremely rare except in the form of LUPUS, which is not infrequent. It is more common in women than in men, and occurs most often between the age of 15 and 30 years. The anterior part of the septum and the adjacent part of the inferior turbinal present characteristic apple-jelly-like nodules. Perforation of the cartilage of the septum may result; sometimes crusting and fœtor. The progress of the disease is very slow—much slower than with syphilis.

Diagnosis.—Atrophic rhinitis is distinguished from these ulcerations by the pallor and thinning of the mucous membrane, the absence of visible ulcers, and the absence of a history of syphilis or tubercle respectively.

The *Prognosis* of nasal ulceration is fairly good if the patient comes under treatment

early, otherwise it leads to considerable destruction of tissue. Lupus Vulgaris may slowly lead to the destruction of the *alæ* of the nose, but syphilis results in the most extensive destruction of the *bones* both of the septum and the palate; the bridge of the nose falls in, and the anterior nares may be represented by a single gaping orifice. It is this extensive and rapid destruction which is so pathognomonic of nasal syphilis.

Treatment.—Carbolic and astringent sprays are useful palliatives, but surgical measures may be called for if the bone is involved. All dead bone must eventually be removed. Neoarsphenamine or large doses of potassium iodide lead to rapid healing of syphilitic ulcerations. For Lupus, in the early stage, general and local light treatment is useful. The galvanocautery or diathermy may be needed in more advanced cases. Calciferol should be given.

§ 182. **Nasal Obstruction, Snoring, and Mouth-breathing.**—*Nasal obstruction may be partial or complete, and it may exist on one or both sides. It is met with in a greater or less degree in nearly all of the various nasal conditions previously discussed, and it is a marked feature in* HYPERTROPHIC RHINITIS (§ 180, II). *Its commonest cause in children is* ADENOIDS (§ 154). *It is also a cardinal symptom in* NASAL POLYPI, DEVIATION OR SPUR OF THE SEPTUM, COLLAPSE OF THE ALÆ NASI, FOREIGN BODIES, NEOPLASMS, HÆMATOMA *and* ABSCESS *of the* SEPTUM.

Effects.—Apart from the inconvenience of snoring, nasal obstruction renders the individual prone to pharyngitis, stomatitis, bronchitis and other results of entry of cold air into the lungs without its being properly warmed by its passage through the nose. Among the other consequences are a nasal quality of the voice, distortion of the chest (when arising early in life), and impeded respiratory functions of the body generally. The ultimate results are quite out of proportion to the degree of local mischief.

I. **Adenoids** are very common and are described in § 154. They are the most frequent cause of mouth-breathing and snoring in children, and are often overlooked by parents, a circumstance greatly to be regretted for three reasons. First, they are one of the chief causes of chronic otitis media and deafness in after-life; secondly, they impede breathing; and thirdly, the characteristic open mouth and vacant aspect produce an appearance of backward intelligence which, in point of fact, may result.

II. **Polypi,** or pedunculated tumours, are the most frequent new growths in the nose. Polypi are of three kinds: (*a*) MUCOUS; (*b*) NASO-PHARYNGEAL; and (*c*) MALIGNANT.

(*a*) Mucous Polypi are frequently seen. They may occur early in life; but are more common after puberty and more frequent in men than women. They are not neoplasms but œdematous mucosa associated with disease of the ethmoidal sinuses. There is often an allergic diathesis. They may be unilateral, but more frequently bilateral. The extent of polypi formation may vary from a few small beads along the under surface of the middle turbinate to enormous masses completely filling both nostrils. If carefully looked for they may be found in many cases of asthma, hay fever and spasmodic rhinorrhœa. In most cases their detection is not difficult; they appear as long, pedunculated, pale grey, glistening bodies.

Antro-choanal polypus is due to chronic infection in the maxillary sinus. The polypus grows out of the antrum and passes back to the naso-pharynx, where

it may be seen with the post-nasal mirror. It usually resembles a mucous polypus, but it may be rather pink in colour.

(*b*) Naso-pharyngeal polypus, or fibroma of the naso-pharynx, a rare but serious disease, grows from the periosteum of the naso-pharynx. It may expand the bones of the face and produce the deformity known as "frog-face." The main symptoms are: nasal obstruction, discharge, headache and epistaxis. They are considered benign tumours because they do not disseminate or involve glands, although they tend to recur locally.

(*c*) Malignant growths or polypi are not common in the nose and naso-pharynx, but epithelioma and sarcoma do occur. They grow rapidly and cause "frog-face," glands in the neck, pain, and a hæmorrhagic and offensive discharge. Simple polypi may occur as a result of septic infection in cases of malignant disease of the ethmoid and antrum. These neoplasms may assume a polypoid appearance but are generally fleshy and bleed readily.

Prognosis and Treatment.—Simple polypi usually recur when removed with a snare or punch forceps. With the judicious use of cocaine this operation is easy and painless. Radical surgical treatment of the ethmoidal labyrinth is the only curative measure. Antro-choanal polypus may be removed by the snare but no satisfactory result is gained unless the antrum is at the same time opened and drained. The fibroma and malignant growths require operations of some magnitude and skilled use of X-rays and radium.

III. **Deflected Septum and Nasal Spur.**—The nasal septum is rarely quite in the median line, but the displacement is often considerable. Sometimes it results from injury. Various consequences may ensue, such as hypertrophied turbinate on one side, atrophic rhinitis on the other. When an angle is formed in the septum nasi, it is spoken of as a "spur," and this is most readily dealt with by the surgeon.

IV. **Hypertrophied Turbinate** is met with usually either as part of, or a consequence of, chronic hypertrophic rhinitis or nasal allergy. It may occur on one or both sides, and in either case, in narrow nostrils, produces partial obstruction, snoring, and mouth-breathing. *Treatment* should first be directed to the causative disease in the nose. Applications of the galvano-cautery are often useful and occasionally partial removal may be required.

V. **Foreign Bodies** within the nose, and **Malignant Neoplasms**, especially of the ethmoid and antrum, may also produce *unilateral* nasal obstruction and discharge. Epiphora is common even before a local swelling appears.

VI. **Hæmatoma of the septum** is almost always due to trauma. The septum swells so as to occlude both nostrils. If not drained, the contents suppurate and *abscess* results.

§ **183. Epistaxis** (bleeding from the nose) may be a symptom of nasal disorders, but if in any appreciable quantity it is usually evidence of some general disorder. Frequently both general and local causes are in operation. The *nasal cavities should be carefully examined.* The blood-vessels give way in this situation (sometimes as a kind of safety valve) merely because they are thin-walled, numerous, near the surface, and liable to traumata great and small. So much is this the case that the diminished atmospheric pressure on high mountains may produce nose bleeding.

(*a*) Local Causes, in which the hæmorrhage consists usually of little more than streaks, may arise from any marked congestion of the mucous

membranes, such as that which accompanies adenoids, polypi, acute rhinitis, multiple telangiectases, worms in the nose; or as a consequence of mechanical violence, applied either directly to the nose or to the base of the skull, traumatism or foreign body. Any destructive disorder—such as new growths, especially malignant, syphilitic, tuberculous, or other ulcerations (which if small are *very apt to be overlooked*)—may be attended by recurrent bleeding. When small in quantity the blood often passes backwards into the throat and is swallowed, or it may be coughed up, and be mistaken for hæmatemesis or hæmoptysis.

(*b*) With Constitutional Causes the bleeding is usually, although not always, of larger quantity, and it may, indeed, be so profuse as to endanger life. In this group the blood comes from a spot near the anterior part of the septum. Among the *predisposing causes*, there is in certain individuals an idiopathic family tendency to bleed from the mucous surfaces (not amounting to hæmophilia) with or without a wound. Epistaxis is more frequent in children, especially in boys. It is also met in the aged, but only when vascular disease and some other conditions about to be mentioned exist. The constitutional causes may be grouped under (*a*) *Alterations in the Cardio-vascular System*, and (*b*) *Altered Blood States.*

(*a*) Epistaxis occurring for the first time in an apparently healthy person over forty years of age should always give rise to the suspicion of chronic nephritis or hypertension. I have observed several patients who, after repeated admissions to hospital for epistaxis, have finally come in to die of cerebral hæmorrhage. Epistaxis frequently occurs with cardiac valvular disease, emphysema, chronic bronchitis, and cirrhosis of the liver: also with thoracic tumours, extremes of temperature, after violent exercise, with the menstrual period, mountaineering and in aeroplanes.

(*b*) *Altered Blood States:* Purpura, hæmophilia, scurvy, leukæmia, anæmia (simple and pernicious), deficiency of blood platelets (thrombocytopenia), and the specific fevers, especially typhoid, acute rheumatism, and the hæmorrhagic forms of the exanthemata. It is in children a not infrequent prodromal manifestation of whooping-cough and other fevers.

Prognosis.—Slight epistaxis in children is of no consequence, but when occurring for the first time in persons at or past middle life it should receive serious attention. Inquiry should always be made as to whether it has occurred previously because, as above mentioned, certain persons have this tendency, and in these the symptom is not important.

Treatment.—Epistaxis which accompanies nephritis and the congestion of cardiac and pulmonary disease should not be checked unless the amount be profuse. In such cases the epistaxis is usually preceded by headache, and is accompanied by high blood-pressure. In all cases of epistaxis, examine the blood pressure. So long as this remains high or moderate no harm can accrue from the epistaxis.

(*a*) The treatment of *the attack* resolves itself into checking the hæmorrhage. The patient should be kept perfectly quiet, sitting up in bed, the head being cool, the feet warm. With the head tilted slightly to one

side, palatal movements should be restricted by instructing the patient to breathe through the mouth, with a dental prop or cork between the teeth (Trotter). Morphia should be given if the hæmorrhage is severe. Pressure should be kept up over the anterior part of the septum with the thumb and forefinger externally. The cautery, at a dull-red heat, may be applied to the bleeding spot. Other useful measures consist of using an adrenalin spray, or in severe cases packing the nose with ribbon gauze soaked in adrenalin or adrenalin and cocaine (5%). Rarely is it necessary to tie, by operation, the anterior ethmoidal artery or even the external carotid artery. Serious anæmia may be suspected when there is extreme pallor of the skin and mucosa; this can be confirmed by a blood examination. In such a case it may be necessary to resort to blood transfusion (§ 537).

(b) *Between the attacks a thorough investigation* of the nasal and post-nasal cavities must be made. A deflection of the septum near the front of the nose on which dust or face-powder collects may be responsible and require correction. Minute lesions are easily overlooked. Vaseline or lanoline introduced into the nostril often helps to prevent attacks: this is most useful where children are prone to epistaxis.

THE THYROID GLAND

This gland is anatomically connected with the upper respiratory passages, but is physiologically quite separate. The activity of the gland is in part controlled by the thyrotropic hormone of the pituitary. Deficiency of thyrotropic hormone leads to underaction of the thyroid gland, whereas excess may produce hyperthyroidism. From the colloid secretion of the thyroid thyroxin has been isolated; it contains iodine. When the thyroid secretion is increased the thyroxin stimulates the sympathetic nervous system, in part by acting on the suprarenal medulla. Together, these stimulate the liver to liberate glycogen, which circulates as glucose. The thyroid is in close relationship with the other ductless glands, especially the suprarenal, pancreas, and ovary. In health it enlarges at puberty, during menstruation, sexual excitement, pregnancy, lactation, and in the presence of most acute specific fevers, notably rheumatic fever. An unusual degree of enlargement at puberty is not pathological unless constitutional symptoms are present.

Symptomatology.—There are two opposite clinical conditions which may arise from disorder of the thyroid gland. In one there is a *diminished* thyroid action, a condition of *Hypothyroidism*, the symptoms of which (lethargy, lowered vitality, and impaired growth and development) are similar in kind but less in degree to those of Myxœdema and Cretinism. The other condition is one of *increased* (or perverted) thyroid action or *Hyperthyroidism* (thyrotoxicosis): this, with the exception of the proptosis, can be produced by the internal administration of thyroid extract or thyroxin in large doses to normal people. It is important to remember that the size of the gland does not necessarily aid diagnosis, for enlarge-

ment of the gland is consistent with diminution of its function; while what appears to be a small gland may be functionally very active.

§ 184. **Physical Examination and Classification.**—There are but two physical signs referable to the thyroid gland—viz., enlargement or diminution of volume, and altered consistency. When the change in volume is only slight it is difficult, if not impossible, to estimate it with accuracy, because it is partially covered by muscles, and is intimately connected with the trachea and other deeper structures. The patient should be instructed to let his head fall forwards and *to swallow* whilst we endeavour to palpate the gland. The thyroid rises during deglutition as does no other tumour in the neck. Note whether the enlargement is regular and diffuse, or irregular and localised. Some idea may be obtained of the progress of a case by measuring the neck from time to time, always exactly at the same level.

When the thyroid fails to develop normally, part of the thyroid tissue may be left at the base of the tongue. There it forms a painless, soft swelling in the mid-line, which may not attract notice until it enlarges at puberty or later. If the swelling be removed, myxœdema or hypothyroidism will follow in the event of there being no other thyroid tissue.

Classification.—There are six well-marked causes of *enlargement* of the thyroid, and several rare causes. *Diminution* of the gland occurs in two well-marked types.

(A) **An Enlargement of the thyroid** is—at some stage of the malady—the essential or pathognomonic feature in—

COMMONER CAUSES.

REGULAR ENLARGEMENTS:

without toxic symptoms ..	Parenchymatous Goitre. § 185.
with toxic symptoms ..	Graves' Disease, or Exophthalmic Goitre. § 186.

IRREGULAR *or* NODULAR ENLARGEMENTS:

without toxic symptoms ..	Simple Adenoma. § 187.
	Colloid Goitre. § 188.
	New Growths. § 189.
with toxic symptoms ..	Toxic Adenoma. § 190.

RARE CAUSES are: Anæmias; Specific Fevers; Leukæmia; Hæmorrhage; Granulomata and parasitic diseases; Reidel's disease; Menopausal goitre; Cretinism; Acromegaly (some forms).

(B) **Atrophy of the thyroid**—or at any rate a diminution of its function (and usually of its size)—is the essential feature in two diseases.

I. Cretinism	§ 191
II. Myxœdema	§ 559

It therefore follows that:

1. Increased or disordered thyroid secretion gives rise to profound disturbance of the general health, and neuro-vascular irritation (Graves' disease).

2. An innocent enlargement of the thyroid, unaccompanied by increased or dis-

ordered thyroid secretion, has no effect on the metabolism (as in many cases of simple goitre).

3. Simple absence or diminution of the thyroid secretion (*a*) when it is congenital or comes on in early life, causes deficient development both mentally and physically (*i.e.*, cretinism); and (*b*) when it supervenes in adult life, causes lethargy and deficient vitality (myxœdema).

(A) *There is a* UNIFORM ENLARGEMENT OF THE THYROID GLAND, WITHOUT TOXIC SYMPTOMS: *the patient is between the* AGE OF 5 AND 20. *The disease is* PARENCHYMATOUS GOITRE.

§ 185. Parenchymatous Goitre. This condition arises especially in endemic areas in England, as well as abroad (especially in Switzerland and certain parts of India).

Symptoms.—(i.) The patient is noticed to have a uniform smooth and rather soft enlargement of the thyroid gland, of small or moderate degree. One lobe or the isthmus of the gland may be enlarged more than the remainder. (ii.) The general health is good, but the patient is often somewhat anæmic. (iii.) Otherwise the symptoms are those of hypothyroidism rather than of hyperthyroidism. (iv.) If the enlargement lasts to adult life, it becomes a colloid or adenomatous goitre.

It may be *Diagnosed* from other tumours in the neck by the fact that it invariably rises with the larynx during deglutition. The enlargement generally increases steadily, but it is rare that there is any danger from tracheal obstruction and asphyxia.

The *Etiology* of the condition is not well known. It starts in childhood or may appear at or near puberty and last to adult life. It affects women more than men. The disease used to be endemic in certain districts in England and still is so in parts of America and the Continent, especially Switzerland and the Tyrol where the food and water are deficient in iodine. All cases however are not so simple. In some the assimilation of iodine is defective, possibly due to intestinal conditions such as absence of vitamin in the diet, or excess of bacteria; in others it may be that the thyroid cannot utilise the iodine. McCarrison's work proved that goitre in Chitral and Gilgit was due to fæcal contamination of the drinking water. The work of McClendon and Hathaway (which has been confirmed by a recent Medical Research Council Report) showed that most forms of goitre were caused by deficiency of iodine. In ordinary diet iodine is obtained from milk, butter, fruits and leafy vegetables; vegetables lose two-thirds of their iodine content in cooking and when fish is canned its iodine content is lost.

Treatment.—Small doses of iodine cure most early cases. In parts of Switzerland, 1–2 grs. of iodide of potassium are given weekly, sometimes in salt, with resulting cure or prevention of goitre. Drinking-water should be boiled. McCarrison obtained cures by vaccines prepared from the stools, and by intestinal antiseptics, especially thymol and lactic acid bacilli. He found small doses of thyroid, gr. $\frac{1}{4}$–$\frac{1}{2}$, with a local application of Ung. pot. iod. over the tumour usually brought about a cure. Surgical interference is necessary if pressure symptoms occur.

There are ENLARGEMENT *of the* THYROID, PROPTOSIS, NERVOUS SYMPTOMS *and* RAPID PULSE; *the patient is usually a* YOUNG WOMAN. *The disease is* EXOPHTHALMIC GOITRE.

§ **186. Graves' Disease** (Syns.: Primary Thyrotoxicosis, Exophthalmic Goitre, Basedow's disease). Usually the onset is insidious, but it may be acute, after sudden shock or acute focal infections. There are *five* groups of symptoms, and the varieties of the disease depend on which of these predominate.

Symptoms.—1. *Cardio-vascular* disturbances are among the earliest and most important symptoms. They are never absent, and may exist for months before any other evidence appears: (i.) Palpitation. (ii.) Tachycardia is present during rest and sleep; a raised pulse rate during sleep aids diagnosis, as psychological causes are thus excluded. The heart rate may be 100 or more at rest, and may rise to 150 or more on slight exertion or emotion. (iii.) The pulse is forcible and the systolic blood pressure raised, with a corresponding rise in the pulse pressure. (iv.) Shortness of breath on exertion, is usual: paroxysmal dyspnœa and a distressing sense of suffocation are sometimes present. (v.) At first the heart is hypertrophied, with a widespread forcible cardiac impulse; later myocardial degeneration with corresponding electrocardiographic changes, premature beats, and in severe cases auricular fibrillation ensue.

(2) *Nervous* disturbances are always present. They are very variable: thus (i.) there may be nervousness, irritability, insomnia, depression alternating with excitement, increased reflexes, hysterical attacks, mania, or melancholia. (ii.) Hyperæsthesia, perverted sensations, neuralgic headache, vertigo, and hallucinations of sight or hearing. (iii.) Other signs are fine and rapid vibratile tremors of the outstretched fingers (always) and protruded tongue. (iv.) Vaso-motor disturbances of many kinds, intolerance of heat, sudden perspirations and cutaneous alterations such as pigmentation and loss of hair. The hands are usually hot and sweating even at rest. (v.) Diarrhœa may be an early symptom.

(3) *Thyroid Enlargement* is present at some stage of the disease, though it is rarely the first symptom noticed by the patient. It is always more marked in women of 15–30. In older subjects the degree of enlargement may be slight. The enlargement varies considerably in different cases, and is by no means proportionate to the other symptoms, because the symptoms depend more upon the histological element of the gland which is involved than the degree of enlargement. Mechanical effects of thyroid enlargement may be present (see §§ 81 VII, 187), and occasionally alteration in the voice from this cause is an early symptom.

(4) *Exophthalmos* (proptosis or protrusion of the eyeballs) is present in a varying degree, though sometimes not until late in the disease (Fig. 2, § 11). It is best detected by seating the patient in a chair, standing behind, and looking down the forehead. As a rule no changes can be detected in the fundi. Later on, ulceration of the cornea occasionally takes place, either from neurotrophic causes or from deficient protection.

Even when exophthalmos is not marked, the patient presents a staring expression partly due to true Exophthalmos (§ 833) and partly to retraction of the upper lids.

Four signs of Graves' disease referable to the eyes bear the names of different physicians. *Von Graefe's* sign is a condition in which the upper eyelid lags behind the eyeball when looking downwards, exposing the white sclerotic. *Mœbius's* sign is an insufficiency of convergence of the two eyes when looking at a near point. *Stellwag's* sign is a deficiency of blinking as an involuntary act. *Abadie's* sign is an involuntary twitching or spasm of the levator palpebræ superioris. All except the first are present only in advanced cases, and are not therefore of great diagnostic value.

(5) The *general health* of the patient is always disturbed. There may be no anæmia, but lymphocytosis is usual. Lack of energy, sleeplessness and undue fatigue are usually present and progressive emaciation is marked, owing to the increased rate of the basal metabolism, which may increase 75 per cent. or more. There is usually a lowered sugar tolerance ; true diabetes may develop later. The menses are usually decreased and abortion is not unusual. The blood cholesterol is decreased.

In certain patients the cardiac symptoms predominate. In these cases (*formes frustes*, masked hyperthyroidism), the patient is usually older and complains of palpitation. There is a rapid, forcible apex beat, high pulse pressure and often auricular fibrillation : the thyroid may be little altered in size but is of firm consistence, exophthalmos may be absent ; some tremor and vascular symptoms are present.

Etiology.—(i.) Upwards of 95 per cent. of cases are females. (ii.) A large number are young adults between the ages of fifteen and thirty. (iii.) Locality has no known influence. (iv.) Heredity sometimes plays a part. Other members of the family often show nervous instability or even disordered thyroid action. (v.) Fright, anxiety, love affairs, and mental overwork are potent factors in determining the disease. (vi.) Toxæmia (oral sepsis, etc.) undoubtedly aggravates the disease. Exophthalmos is believed to be due to overaction of the thyrotropic pituitary hormone.

Diagnosis.—The cardinal symptoms are : (i.) the characteristic facies (§ 11) ; (ii.) thyroid enlargement ; (iii.) proptosis ; (iv.) rapid and forcible cardiac action ; (v.) fine tremors of the fingers ; (vi.) mental and emotional instability ; and (vii.) increased basal metabolism, sweating and emaciation.

Prognosis.—The duration of the disease varies from some twelve months to many years : two years may be considered an average. It may certainly shorten life, but many very severe cases recover with modern methods of treatment. The mortality has been variously stated ; modern statistics give from 5 to 10 per cent. If the duration be prolonged, the disease will certainly leave its mark upon the cardiovascular system. Progress may be judged by estimating the basal metabolism. The prognosis in severe cases is worse with myocardial degeneration and mental instability. Those who recover may develop myxœdema in later years.

Treatment. Early recognition of the disease is very important, for much can then be done. Rest in bed with freedom from fuss and worry are the prime essentials, preferably in the country. Treatment in a general hospital ward is therefore undesirable, especially as these patients are

very susceptible to infections such as an epidemic sore throat. The diet should be of high calorie value to counteract the loss of weight. Sleep is most important, and sedative doses of phenobarbitone, chloral and potassium bromide should be used when necessary. All sources of toxæmia must be sought for and eliminated as soon as the patient's condition permits. Liquor iodi aquosus (Lugol's solution) 5–10 minims in milk, twice daily, appears to hasten the cure, but should be given in short courses of 3–4 weeks rather than for longer periods. If operation is contemplated, the course of iodine must be reserved as preoperative medication. Thiouracil, or the more satisfactory methyl thiouracil, reduces the formation of thyroxin and is most beneficial before myocardial damage has occurred. With initial doses of 0·2 G. t.i.d. or q.i.d. for 3 weeks, in the second and third week the thyrotoxic symptoms begin to subside with a gain in weight, reduced metabolic rate and rise in blood cholesterol. The dose should then be reduced to 0·1 G. daily or on alternate days for a period of 12 months: if treatment stops sooner, relapse is probable. Toxic symptoms develop in some 20 per cent. of patients, due to overdose or idiosyncrasy to the drugs, the dose of which must be reduced or stopped according to the effects produced: the commonest are granulopenia or agranulocytosis, skin rashes, toxic jaundice, drug fever and lymphadenopathy. These drugs have also been used in combination with iodine as pre-operative treatment.

Surgical treatment is indicated: (i.) when the gland is greatly enlarged and is producing pressure symptoms, (ii.) after failure of medical treatment, (iii.) when the heart shows myocarditis to be developing. When auricular fibrillation has occurred, this must be controlled with digitalis, and then operation resorted to. Excision of the greater part of the gland is usually most satisfactory, and is attended by a very low mortality in expert hands. In severe cases a preliminary ligature of the superior thyroid arteries is of help. Small doses of X-rays have given good results in many cases. Exophthalmos persisting or increasing after partial thyroidectomy may disappear after complete removal of the thyroid.

IRREGULAR *or* NODULAR ENLARGEMENTS.

The patient is a MIDDLE-AGED *or* ELDERLY *person (usually a woman), with a* NODULAR SWELLING *in the* THYROID GLAND; *otherwise she is in good health. The condition is* SIMPLE ADENOMA.

§ 187. Simple Adenoma.—This common condition comes on later in life than those mentioned above.

Symptoms.—(i.) There are one or more smooth firm nodules in the substance of the thyroid gland which very slowly enlarge over a period of years. (ii.) There are no toxic symptoms, but anxiety may be caused by the size of the swelling. (iii.) If the swellings are large enough there is pressure upon or displacement of the trachea, and occasionally of other structures in the neck. (iv.) Some of the swellings may be soft and even cystic as the result of colloid degeneration or hæmorrhage.

Etiology.—This innocent type of new growth is the result of focal hyperplasia in a gland previously damaged by chronic parenchymatous changes or by focal inflammation. A single adenoma occurring in younger adults may arise from an embryonic cell rest: this "fœtal adenoma" is particularly liable to malignancy.

Treatment.—No treatment is necessary or desirable provided pressure symptoms are absent; otherwise surgical intervention is needed.

The THYROID GLAND *is* ENLARGED THROUGHOUT *and may be* ENORMOUS; *there are* IRREGULAR LARGE CYSTIC SWELLINGS. *The condition is probably* COLLOID GOITRE.

§ 188. **Colloid Goitre.**—Colloid change may arise in a simple parenchymatous goitre in areas where the disease is endemic, or it may be sporadic.

Symptoms.—(i.) The gland slowly enlarges and may form a tumour involving all parts of the gland and weighing many pounds. (ii.) It occurs generally in adolescent girls, but persists into adult life. (iii.) The surface is firm, but not hard, and localised cystic swellings can often be clearly distinguished. (iv.) The enlargement frequently surrounds the trachea, causing atrophy of the tracheal rings. Pressure on the trachea produces feelings of suffocation, and on the œsophagus difficulty in swallowing. (v.) The patient usually shows the early symptoms and signs of hypothyroidism.

Treatment.—If necessary for cosmetic reasons, or if pressure symptoms are troublesome, one or both lobes of the gland will have to be surgically removed. Thyroid will probably have to be prescribed later.

There is a SMALL *or* MEDIUM-SIZED MASS *of almost* STONY HARDNESS *in one part of the thyroid gland; with* ENLARGED CERVICAL GLANDS *and/or signs of* MALIGNANT DEPOSITS ELSEWHERE. *The condition is* MALIGNANT DISEASE.

§ 189. **Malignant Disease** of the thyroid gland is known by (i.) a very hard mass in the gland; (ii.) this grows and becomes fixed to surrounding structures; (iii.) the lymphatic glands in the posterior triangles of the neck are involved early. (iv.) Invasion of adjacent parts produces recurrent laryngeal paralysis, tracheal stridor, and/or dysphagia. (v.) When the primary growth is small, and found only after careful examination, the patient may show signs of deposits elsewhere in the body, particularly in the bones, with spontaneous fractures; (vi.) anæmia of the leuco-erythroblastic type occurs when the bone marrow is involved.

Treatment is surgical when the condition is diagnosed before metastatic deposits develop. Injection of radio-active iodine is on trial.

There is ENLARGEMENT OF THE THYROID, *diffuse or localised, which has* LASTED FOR YEARS *before the appearance of symptoms of hyperthyroidism; the patient is usually a woman of* MIDDLE AGE—*the disease is* TOXIC ADENOMA.

§ 190. **Toxic Adenoma** (Syns. Secondary Graves' disease, secondary thyrotoxicosis) is a condition in which the enlargement of the thyroid may be diffuse or localised, sometimes of considerable size, continuing for years before the appearance of symptoms suggestive of hyperthyroidism.

Symptoms.—(i.) The patient is usually a woman of middle age. (ii.) Cardiac symptoms usually predominate. Palpitation, tachycardia, and shortness of breath on exertion are early symptoms. Sometimes the patient first seeks advice on account of the symptoms of myocardial degeneration, or when auricular fibrillation and heart

failure are already present. (iii.) There are few nervous signs such as tremor, etc. (iv.) Exophthalmos is slight or absent. (v.) The condition is not improved, or is made worse, by the administration of iodine, whereas in primary Graves' disease iodine brings about dramatic early improvement.

In toxic adenoma the patient has had thyroid enlargement for some time. Mild types exist, according to the intensity of the toxæmia.

Treatment consists in the adoption of every measure which can improve the health, such as adequate rest and removal of sources of sepsis and toxæmia. Iodine is better avoided except pre-operatively: drugs of the thiouracil series diminish symptoms, but are not as beneficial as in primary thyrotoxicosis. X-ray treatment is not advisable; surgery is indicated in most cases, especially when carditis is present.

Rarer Causes

Enlargement of the thyroid is also met with (i.) in anæmias, and (ii.) in acute thyroiditis, which may occur with the acute specific fevers. It may go on to abscess formation, as in typhoid fever. (iii.) Rarely, it enlarges during the course of leukæmia. (iv.) Acute hæmorrhage may occur in the gland. (v.) Syphilis, tubercle, lymphadenoma, actinomycosis and parasitic diseases. (vi.) **Riedel's disease** is a chronic inflammation which leads to the slow formation of a hard mass of fibrous tissue which is fixed to surrounding structures, and become dangerous to life from pressure upon the trachea. It is often aggravated by prolonged iodine administration; surgical treatment may be required when dyspnœa and dysphagia are marked. (vii.) **Lymphadenoid** goitre (Hashimoto's disease) occurs in middle-aged women with a long-standing adenomatous goitre. The gland is fairly uniformly hard and nodular but not fixed to surrounding structures: it is invaded by plasma cells. *Symptoms* of weakness, ready fatigue and obesity are accompanied by slight pressure symptoms and a low B.M.R. *Treatment* should be by radiotherapy. (viii.) **Menopausal** goitre is a soft, uniform enlargement of the thyroid, which sometimes occurs in women near the menopause; it is accompanied by a mild degree of hypothyroidism and it may pass on to myxœdema if the general health of the patient is not treated. (ix.) In some types of cretinism defective thyroid activity is associated. (x.) In acromegaly the thyroid is sometimes enlarged.

(B) *Diseases in which the thyroid may be* DIMINISHED *in size—viz.,* I. CRETINISM, II. MYXŒDEMA. *The latter is described elsewhere, since the leading symptom is General Debility.*

§ 191. I. **Cretinism** is a condition of dwarfism and deformity attended by mental imbecility, due to an absence or perversion of the thyroid secretion, and is endemic in certain districts. In advanced and typical cases the face is characteristically broad and flat, the tongue protrudes from the mouth, the eyes are wide apart, and the head is brachycephalic (*i.e.*, broad transversely). The skin and hair are dry and coarse, and the mental condition is extremely backward. In severe cases the body may be so dwarfed that a person of twenty is the size of a child of five. X-ray reveals delayed epiphyseal formation. The limbs are shortened, the neck stunted; pads of fat are present above the clavicles; the hands are short and square (spade-like), the abdomen prominent and an umbilical hernia is often present. Constipation is an early and persistent symptom. Puberty is delayed indefinitely. The thyroid may be enlarged, small, or absent. In *juvenile myxœdema* development occurs normally till a certain age, then suddenly ceases, with signs resembling adult myxœdema. This usually follows an infection, such as measles.

Etiology.—Cretinism is endemic in certain districts, *e.g.*, the valleys of Switzerland, Northern Italy and India. Cases used to occur in certain parts of England. Some of these cases have a large thyroid, but in such patients the cretinism preceded the development of the goitre. Sporadic cases, with atrophic thyroid, are found in

healthy families. In other cases the cretin is the child of goitrous parents; when a goitrous mother has been cured with iodine, her subsequent offspring are healthy.

Prognosis.—The patient may grow up capable of doing light manual work, or may remain an idiot. Under treatment begun early, the child may recover completely, but in other cases, although the body is greatly improved, the mind does not improve in proportion.

Treatment.—Thyroid B.P., beginning with ½-gr. doses, causes a rapid and remarkable change. The skin becomes soft, the general conformation normal, and if the treatment has not been too long delayed, the mind assumes its natural vigour. The patient must *continue* to take thyroid all his life, or else he will relapse. A case showing the remarkable efficacy of this treatment is figured in § 19, Figs. 8A, B and C.

II. Typical MYXŒDEMA is described in detail elsewhere (§ 559). It should be remembered that there are minor degrees of thyroid insufficiency which, though falling short of typical cretinism or fully developed myxœdema, are nevertheless sufficient to account for many of the minor troubles for which patients seek advice. In adults, especially in women about the menopause, increase of weight (especially deposits on the back of the neck and the shoulders), falling hair, intolerance of cold, constipation, muscular fatigue, a slow pulse, a dry skin with a tendency to chronic eruptions, are all suspicious features. In younger women premature greyness is also suggestive. Rarefaction amounting to complete absence of the outer two-thirds of the eyebrow is a fairly constant sign. The treatment is started with thyroid B.P. in very small doses—⅛ to ¼ gr. three times daily, and the dose increased until the symptoms go and the basal metabolism becomes normal (Fig. 1).

CHAPTER VIII

THE MOUTH, TONGUE, AND ŒSOPHAGUS

THE MOUTH

(Lips, Breath, Saliva, Teeth, and Gums.)

INSTRUCTIVE information is afforded by a thorough examination of the mouth. Anæmia, lead and bismuth poisoning, scurvy and leukæmia may be recognised from an inspection of the mouth. Many of the indications of syphilis, hereditary or acquired, are here revealed. Make a thorough examination of the TONGUE, the LIPS, the BREATH, the SALIVA, the TEETH, and the GUMS. The symptoms referable to these structures are considered below.

§ **200. The Lips.**—*Dryness* of the lips is often one of the most conspicuous evidences of gastric and intestinal disorder. *Swelling* of the lips is common with urticaria, angio-neurotic œdema, and with cheilitis. In one type the mucous glands of the lips and their ducts are swollen and dilated; in another, there is persistent exfoliation, with scaling and crusting of the lips. The lips are *pale* in anæmia; *cyanosed* in advanced bronchitis with dilated right heart, and other conditions (see Cyanosis, § 30). A *lilac blue* colour of the lips is often seen with constipation and intestinal dyspepsia. The hard chancre of syphilis, and in elderly men epithelioma, may occur on the lip. *Stellate fissures* around the lips are an almost infallible sign of syphilis, especially when surrounded by a dull red infiltration. This infiltration helps us to distinguish a syphilitic fissure from those due to streptococcal or fungal infections (*perlèche*). *Cracked lip* is seen in nervous people who lick and bite their lips and are exposed to cold winds. It also occurs with dribbling saliva, dyspepsia, constipation and cheilitis; it generally yields to vitamin B_2 together with some simple ointment, unless a secondary infection has occurred. By pressing the corner of the mouth inwards and forwards when the patient opens it, we may detect a mucous patch surrounding a syphilitic fissure inside the mouth. The *scars* left by syphilitic fissures, usually congenital, are white and stellate. (And see § 11.)

Certain *skin lesions* may invade the mucous membrane of the mouth, such as the rashes of small-pox, chicken-pox, measles, lupus erythematosus, pemphigus, and herpes. In measles, the spots, first described by Koplik, appear on the inner sides of the cheeks, opposite the bicuspid or molar teeth, before the skin eruption occurs. They appear as a greyish-white stippling on a slightly raised purplish base, and afford considerable aid in the early diagnosis of the disease. With Wood's glass under ultra-violet light they are readily seen. Lichen planus may affect the mucous membrane of the mouth and tongue long before it appears on the skin; in the mouth it has a whitish appearance resembling secondary syphilis, for which it has sometimes been mistaken. Lupus vulgaris chiefly affects the palate.

§ 201. **The Breath** should normally be quite free from any kind of odour. Offensiveness of the breath (halitosis) may arise from several sources: (1) A want of cleanliness *in the mouth*, particles of decomposing food, pyorrhœa, stomatitis, septic teeth and dental caries. (2) Septic *tonsils*, and other *throat* maladies. (3) Liver disturbance, dyspepsia constipation, and other conditions of the *alimentary canal* (§ 273), and the disordered digestion in *fevers*. (4) Some *diseases of the nose*, *antrum* and *sinuses*; it always accompanies ozæna. (5) A large cavity in the *lungs*, especially if *bronchiectatic*, fœtid bronchitis and gangrene of the lungs produce a putrid odour (§§ 143, 144). The odour of bronchiectasis is characterised by being intermittent; it comes on suddenly, lasts a day or two, and disappears gradually. (6) Certain general conditions are attended by a more or less characteristic odour of the breath. Thus, in *acidosis* it is sweet; in acute *alcoholism* it is alcoholic or ethereal. In *uræmia* it is often urinous. (7) Certain *drugs* cause a characteristic odour in the breath—*e.g.*, turpentine (a resinous odour), chloral (odour of chloroform), bismuth (odour of garlic), paraldehyde and opium (odour of the drugs). Alcohol, ether, chloroform, and other volatile substances are partly excreted by the breath. A **Bad Taste** in the mouth accompanies most of the conditions which give rise to foul breath.

§ 202. **The Saliva** may be *increased* (ptyalism) (i.) in inflammation of the mouth as in stomatitis, and during dentition; (ii.) in chronic gastritis there may be such a profuse flow of saliva during the night that it gives rise in the morning to vomiting of clear alkaline fluid (water-brash or pyrosis). Salivation may occur after a heavy meal, especially with exercise when the stomach is loaded, or after certain foods, such as excess of sugar or sour fruit. (iii.) During pregnancy, in mania, hydrophobia, and some other nervous diseases; (iv.) after the administration of mercury, physostigmine, iodides, bitters, and sometimes alkalies and acids. The saliva may appear to be increased, owing to defective swallowing, in bulbar paralysis, myasthenia gravis, encephalitis lethargica and other paralytic conditions; and with sore throat or other causes of difficult swallowing. "Dry mouth" (xerostomia) occurs with deficiency of saliva. The saliva is *decreased* in dehydration, especially (i.) in certain fevers, (ii.) in diabetes, (iii.) severe diarrhœa, (iv.) chronic nephritis, (v.) after atropine, morphine or stramonium, and (vi.) with emotions of fear or nervousness. (vii.) Sometimes it is associated with calculus of the salivary glands and with old age. (And see § 212.)

Thirst (polydipsia) accompanies all febrile conditions. It is met with also in diabetes, after various causes of loss of fluid, *e.g.*, diarrhœa, perspiration, hæmorrhage, and vomiting, chronic interstitial nephritis, after a diet excessively salted, and with dyspepsia and gastritis.

§ 203. **The Palate** may be "cleft" from childhood, otherwise a hole in this situation is practically always evidence of past syphilis. The *soft* palate shares in the diseases of the fauces (§ 153). It is a favourite position for the membrane of diphtheria, which distinguishes it from follicular

tonsillitis. The *hard* palate is sometimes involved in the diseases of the floor of the nose. A swelling here is commonly due to the presence of pus originating from the lateral incisor, second premolar or first molar tooth, to a gumma or, rarely, to the pointing of an antral abscess.

§ **204. The Teeth** are subject to a certain amount of variation, even in health. The *average* dates of the eruption of the temporary and permanent teeth are as follows:

TABLE XIII.

Temporary or "Milk" Teeth.	*Permanent Teeth.*
6th to 8th month, central incisors.	6th year, first molars.
8th to 10th month, lateral incisors.	7th „ central incisors.
12th to 14th month, first molars.	8th „ lateral incisors.
18th to 20th month, canines.	10th „ first premolar.
2 to 2½ years, second molars.	11th „ second premolar.
	11th to 12th year, canines.
	12th to 13th „ second molars.
	17th to 25th „ third molars.

One quarter of the mouth may be represented diagrammatically thus:

Teeth	I.	I.	C.	M.	M.
Month of eruption.	6	9	18	12	24

Teeth ..	I.	I.	C.	PM.	PM.	M.	M.	M.
Year of eruption	7	8	11	10	11	6	12	24

The normal order of eruption of teeth may be represented thus: MILK teeth, 6, 9, 18, 12, 24 MONTHS; and PERMANENT teeth, 7, 8, 11, 10, 11; 6, 12, 24 YEARS. These details are worth remembering, because defective or deficient teeth are a frequent cause of faulty digestion. Every Mongol has an irregular order of dentition.

Septic teeth, dental caries and pyorrhœa alveolaris are common causes of dyspepsia and serious ill-health. The causes of dental caries are still debated. It is *predisposed* to by some underlying systemic factor and by deficiency of calcification of the tooth substance; to overcome the latter various preparations containing calcium and vitamin D can now be prescribed. The *exciting* cause is apparently the presence of acid-producing organisms which multiply in the food débris around the teeth. Soft, pulpy, sweet and farinaceous foods encourage the growth of these organisms. Hence the importance of adequate cleansing of the teeth after such food. Sim Wallace recommends that every meal should finish with firm raw fruit, such as an apple, which is a natural cleanser of the teeth. Hard foods, which require thorough mastication, act similarly.

The permanent teeth are altered in appearance by constitutional upsets occurring at the time of calcification. They present transverse ridges or lines of pits in the enamel as a result of exanthematous fevers or rickets. Those affected are the incisors, canines or first molars. "Hutchinson's teeth" are due to congenital syphilis—the upper incisors are narrowest at the free edge, which shows a semilunar notch; the molars are dome-shaped, and all the teeth are spaced and liable to caries owing to calcium

deficiency. The face often presents a typical syphilitic facies. The onset of acromegaly and of Paget's disease may sometimes be detected by the alteration of the "bite" of the teeth owing to an unequal increase in the size of the jaws.

§ **205. Toothache** (odontalgia) is produced by acute or chronic inflammation of the tooth pulp or of the periodontal membrane. Irritation of the tooth pulp is due to (1) presence of a carious cavity, (2) exposure of dentine or cementum with or without caries, (3) filling too near the pulp, (4) a blow on a sound tooth. The pain is neuralgic in character, and intensified by extremes of temperature. It ceases on the death of the pulp and is followed by inflammation of the periodontal membrane (periodontitis) due to the passage of septic matter through the apex. The tooth then becomes tender on pressure. Later the gum shows signs of extension of the inflammatory process and the formation of pus. Lymphatic glands draining the area are enlarged and tender and diffuse swelling of the neighbouring soft tissues ensues. Situations in which an alveolar abscess may point are (1) usually in the mucous membrane overlying the affected tooth; (2) the palate, especially arising from lateral incisor, premolar and molar teeth; (3) antral cavity, from any tooth whose roots are in proximity to the antral floor; (4) on the face along the lower border of the mandible from lower premolar and molar teeth.

NEURALGIC PAIN, either local or referred from one jaw to the other, but never across the mid-line, may also be due to impacted teeth, chronic apical abscesses, odontomes, fragments of root remaining after incomplete extraction and to empyema and growths of the antrum. A not uncommon cause of neuralgia is pressure by a lower denture on the mental nerve exposed by extensive loss of bone subsequent to the extraction of heavily infected teeth.

The *treatment* belongs to the dental surgeon. For drops to apply for temporary relief of pain due to a carious tooth see Formula 23. If the tooth is tender on pressure, indicating periodontitis, hot mouth-washes such as carbolic (1–200) are advantageous, with or without the application of counter-irritants such as equal parts of the tinctures of aconite and iodine to the over-lying gum.

For Trigeminal Neuralgia see § 822, and Dental Causalgia § 823.

SWELLINGS OF THE JAWS. *Fluid swellings* are regular and smooth, enlarging the outer wall of the jaw as they increase in size. The most common are (1) acute or chronic alveolar abscesses; (2) odontomes, such as a dental cyst on a dead tooth or a dentigerous cyst on an unerupted tooth. Innocent *solid swellings* include fibroma, chondroma, osteoma and solid odontomes, but sarcoma and carcinoma are often seen.

TRISMUS of local origin may be due to a fracture of the body or the ramus of the mandible, or to the extension of the inflammation from an alveolar abscess or a septic wisdom tooth to the surrounding muscles.

The **Gums.** Examination of the gums gives important clues, apart

from the pallor of anæmia, in the diagnosis of disease. The forms of metallic poisoning are often recognised by the appearance of the gums; in bismuth and lead poisoning a blue line is seen below the free margin of the gums, due to deposit in the gum tissue itself. Various forms of stomatitis accompanying constitutional conditions, such as leukæmia, scurvy, purpura, agranulocytosis, syphilis, are described under *Etiology* in § 210. Localised swelling with a greenish discharge occurs with actinomycosis. Pigmented patches are seen with Addison's disease.

TUMOURS of the gums and mucous membranes are quite common and include: (1) polypus due to local irritation; (2) epulis, usually pedunculated, growing from the junction of the periosteum with the periodontal membrane; (3) papilloma—all of which are treated by excision; and (4) epithelioma and sarcoma; (5) gumma.

Oral Sepsis includes affections of the teeth, gums and alveolar bone.

§ **206. Dental infections** present two different forms: (1) *Closed infection—i.e.*, where there is no drainage and where toxins are absorbed directly by the blood-stream from apical abscesses, granulomata and cysts on dead teeth. This type of infection may be serious because it is unsuspected and revealed only by radiographic examination, there being usually no local clinical signs. It may be responsible for joint, muscle, eye, heart and numerous other lesions. Dead teeth which are apparently normal on X-ray examination are commonly infected and must be regarded with suspicion. When all the teeth have been extracted " residual infection " may persist in the alveolar bone. This can be eliminated in mild cases by the application of diathermy or infra-red rays: in severe cases scraping of the infected areas is necessary. (2) *Open infection—i.e.*, where drainage permits the swallowing of the products of the inflammation, as in cases of broken stumps and carious teeth, infection of the gums, alveolus and mucous membranes of the mouth.

§ **207. Inflammation of the Gums—Gingivitis.**

Symptoms.—The gum margins are slightly swollen, reddened, and bleed easily; they appear to have a smooth, glossy surface. Clinical and X-ray examination of the teeth do not reveal involvement of the periodontal membrane or alveolus.

Etiology.—The commonest cause is lack of oral hygiene, food stagnating round the teeth and gums. Putrefaction occurs, followed by infection. Deposits of tartar act as a predisposing factor. Prolonged administration of mercury, bismuth, arsenic, gold and epanutin are also common causes. It may also be associated with general diseases such as diabetes and nephritis.

Treatment consists in the maintenance of strict oral hygiene. It is essential to clean the teeth regularly and to use floss silk between the teeth to remove débris. Deposits of tartar must be removed from around the teeth by the dental surgeon. The regular use of a warm mouthwash of hydrogen peroxide (2 vols.) promotes cleanliness. Local astringents such as tannic acid or massage with glyc. acidi tannici are

extremely useful. Under normal conditions the disease can be completely eliminated.

§ 208. **Ulcerative Gingivitis** (Vincent's Infection) is due to infection of the gum margins by fusiform bacilli and spirillæ in symbiosis.

Symptoms.—The gums are inflamed and sore, with yellowish marginal ulcers. The breath is offensive and the tongue coated. The onset and spread of the disease are rapid, the tonsils are often involved. Constitutional symptoms may be severe, with pyrexia and enlargement of the submaxillary and cervical glands. The disease is highly contagious and there are often epidemics in institutions. A smear taken from around the gums, when stained and examined microscopically, confirms the diagnosis.



Fig. 63.—Photo-micrograph of Smear of Gum affected by Vincent's Infection, showing Fusiform Bacilli and Spirillæ. (Lent by Dr. Arthur Bulleid.)

Treatment consists in isolating the patient. As the organisms producing the disease are penicillin-sensitive, the lesions disappear within 2–3 days if tablets containing 500 units of penicillin are allowed to dissolve slowly in the mouth every two hours. Meanwhile, no antiseptic mouthwashes must be used, but the mouth may be irrigated with warm saline. When the ulcers have healed any local factors predisposing to gingivitis must be treated, otherwise the disease is likely to recur.

§ 209. **Inflammation of the Gums and Alveolus—Pyorrhœa Alveolaris.**—If untreated, the infection of gingivitis spreads to the periodontal membrane and the supporting alveolus. The bone becomes infected and subsequently absorbed.

Symptoms.—The gum margins are usually engorged and swollen ; there are pockets of varying depth around the teeth from which pus can be expressed. The breath may be offensive and the swallowing of pus may be an exciting factor in the formation of gastric or duodenal ulcers. X-ray examination shows evidence of destruction of the periodontal membrane and alveolus.

Treatment.—When the loss of alveolar bone is not extensive, skilled dental treatment can accomplish much. Removal of all tartar and strict oral hygiene are of primary importance. The gum forming the pockets around the affected teeth is resected. With advanced disease and extensive loss of alveolar bone, extraction of the teeth is necessary, especially if it is suspected that the condition may be producing lesions in other parts of the body. It is unwise to remove many teeth at one operation ; it is often necessary to extract only one at a time. The injection of 100,000 units of penicillin half an hour before the extraction and again an hour later is

often of advantage, especially when the patient has a rheumatic heart lesion.

§ 210. **Inflammation of the whole mouth—Stomatitis.**—This is a widespread inflammation of the mouth, evidenced by redness, swelling, pain, and tenderness of the mucous membrane, swelling and protrusion of the lips in severe cases, offensive odour of the breath, and usually excess of saliva. There are several forms: (*a*) *Catarrhal Stomatitis* is often associated with catarrh of the nose and throat, or with the acute specific fevers. The mucous membrane of the mouth is raised and reddened, and may develop into ulcerative stomatitis. (*b*) *Aphthous* and the allied *Herpetic Stomatitis* form small tender vesicles with a red base and sharply defined circular margin: the vesicles ulcerate and occur especially on the lower surface of the tongue, the lips or on the gums and cheeks. They often recur in crops, especially before the menses. The

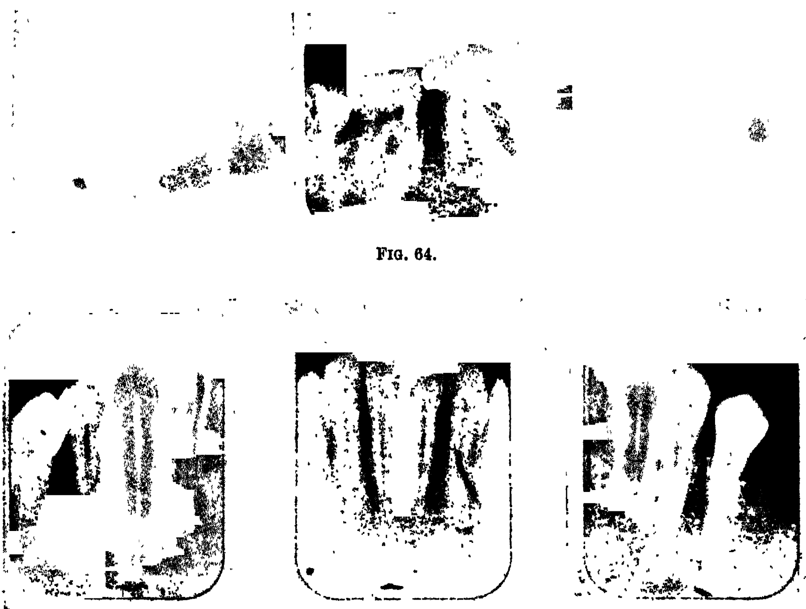


FIG. 64.

FIG. 65.

FIGS. 64 and 65. RADIOGRAPHS to illustrate (Fig. 64) chronic apical abscesses, with destruction of the periodontal membrane and lamina dura around the apices of the teeth, and infection of surrounding bone: (Fig. 65) destruction of periodontal membrane and alveolus around the necks of the teeth typical of pyorrhœa alveolaris.

condition is believed to be due to a virus. *Pemphigus* is distinguished by the formation of blisters, which in the early stages are not surrounded by a red margin, and by lesions elsewhere. (*c*) *Ulcerative Stomatitis* is due to infection with Vincent's organisms from the gums, spreading widely over the mouth. The symptoms are those of ulcerative gingivitis (§ 208), only more severe. (*d*) *Gangrenous Stomatitis* (Cancrum Oris) occurs especially in those suffering from measles and other acute specific fevers. Ulceration, usually starting on the cheek or lip, sometimes spreads to the gums and gives acute pain, but as this passes off a black spot forms (usually both internally and externally), which spreads and leads to perforation of the cheek. There is considerable prostration, and high fever. (*e*) *Parasitic Stomatitis* (Thrush) occurs in infants suffering from gastro-intestinal disorders, and in adults suffering from tuberculosis or other wasting diseases.

The mucous membrane of the mouth is covered with white spots which coalesce to form large areas. The surface epithelium is lost, leaving large red areas (§ 214). (*f*) *Stomatitis* due to *Drugs* and *Chemical Substances* is a more severe stage of gingivitis produced by the prolonged use of mercury, bismuth, gold, epanutin, phenobarbitone, phosphorus, and occasionally arsenic. Mercurial stomatitis, now uncommon, produces fœtor of the breath, with swollen bleeding gums, and later ulceration spreading to the cheeks, tongue, and floor of the mouth. Phosphorus produces ulcerative stomatitis, with necrosis of the jaw. *Electrical action*, due to the presence of dissimilar metals used in dentistry, can cause stomatitis and leucoplakia. (*g*) *Foot-and-Mouth Disease* (Syn.: epidemic stomatitis; aphthous fever) is an acute infectious disease attacking pigs, sheep, cattle, and other domestic animals. Epidemics have been reported in which the disease was transmitted to man, with symptoms of fever, gastro-intestinal derangement and vesicles on the lips, mouth, and pharynx, and sometimes near the nails of fingers and toes. (*h*) With *Espundia*, in S. America, oro-pharyngeal ulceration follows the primary skin lesion, which is due to a type of Leishmann-Donovan body, transmitted by a bug. Tartar emetic is specific for this condition.

Etiology.—(1) LOCAL CONDITIONS, especially faulty dentition, tartar, ill-fitting and dirty dentures, the irritation of a jagged tooth, excessive smoking, dirty feeding-teats, hot fluids and caustics, new growths (simple or malignant), and gummata. In most of these cases the stomatitis takes the form of (*a*) or (*b*) above. (2) CONSTITUTIONAL CONDITIONS: (i.) lowered vitality, met with in tuberculosis and other wasting disorders, or in badly-fed children, in whom the stomatitis may be aphthous, ulcerative, or due to thrush. Epidemics of ulcerative stomatitis have occurred in gaols, hospitals and camps. (3) Certain BLOOD DISEASES (*e.g.*, *scurvy* and *purpura*) are attended by swollen, spongy gums and ulcerative stomatitis. *Acute leukæmia* shows marked stomatitis due to the presence on the gums of small pin-head to sago-grain-sized lymphoid nodules which readily ulcerate. Not infrequently such cases are treated without any suspicion of their true nature, although the nodules in question are very characteristic. The degree of swelling is usually much greater than in lesions due to infection. *Agranulocytosis* (§ 155c) is seen in the mouth, often in the region from which teeth have been extracted a few days previously. *Sprue*, *Pellagra*, *Pernicious Anæmia* and other conditions associated with *Vitamin B deficiency* predispose. *Local streptococcal infection*, especially in women, and certain *skin diseases* such as herpes simplex, lichen planus, lupus erythematosus, erythema multiforme and pemphigus, may be mistaken for aphthous stomatitis, because they show grey streaks or patches. *Syphilis* causes a special variety of the catarrhal form (§ 211).

Prognosis.—Stomatitis is not serious, except the ulcerative type and that form known as cancrum oris, in which the mortality is 80 per cent. Catarrhal and aphthous stomatitis generally end in recovery in a week or two. Cases due to constitutional conditions are usually more serious and obstinate than those due to local or removable conditions. The complications of cancrum oris are diarrhœa, broncho-pneumonia, and gangrene in other parts of the body, especially the organs of generation (noma pudendi); death is usual in seven to ten days. The stomatitis of mercury may be extremely severe, but is rarely seen nowadays. Stomatitis in children, especially the mercurial form, is apt to cause discoloration, pitting and transverse ridges along the permanent teeth which are calcifying at the time. When aphthous stomatitis occurs in adults, accompanying a lingering disease, it is very obstinate, and is, in itself, a very grave omen. The prognosis is grave in epidemic stomatitis.

Treatment.—In all varieties (1) remove the cause, (2) alleviate the local inflammation, and (3) attend to the general health. The teeth should be scaled after acute symptoms have subsided, and any septic stumps removed. After every meal the mouth should be cleaned of débris with a soft brush, by rinsing repeatedly with warm water, then with an antiseptic solution. One of the best is hydrogen peroxide (2½ to 10 vol.) or glyc. thymol. co. (B.P.C.). In aphthous, herpetic, or parasitic stomatitis glycerin and borax gently applied or potassium chlorate as a mouth-wash, are useful; tablets containing formalin may be sucked at frequent intervals, and are of especial use

in children. The ulcers are best treated by touching them with solid silver nitrate or copper sulphate. In gangrenous stomatitis prompt measures are necessary to avert a fatal issue. The affected area should be excised as freely as possible, and plastic operations will be necessary later. Nourishing fluids and the free use of stimulants are called for. Vitamins B and C should be administered freely in all forms of stomatitis.

§ 211. **Other Lesions of the Mouth.**—(i.) *Leucoplakia* (§ 214, II) of the hard palate, tongue and cheeks may occur, usually in syphilitics and heavy smokers. Patches of white epithelium appear on the surface; they begin with sensitive red areas, which soon become white, hard and raised, then fissured and malignant. (ii.) *Syphilis* in any of its stages may affect the mouth: (*a*) the primary lesion may, on rare occasions, show itself on the gums and tongue; (*b*) the mucous patches, secondary lesions, occur on the inner side of the cheeks and the edge of the tongue; ulceration may follow, producing typical "snail-track" ulcers (§ 158). (*c*) The gummata of the tertiary stage, with typical deep, excavated ulcers are sometimes seen (§ 646, II).

THE TONGUE

Apart from the local diseases which may affect the tongue, its appearance aids in the diagnosis and prognosis of certain general diseases. Examination should be made of its surface as regards (*a*) furring, moisture and dryness; (*b*) its colour, and other alterations of the surface; (*c*) the presence of white patches; (*d*) altered size, warts, growths and fissures; (*e*) ulcers; (*f*) note also the method of protrusion. A mother sometimes speaks of her child being "*tongue-tied*" when the frenum is too short: in some cases this is really so, or the structure may be attached to the tongue too far forward, but it exists much less frequently than parents suppose.

§ 212. (*a*) **Furring and Moisture of the Tongue.** In health the tongue is clean and moist, although a slight deposit over its posterior third is not unusual. *Furring* occurs when the greater part of the tongue is covered by a white or greyish layer; when a thick brown and dry crust forms over the surface, the tongue is said to be *coated.* Furring or coating therefore occurs with (i.) local irritation or *sepsis in the mouth*—excessive tobacco smoking, tonsillitis or pharyngitis, dental caries, gingivitis or pyorrhœa. An unpleasant taste in the mouth, or unpleasant breath (halitosis, § 201), may accompany such conditions. (ii.) In most *febrile states* some degree of furring is the rule. Its degree is often in proportion to the toxæmia present and it is therefore a guide to prognosis; with defervescence of fever the tongue cleans. Special importance attaches to the tongue in typhoid fever. In the first week the dorsum is covered with a thin dirty-white fur, but soon the tip and the edges begin to clear so that by the third week the fur has disappeared, and the tongue becomes glazed and dry, or red and smooth. In scarlet fever the fur with the initial tonsillitis rapidly strips, especially from tip and edges, so that by the fourth day there is a bright red raw tongue, with prominent fungiform

papillæ (strawberry tongue, § 477). In measles the tongue is dry and heavily coated at first, but later it peels, leaving a papillated tongue very similar to that of scarlet fever. In typhus the tongue is at first flabby and coated with a thick brown layer; later it becomes extremely dry, often tremulous, and in severe cases dark and shrivelled. (iii.) The condition of the tongue gives much help in the diagnosis of *abdominal conditions*. In acute gastritis and enteritis, the tongue is heavily coated, and is associated with heartburn and an unpleasant taste in the mouth, whereas in chronic gastritis, cirrhosis of the liver, atonic and gouty dyspepsia, the tongue shows a thin white coating, is large, pale and flabby, with a broad tip and indented edges. A red tongue, with sharp red tip and edges, in which the hyperæmic papillæ contrast strongly with the slight white coating in the centre, is found in diabetes and hyperchlorhydria. In acute appendicitis, the tongue is almost invariably furred at an early stage, and later is coated and dry, especially when peritonitis follows. (iv.) *Toxic absorption* usually produces furring. The commonest cause is constipation, which may be "occult." Any bacterial focus, *e.g.*, intestinal obstruction, pyelitis, sinusitis, or chemical poisoning—*e.g.*, chronic alcoholism, chronic arsenical poisoning—act similarly. (v.) *Deficient secretion of saliva* causes a tongue which is dry and often furred. A dry tongue, in the absence of fever, indicates a lack of appetite (except in diabetes mellitus) or a depletion of water, as in diabetes insipidus, after profuse perspiration, diarrhœa (especially cholera) or vomiting. In asthenic states the tongue becomes very dry and coated, *e.g.*, coma, abdominal cancer, advanced phthisis. (vi.) In these extreme conditions a *denuded red tongue* generally follows as the crust falls off—the tongue is red, shining, smooth, dry and often cracked. It is found in the advanced stages of any chronic ailment, and indicates a grave prognosis. Aphthous stomatitis or thrush may supervene. (vii.) A rare condition, *black* or "hairy" tongue, is due to elongation of the papillæ at the back of the tongue; they resemble dark hairs. The hyperplasia of the papillæ permits growth of organisms, usually a streptothrix variety. Discontinuance of tobacco smoking has cured some cases.

§ 213. (*b*) **Other Characters of the Surface of the Tongue.** The *colour* of the tongue is an important indication of the state of the blood. It is *pale* in all anæmic conditions except when the tongue is also inflamed. With the modern use of cosmetics in women, the colour of the tongue is a much more reliable indication of anæmia than is the colour of the lips, cheeks or even conjunctivæ. The tongue is stained *black* when a patient takes iron mixtures. *Blueness* occurs in cyanotic states, and during nitrous oxide anæsthesia. *Excessive redness* occurs (i.) with polycythæmia, (ii.) scarlet fever, typhoid, advanced cachectic conditions, and hyperchlorhydria (see § 212); (iii.) with acute or chronic inflammatory changes (glossitis). In the early stages the papillæ hypertrophy, but later atrophy, and the tongue becomes *smooth* or *bald*. This may occur in local patches; later, the whole tongue is involved and still later

fissuring occurs, from the contraction of subepithelial scar tissue. There may be local *ulceration, tenderness* or *soreness* with streptococcal invasion along the margins, especially with oral sepsis or in association with achylia gastrica, pernicious anæmia, subacute combined degeneration, sprue, pellagra and other allied conditions. Diffuse soreness of the tongue and cheek with no visible lesion may be met in cancerphobia. The "Geographic" tongue and leukoplakia are described in § 214.

The *treatment* of these conditions is to remedy the cause. In persistent furring local conditions are often overlooked and an abdominal cause sought for. It is an old saying that a red tongue requires alkalies and a white tongue acids. A dry tongue indicates either dehydration, or no appetite and deficient gastric secretions, therefore the patient should be fed on fluids, soups, jellies and other foods requiring little digestive power. In painful conditions of the tongue, condiments, acid, rough and irritating foods must be forbidden, and in achylic conditions, hydrochloric acid administered regularly: local painting with silver nitrate 4% is helpful.

§ **214.** (*c*) **White Patches** are not infrequently met with on the tongue, and may result from: I. Thrush; II. Leukoplakia; III. Geographical tongue; IV. Aphthous Stomatitis (§ 210); V. Syphilitic Patches (§ 158).

I. In THRUSH (parasitic stomatitis) there are white membranous patches, like milk curd, sometimes with an areola round them. They are distinguished from other similar affections by (i.) leaving a bright, bleeding surface when scraped off, and (ii.) by the detection of the fungus *Oidium albicans* (Fig. 66) on microscopical examination. It usually starts on the tongue or cheek, but may invade the lips and the whole interior of the mouth and pharynx. The ulceration and the salivation seen with aphthous stomatitis are absent. The disease occurs chiefly in infancy. It generally arises in bottle-fed children under bad hygienic conditions and is often attended by diarrhœa. It is contagious from child to child. In the adult it may occur at the end of wasting disorders. It yields to glycerine and borax, or weak carbolic lotion (1 in 500), but not to penicillin. In children excoriations may be seen around the anus, and the mother thinks the "thrush has gone through the child." Occasionally it attacks the skin of adults, spreading rapidly over groins, abdomen and axillæ; it is usually mistaken for eczema intertrigo, but readily yields to a weak solution of iodine. Rarely, the nails are affected.

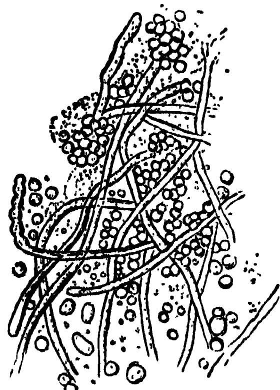


FIG. 66.—OIDIUM ALBICANS OR THRUSH FUNGUS.

II. LEUCOPLAKIA LINGUÆ is a term applied to flat, whitish patches on the tongue. At first the areas are red and sensitive, with hypertrophy of the papillæ; later these atrophy and become slate coloured or white due to a heaping up of the epithelium. The disease may appear in small patches or may involve a considerable area. The patches may also invade the cheeks, gums and palate, and give rise to discomfort and tenderness. This condition is variously attributed to excessive smoking, jagged teeth, drinking strong spirit, and syphilis. Syphilis is the usual cause in cases which show a glazed and atrophic tongue. In 30% of these cases malignant disease supervenes. The *Treatment* is, as a rule, very unsatisfactory, unless the disease be met in the early stages. A mouth-wash, consisting of bicarbonate of soda (1 in 24), or a

saturated solution of chlorate of potash, sometimes relieves the symptoms. Superficial cauterisaton or diathermy fulguration are good methods of treatment; zinc or salicylic ionisation is of value. Antisyphilitic remedies should be tried, but are not often successful. Alcohol, smoking, and other irritants must be avoided.

III. In GEOGRAPHICAL or "Mapped" tongue the normal desquamation of the tongue takes place irregularly, with the formation of more or less circular patches surrounded by margins of slightly proliferating whitish-grey epithelium. Although the cause is unknown it indicates impaired health. It may disappear spontaneously.

§ 215. (*d*) Alterations in the Size of the Tongue. An **Enlarged Tongue** may be due to ACUTE SWELLING, HYPERTROPHY, MACROGLOSSIA and TUMOURS.

ACUTE SWELLING OF THE TONGUE may be due to (I) *Acute Glossitis* or (II) *Acute Œdema*. In both the tongue rapidly enlarges, and may even protrude beyond the teeth. Much pain is present, and difficulty in swallowing and speaking.

(I) ACUTE GLOSSITIS may be due to local causes—*e.g.*, the sting of an insect, streptococcal infection from the teeth or throat, biting or wound of the tongue, acute ulcers or to constitutional conditions—*e.g.*, mercurial salivation and acute specific diseases, such as erysipelas and pneumonia. It may be, like Angina Ludovici (§ 160), of an erysipeloid nature. The onset is rapid, though not so rapid as in acute œdema; the swelling extends to the neck, and the glands become involved. *Treatment* must be prompt, to avert a fatal issue—ice to suck and cold compresses to the neck. Penicillin injections or sulphonamide compounds must be given early. Tracheotomy may be necessary.

(II) ACUTE ŒDEMA OF THE TONGUE is serious, because of its liability to involve the glottis. It may be associated with urticaria and angio-neurotic œdema. The œdema comes on suddenly; in the course of a few hours the tongue may protrude from the mouth. The swelling rapidly extends to the throat, nose, and down the œsophagus and trachea. There is inability to speak, to swallow, sometimes even to breathe. It is *diagnosed* from simple acute glossitis by (i.) its rapid advent; (ii.) the rapid extension to the throat and other parts; (iii.) the presence sometimes of urticaria, or a history of sensitiveness to some article of food (§ 609).

Prognosis and Treatment.—The disease comes on rapidly, and runs a very rapid course, subsiding in the course of twenty-four hours. It is apt to cause suffocation. Prompt measures are necessary. A strong purge or a turpentine enema should be given at once. Adrenalin B.P. should be kept constantly painted on the tongue and injected (0·25 to 1·0 c.c.). Benadryl may be very helpful. The practitioner must be ready to perform tracheotomy if necessary.

HYPERTROPHY AND MACROGLOSSIA. Simple hypertrophy occurs in cretinism, myxœdema, acromegaly, mongolism and with acquired syphilitic lesions. MACROGLOSSIA is a congenital condition of an enormously enlarged tongue due to an overgrowth of the lymphatic, muscular, arterio-venous or neurofibromatous tissues. If persistent application of mild caustics or the galvano-cautery fails to relieve the condition, operation must be resorted to.

TUMOURS of the tongue are rare; for diagnosis and treatment of these a surgical work must be consulted. Overgrowth of the *lymphoid tissue* at the base of the tongue (the "lingual tonsil") is found in local septic conditions and acute blood diseases. Rarely, *thyroid tissue* remains at the base of the tongue as a developmental defect.

A SMALL TONGUE which is tremulous occurs in hyperthyroidism. ATROPHY of the tongue (microglossia) usually arises from nerve lesions (§ 864).

WARTS are simple or syphilitic. *Simple* warts are distinguishable by the fact that they are soft; they are raised, and often pedunculated, and there is but little secretion. The glands are not shotty to the touch. *Syphilitic* warts are hard, with infiltration; they are never pedunculated, secretion is present, and the glands in the neck and elsewhere are shotty.

FISSURES may be simple or syphilitic. The *simple* can generally be accounted for by some such cause as the irritation of a ragged tooth, and are never infiltrated.

On pinching *syphilitic* fissures between the fingers, infiltration is felt. CICATRICES.—Simple ulceration rarely leaves a scar, but if so, it is never hard. Hard, stellate scars invariably indicate syphilis.

(*e*) § 216. **Ulcers of the Tongue** may be Simple, Syphilitic, Malignant, or Tuberculous.

I. SIMPLE ULCERS of the tongue are known by their superficial character, by the presence of some local cause, such as a jagged tooth or other local irritation. They also occur in chronic glossitis and ulcerative stomatitis (§ 210). The *frenum* is apt to be ulcerated in whooping-cough, due to friction against the lower teeth; this is a useful aid in diagnosis.

II. SYPHILITIC ULCERS are of two kinds: (*a*) superficial, (*b*) deep.

(*a*) *Superficial Syphilitic Ulcers* of the tongue are met with usually at the side, or in the form of fissures on the dorsum (*cp.* § 158) or superficial circular "punched-out" ulcers.

(*b*) *Deep Syphilitic Ulcers* are preceded by the formation of a roundish nodule (a gumma) which ulcerates. They are recognised by (i.) their site, which is usually on the centre of the dorsum; (ii.) their raised, ragged, and sometimes undermined edges; (iii.) the yellow slough which covers the base; and (iv.) the fact that they leave deep stellate scars. Syphilitic ulcers are usually multiple; difficulty in diagnosis arises in the case of a single ulcer as to whether it be syphilitic or cancerous. Syphilitic ulceration is differentiated by (1) the relative absence of surrounding induration, and consequently less interference with the movements of the tongue; (2) the dorsal site; (3) less glandular enlargement, and the glands have a shotty feel; (4) the age of the patient, malignant ulcers rarely occurring before forty; (5) little or no pain; and (6) a history of syphilis, a positive Wassermann reaction and the lesion *heals with iodide of potassium.*

III. MALIGNANT ULCER is known by (i.) its site, usually on the side of the tongue; (ii.) its hard, raised, everted edges, and uneven warty base, with foul discharge and tendency to hæmorrhage; (iii.) the induration around, and the early involvement of the glands; and (iv.) the early impairment of the movements of the tongue with great pain. These characters in an advanced case render the diagnosis from syphilis relatively easy. In an early stage the diagnosis may be very difficult. In that stage a cancerous ulcer has flat sloping edges and scanty secretion, *its progress is very slow,* and it does not yield to iodides. Before an ulcer has existed for any length of time, a Wassermann test should be made and a piece excised for microscopic examination.

IV. TUBERCULOUS ULCERS are not common. They are superficial, with a yellowish discharge, usually near the tip, and they only occur in advanced stages of tuberculosis of the lung or throat. The tubercle bacillus may be found in the scrapings and a biopsy is usually confirmatory.

Prognosis.—Simple ulcers are easily dealt with, but other ulcers of the tongue are dangerous chiefly from their liability to hæmorrhage and because of the important structures around. The diagnosis of syphilitic from malignant lesions is as important as it is difficult, for however

advanced the former may be, they yield to appropriate remedies, but the latter are necessarily fatal unless removed early. The deep ulcers often seen in advanced syphilitic glossitis are dangerous, as the deeper parts may be affected by malignant change.

The Treatment consists in removing local sources of irritation. In syphilitic cases, potassium iodide in large doses, and the normal anti-syphilitic remedies must be given. Malignant disease must be treated surgically or by radium.

(*f*) **The Method of Protrusion of the Tongue.**—The tongue usually protrudes evenly between the teeth, and is equally developed in its two halves. In health there may be constant slight deviation to one or other side, of no organic significance. *Tremor* of the protruded tongue occurs in paralysis agitans, general paralysis of the insane, chronic alcoholism, and lead and mercury poisoning. Coarse *jerky movements* are one of the early signs of rheumatic chorea. *Deviation* of the tongue to the paralysed side, forming a sickle-shaped tongue, occurs in hemiplegia or unilateral hypoglossal paralysis. *Failure to protrude* is evidence of an organic lesion involving the nerve supply or the muscles of both sides of the tongue. *Fibrillary twitchings and wasting* should also be looked for (and see § 864).

THE ŒSOPHAGUS

§ 217. **Symptomatology.**—Diseases of the œsophagus have practically one symptom which is common to all—namely, *dysphagia*—*i.e.*, a difficulty in swallowing. It is necessary to distinguish between pain on swallowing and real difficulty due to obstruction. There are certain features which aid diagnosis:

First, does the difficulty apply to both liquids and solids? This gives us an idea of the *degree* of the obstruction. *Secondly*, does the food return? and if so, after what interval? This is sometimes a guide to the *seat* of the obstruction. Obstruction of the *œsophagus* has to be distinguished from obstruction at the pyloric end of the *stomach* (i.) by the easy way in which food regurgitates as compared with the vomiting which accompanies pyloric obstruction; and (ii.) by the absence of acidity or bile or evidence of digestion in the material returned. *Thirdly*, is there any pain? Its situation aids diagnosis of the position of the lesion. Is it present only after the ingestion of food? Constant pain may occur in malignant disease. *Fourthly*, what is the duration of the dysphagia? Has it been persistent, and become progressively worse? The last named is the leading feature of organic, as distinguished from functional, dysphagia, which is frequently intermittent, and by no means progressive. *Fifthly*, is there any regurgitation through the nose? This feature implies paralytic dysphagia, with paralysis of the soft palate. *Sixthly*, is there loss of weight, or any symptom referable to other organs? Emaciation coming on early in a patient beyond middle life is characteristic of carcinoma.

§ 218. **Physical Examination.**—Patients may complain that they have *difficulty in swallowing*, yet the condition may not be true dysphagia.

Thus, for example, tenderness and painful lesions of the mouth, throat and larynx may make it impossible to take solid or liquid food. The hysterical symptom *globus* may be mistaken for dysphagia; the patient complains of a sense of constriction in the throat or high in the epigastrium, or of a "ball rising up in the throat" (§ 888). A careful *inspection* of the throat should be made with and without a tongue depressor and a laryngeal mirror. The dysphagia may arise from tonsillitis or other pharyngeal or laryngeal conditions. Paralysis of the palate which succeeds diphtheria, or the paralysis of the face, tongue and palate in bulbar palsy, may thus be detected. Any swelling should be carefully examined, such as retro-pharyngeal abscess, tumour or foreign body in this situation. A toothbrush bristle in the pharynx can cause serious difficulty in swallowing. In children dysphagia is often due to pain on swallowing; in adults tuberculous laryngitis is a not uncommon cause.

Special Examinations.—In cases of dysphagia a skilled *X-ray examination* is necessary. First, the chest should be examined with the fluorescent screen, which enables one to identify extra-œsophageal causes of dysphagia such as aneurysm, mediastinal tumour, etc. Then an opaque meal is given: a thick emulsion allows more detail to be made out, especially if the patient is lying down or in the Trendelenberg position. On the screen the progress of the meal is watched and any obstruction noted; its characteristics will usually make the diagnosis clear. The use of the bougie is dangerous and is now almost abandoned. It is safer to use a soft stomach tube if anything of this nature has to be carried out. Bouginage should only be performed under direct vision through an œsophagoscope. The œsophagus starts at the cricoid cartilage, opposite the sixth cervical vertebra, and ends opposite a point between the ninth and tenth dorsal vertebræ, a distance of 10 inches.

The *œsophagoscope* is most useful in skilled hands. With it the exact site of the obstruction may be viewed, and when doubt exists as to its nature, a piece of tissue may be removed for microscopical examination. Early œsophagoscopic examination for any œsophageal symptom, however slight, probably offers the best chance in the future for improvement in the results of treatment. The œsophagoscope is also useful for (i.) the removal of foreign bodies, (ii.) the treatment of malignant stricture by the introduction of Souttar's tubes, radon seeds, or radium, (iii.) the treatment by dilatation of non-malignant strictures.

Auscultation affords an additional means of detecting both the presence and position of an œsophageal stricture. Place the chest end of a stethoscope over the interval between the xiphoid cartilage and the left costal arch. Two gurgling sounds can be heard in this situation if the patient swallows *one* gulp of fluid; the first is when it passes from pharynx to œsophagus, the second is when it passes from œsophagus to stomach. The normal interval between these two is *six seconds*, but if there be any obstruction in the gullet this interval becomes increased. If the first sound cannot be distinctly heard, the moment of its occurrence can be judged by looking at the throat. Again, by placing the stethoscope on the left side of the neck in a healthy person a gurgling sound will be heard during the act of swallowing. This normal sound may be *traced round and down the back* on the left side of the

vertebral spines as low as the tenth dorsal vertebræ. But if a stricture be present it will be delayed or *absent below the seat of stricture.*

§ 219. **Causes of Dysphagia.**—*When a patient complains of* DIFFICULTY IN SWALLOWING, *or that the food returns to his mouth, the practitioner should first think of* CANCER, *secondly of* ACHALASIA. The COMMONER CAUSES *are*—

I.	Cancer of the gullet	§ 220
II.	Achalasia of the cardiac orifice	§ 221
III.	A tumour pressing upon the gullet from the outside ..	§ 222
IV.	Cardio-vascular disorders	§ 223
V.	Simple or non-malignant stricture	§ 224
VI.	Foreign bodies, acute œsophagitis, and simple ulcer	§ 225

LESS FREQUENT CAUSES are—

VII.	Paralysis of the pharynx	§ 226
VIII.	Plummer-Vinson syndrome	§ 227
IX.	Diverticulum or pouch of the pharynx..	§ 228
X.	Functional dysphagia	§ 229
XI.	Congenitally short œsophagus	§ 230

§ 220. I. **Malignant Disease** of the œsophagus is in the large majority of cases an epitheliomatous growth in the wall, usually primary, which goes on to ulceration, and forms a stricture from 1 to 4 inches long; or it may be due to extension upwards of malignant disease at the cardiac end of the stomach. Rarely the growth is sarcomatous. It is important to emphasise that any œsophageal symptom, even the slightest, should be carefully investigated. Dysphagia is a late symptom of cancer of the œsophagus. A lumen of 5 mm. is sufficient to swallow chewed food. The favourite sites of malignant stricture are opposite the cricoid cartilage, 6 inches from the teeth (this is especially common in women—post-cricoid carcinoma); opposite the bifurcation of the trachea, 10 inches; and at the lower end of the œsophagus, 16 inches from the teeth. The diagnostic features of epithelioma of the œsophagus are: (i.) The patient is past middle life and is usually a male. (ii.) The dysphagia becomes steadily and progressively worse; in rare cases it may be intermittent. At first a difficulty exists only with solids, but later on fluids will not pass or also are returned. In some cases sudden complete obstruction to swallowing occurs when a large piece of food blocks the narrowed lumen. There may have been no previous symptoms. The duration of the whole illness rarely exceeds 12–18 months. (iii.) Emaciation and other evidence of cachexia occur quite early in the illness, owing to deficient nourishment. (iv.) There is usually no evidence of metastasis, but there may be enlarged glands, especially above the left clavicle. (v.) Pain and sometimes a dry cough may be persistent, independent of although aggravated by food. It may be slight or very severe. (vi.) When the cervical œsophagus is involved, weakness or loss of voice may occur from recurrent laryngeal paralysis. (vii.) A fistula into the trachea or left bronchus or a peri-œsophageal abscess may form. (viii.) X-ray examination may be conclusive. (ix.) Œsophagoscopy and biopsy clinch the diagnosis.

Fibroma and *Myoma*, and other benign growths in the œsophagus, sessile or pedunculated, are very rare. They may cause no trouble, or only vague and trifling symptoms. Their discovery is usually accidental.

§ 221. II. **Achalasia** (often called Cardiospasm) is a condition in which there is long-standing obstruction at the lower end of the œsophagus, and in which simple stricture and new growth can be excluded. The exact site of the obstruction is thought to be at the point where the œsophagus passes through the diaphragm; in this area there is often fibrosis of the œsophageal wall with kinking.

Symptoms.—(i.) Achalasia is usually met with in men from the ages of 30 to 50. (ii.) The food is felt to stick at the lower end of the œsophagus. (iii.) At a variable time after a meal, the food may pass on into the stomach or it may be vomited in an undigested condition. (iv.) On X-ray examination, the barium meal does not pass into the stomach but accumulates above this in a tremendously distended and rather coiled œsophagus. The amount of dilatation is much greater than is found from any other cause of dysphagia. A height of about 8 inches of barium collects above the sphincter, and if still more barium (or food) is taken, the weight of the food is often sufficient for the obstruction to be overcome until the level is reduced to the original height. The *etiology* of the condition is not yet settled; there is degeneration of the cells of Auerbach's plexus of the œsophagus. According to some the obstruction is caused by the crural fibres of the diaphragm, fibrosis of the œsophageal wall resulting in some cases at the region where they press upon it. Achalasia is differentiated from stricture or new growth by passing a heavy tube filled with mercury: this forces the sphincter open, and if the tube be alternately moved up and down, one finds that it is not gripped.

§ 222. III. **Pressure** upon the gullet from outside is a fairly common cause of dysphagia. Any intrathoracic tumour may, by its pressure, narrow the lumen of the gullet. Other tumours are cancer of a neighbouring organ, enlargement of the bronchial glands, lympho-sarcoma or other mediastinal tumour, goitre, pericardial effusion, and diverticulum of the pharynx filled with food (§ 228). The features common to all such tumours are the slowly progressive character of the dysphagia, the symptoms of pressure on other viscera, and the physical signs of the tumour in question. The differential features vary according to the nature and position of the tumour.

§ 223. IV. Cardio-vascular Disorders, which occasionally cause dysphagia, are a saccular aneurysm of the descending aorta, a large left auricle due to mitral stenosis and a large pericardial effusion. The *signs* of these conditions usually determine the cause.

In *aortic aneurysm* the amount of dysphagia is rarely very great at any time, although it is slowly progressive (§ 80). Rest in bed generally improves the dysphagia. The physical signs of aneurysm are commonly absent on account of its deep-seated position.

§ 224. V. **Simple or Non-Malignant Stricture** of the œsophagus is most

frequently caused either by the narrowing due to after effects of swallowing foreign bodies or a corrosive liquid, the cicatrisation which follows a simple ulcer of the œsophagus, syphilitic infiltration or the contraction which it subsequently leaves. Dilatation may take place above the stricture. The differential features of this condition are: (i.) The dysphagia comes on gradually, and, having reached a certain degree, is apt to remain stationary; the patient may be unable to swallow solids, but lives for many years on liquid food. (ii.) The patient may be young, or he may be of any age; the cachexia of cancer is wanting; and pain is not a prominent feature in the case. (iii.) The œsophagus is apt to dilate above the stricture, and the food returns after an interval, which becomes progressively longer as the dilatation becomes greater. (iv.) There is nearly always a history of one of the three causes above mentioned.

§ 225. VI. **Foreign Bodies, Acute Œsophagitis, and Simple Ulcer.**—The symptoms of these conditions are much alike. Acute œsophagitis occurs after traumatism, as after swallowing corrosive fluids, or in a localised form from the presence of foreign bodies or malignant disease. It sometimes occurs in the course of the specific fevers, and in infants at the breast from unknown causes. A slighter degree of *localised* inflammation arises by no means infrequently when a fish-bone, needle, pin, bristle of a tooth-brush, an impacted denture, or other solid particle, sticks in the folds of the œsophagus. This dysphagia takes the form of a difficulty and pain during the act of swallowing, at one particular spot. The symptoms here start suddenly and reach a maximum at once. This source of trouble is very apt to be overlooked when the patient has forgotten the incident which led to the lodgment of the foreign body. When the inflammation is *generalised*, there is great pain, with consequent spasm and regurgitation on attempting to swallow. Thirst and, if the condition be severe, feverishness are present. Mucus, pus, and blood may be vomited should ulceration ensue.

A *Peptic Ulcer* of the lower end of the œsophagus is rare. An ulcer is sometimes due to syphilis. Acute pain and tenderness are prominent features, with spasm on swallowing or on attempting to pass a bougie. The affection can sometimes be demonstrated by X-ray.

We now turn to the **rarer causes of Dysphagia.**

§ 226. VII. **Pharyngeal Paralysis.**—Paralysis of the *pharyngeal constrictors* is not uncommon as an accompaniment and complication of diphtheria. Difficulty of swallowing under these circumstances may be one of the first evidences of diphtheritic paralysis. It also occurs in polioencephalitis, syphilitic pachymeningitis, syringobulbia, bulbar paralysis, polyneuritis and myasthenia gravis. Thrombosis, hæmorrhage and new growth at the base of the brain are other causes. It differs from the other causes of dysphagia in being attended by regurgitation of fluids through the nose, owing to the paralysis of the soft palate. There is often associated paralysis of the tongue. Solids are often swallowed with less difficulty than liquids.

§ 227. VIII. **Plummer-Vinson Syndrome.**—The dysphagia is associated with anæmia, a smooth, bald tongue and pharynx, and usually also achlorhydria. It

occurs in association with the hypochromic anæmia of women and in pernicious anæmia. The condition seems definitely to predispose to post-cricoid carcinoma.

§ 228. IX. **Diverticulum, or a Pouch of the Pharynx.**—(i.) A *pressure* diverticulum forms by herniation of the mucous membrane through the muscular wall. These pouches usually arise in the lowest part of the pharynx, probably from incoordination of the muscles of the pharynx and the cricopharyngeus guarding the entrance to the œsophagus. (ii.) *Traction* diverticulum of the œsophagus, due either to adhesion between the œsophagus and neighbouring glands, or other structures, pulling out the œsophageal wall as they contract. This variety does not usually give rise to symptoms.

The *symptoms* are : (i.) Regurgitation of food after an interval varying from a few minutes to a few hours after ingestion. It is apt to be mistaken for persistent vomiting, but the ease with which the food is returned, and the absence of acid in it, should make us suspect this condition. (ii.) The regurgitation gradually increases in amount, and the breath is sometimes foul from the decomposition of food in the gradually enlarging pouch. (iii.) Sometimes the pouch forms a definite tumour in the neck. (iv.) X-ray reveals the pouch.

§ 229. X. **Functional Dysphagia** is not infrequently associated with hysteria and other functional neuroses. Its differential features are fairly characteristic : (i.) The dysphagia is never progressive. It may come on somewhat suddenly, dating perhaps from an emotional shock or trouble, and it is very often intermittent, the patient being well enough in the intervals. Sometimes solids can be taken, while fluids are regurgitated, or *vice versa.* (ii.) It is unaccompanied by emaciation or cachexia ; indeed, the patient sometimes appears to be in perfect health, a feature in which it differs from all other causes of dysphagia. There is usually little or no pain, and never any bleeding. (iii.) The dysphagia may last intermittently for years. (iv.) The passage of the œsophagocope, or mercury bougie, or flexible stomach tube, generally results in curing the condition, at any rate for a time. (v.) The patient is usually of the female sex, and often presents other evidences of hysteria ; but it occurs also in males. There is often great fear of malignant disease. (vi.) X-ray examination reveals no organic disease.

§ 230. XI. **A Congenitally Short Œsophagus** with partial intrathoracic stomach is a rare cause of dysphagia. The patient has regurgitation, and sometimes discomfort after a meal is relieved in one special position. In some cases the symptoms are those of dyspepsia only. The condition can be diagnosed by X-ray examination of the œsophagus and of the stomach in the Trendelenberg position.

§ 231. PROGNOSIS OF DYSPHAGIA.—Dysphagia is in most cases a symptom of considerable gravity, and in severe cases it often results in death by starvation. Of all causes, malignant stricture is the most serious, and, in spite of the methods of modern surgery, patients rarely live more than a year or eighteen months. The length of time depends on the maintenance of the nutrition of the individual. Next in order of gravity come tumours pressing on the œsophagus, when the prognosis depends on the nature of the tumour and its amenability to treatment. Patients with simple stricture, and with dilatation, may live for many years on fluid diet, with or without gastrostomy. Diverticula may or

may not require operation. Some patients can manage, with a little trouble, for years. Of all cases functional dysphagia is the most curable, although it is apt to return.

The cause of death in dysphagia is usually starvation or a low form of pneumonia. This may arise from perforation into the bronchus or by the food passing into the glottis. In either case death is expedited by the lowered vitality of the patient. Perforation may occur in other directions—*e.g.*, a case of malignant disease of the gullet may die from hæmorrhage consequent upon perforation into the aorta.

Treatment of Dysphagia. The indications are to remove the cause of the obstruction, to maintain the strength and nutrition of the patient and to relieve any concurrent symptoms. In *malignant disease* the introduction of a Souttar's tube through an œsophagoscope may give much relief and the patient may continue to swallow normally for some months, when it may be necessary to introduce another tube. Octyl nitrite inhalations during a meal often make swallowing easier for a time. Treatment by deep X-ray therapy is at present the commonest form of treatment. The insertion of radon seeds or the introduction of radium is often useful. Radical cure, by surgical removal, is being attempted more and more; the mortality of the operation is between 20 and 50 per cent. Deep X-ray therapy and early gastrostomy offer the best chance of prolonging life in every case of malignant stricture. If, when the case comes under treatment, debility is very marked, complications are present, and there are evidences of cancer elsewhere, gastrostomy is the only treatment of any avail. Morphia should be used freely for pain in the terminal stages. In *achalasia*, a heavy rubber tube filled with mercury should be passed just before meals. At first this must be done before every meal, but as the symptoms are relieved, it is required less and less often, until, in most cases, once a week or once a fortnight suffices. Operations are better avoided. In *simple stricture*, bougies of gradually increasing size should be passed under direct vision through an œsophagoscope; force must not be used in so doing. If syphilis be suspected as the cause, anti-syphilitic treatment must be given. *Foreign bodies* in the œsophagus need prompt attention, else they may perforate and produce mediastinitis. With the aid of the œsophagoscope they may be readily removed. In *acute œsophagitis* the pain must be soothed by morphia hypodermically, by cocaine lozenges, or by opium given with tragacanth. Thirst may be allayed with spoonfuls of iced water, in which small doses of opium, cocaine, and milk may be administered. During the acute stage the patient may require nutrient enemata; with the œsophagoscope local treatment can be administered. In *pharyngeal paralysis* the patient must be fed by a nasal tube; semi-solid foods are swallowed more easily than solids or fluids. In the *Plummer-Vinson Syndrome* the anæmia should be treated by massive doses of iron and vitamin B complex or riboflavin, or by liver injections if pernicious anæmia is present. The passage of the œsophagoscope usually relieves the dysphagia. For a

diverticulum, operation may be necessary if the dysphagia is too great. In *functional dysphagia* the passage of the œsophagoscope is helpful. The general condition may be treated; belladonna and valerian are effective in hysteria.

Feeding by a stomach tube is a most useful measure when dysphagia is marked: it is also used for continuous drip feeds in cases of gastric and duodenal ulcer. A rubber tube (size 6) or a latex tube is most comfortably passed into the stomach through the nose and need only be changed each 3 days: milk and milky foods, milk and eggs, chocolate, etc., may have malt or sugar added to increase the caloric value.

CHAPTER IX

THE ABDOMEN

THE abdomen contains a large number of very important organs and structures, but just as their physiology and pathology are in some instances obscure, so also are the means at our disposal for their thorough clinical investigation imperfect. It is in this region that we have to deal with symptoms which on the one hand may be of trivial order, or on the other of extreme gravity; symptoms and conditions the issue of which will largely depend on the promptitude, knowledge, and skill of the medical practitioner and upon his adequate comprehension of their true meaning.[1]

PART A. SYMPTOMATOLOGY

§ 238. **Local Symptoms.**—The symptoms referable to disease situated within the abdomen are necessarily of the widest and most varied kind, but there are only three which are sufficiently constant to be regarded as cardinal symptoms, all of which are referable to the abdomen itself—viz., ABDOMINAL PAIN, GENERALISED ENLARGEMENT, and LOCALISED TUMOUR.

VOMITING is a fairly constant accompaniment of all acute abdominal conditions, whether the stomach is involved in the lesion or not. Its causes are discussed in § 271.

The presence of DIARRHŒA and CONSTIPATION depends very largely on whether the intestinal canal is affected, and these are fully dealt with in Chapter XI. The other symptoms also depend largely upon which of the abdominal organs is affected, with one important exception—viz., " INDIGESTION." In all chronic abdominal disorders, no matter which organ is affected, we are often consulted for " INDIGESTION " ; in fact, nausea and all the other symptoms of pronounced dyspepsia may be due to disease quite unconnected with the stomach, and located, for instance, within the uterus, appendix, gall-bladder, colon, kidneys, prostate, liver, lungs, pancreas or other organs.

ABDOMINAL PAIN, if acute and sudden, is a medical emergency of the most important kind ; if chronic, it presents many difficult questions for diagnosis. It therefore merits the most careful study and analysis (§ 242). The diseases *outside the abdomen* which may cause it are :

1. *Diaphragmatic pleurisy*, or a basal pleuro-pneumonia, may give rise to acute abdominal pain of sudden onset (often referred to the correspond-

[1] Although in one particular patient there is usually only one pathological process at work, it must not be overlooked that a patient with tabes dorsalis may also be suffering from a perforated peptic ulcer, or that a patient with pneumonia does occasionally develop acute appendicitis at the same time.

ing iliac region), and to abdominal rigidity and other symptoms of acute peritonitis, which can only be differentiated by the pulse-respiration ratio and the concurrent symptoms.

2. *Root pains* from the spinal nerves may be referred to the abdomen. In this way spinal caries (especially in children), a spinal tumour, or the crises of locomotor ataxy may be mistaken for various abdominal diseases.

3. An *abscess* in the abdominal wall, a bruise, or a ruptured muscle may be similarly mistaken, but these should present no difficulty. *Fibrositis* of the abdominal wall has led to mistaken diagnoses of appendicitis and ovaritis, or of the diaphragm to confusion with an upper abdominal lesion.

4. *Diabetic coma* is occasionally heralded by pain simulating appendicitis.

5. *Paroxysmal tachycardia*, *pericarditis* and *coronary thrombosis* have been mistaken for a condition requiring laparotomy.

ABDOMINAL ENLARGEMENT and ABDOMINAL TUMOUR are considered in Part C.

The **General** or **Remote Symptoms** met with in abdominal disorders are, as just mentioned, of an extremely varied nature, and our endeavour should be to associate correctly these symptoms with the abdominal organ which is affected.

§ 239. **Shock** (or **Collapse**) is a frequent general symptom in acute abdominal disease; it is a condition of extreme prostration. Shock and collapse are clinically identical. There is paresis of all the muscles, voluntary and involuntary (muscles of the limbs, of respiration, of the heart and arteries), and a rapid fall in blood pressure. The *Symptoms* may be arranged under the following headings: (1) The skin (especially of the extremities) is pale, cold, and clammy; the surface temperature is 2° F. or more under normal; the pupils are dilated, and react slowly to light. (2) The circulation and respiration are feeble, the pulse being rapid, of low volume and often scarcely perceptible. (3) The temperature is sub-normal. (4) Restlessness, air hunger and marked pallor are present in shock accompanied by profuse hæmorrhage. (5) There is apathy, but the intellect is clear. The urine and other secretions are diminished or suppressed. The patient may die, or may pass into a reaction stage, with slight pyrexia and sometimes vomiting.

Diagnosis.—In *coma* the mind is completely obscured, and the respiration laboured and stertorous. Save for the vital functions, all is in abeyance. (See § 711 *et seq.*) In *syncope* consciousness is generally lost, but the condition is transient.

The *Causes* of shock may be divided into those of sudden and those of gradual onset. The depth of shock varies with the causative lesion.

Surgical shock is frequently divided into primary and secondary stages: the former comes on rapidly and is believed to be due to afferent nervous impulses acting on the brain, producing paresis of the vaso-motor centre and dilatation especially in the splanchnic vessels. Secondary shock is much more insidious and more dangerous: it is the result of loss of blood plasma through the capillary walls, often in association

with pooling stagnant blood in a dilated capillary bed: later it is followed by absorption of toxic products from the site of injury.

(*a*) OF SUDDEN ONSET (often due to primary shock). These may be subdivided into: (1) Those due to *external injury*. (i.) Traumatic shock such as gunshot wounds and accidents. The amount of shock varies, especially with the extent of hæmorrhage, and to a less extent with the amount of injury. (ii.) Fractures of long bones produce an amount of shock out of proportion to the apparent injury. (iii.) Severe burns, especially on the trunk. (iv.) Head injuries with concussion. (v.) From electrical currents. (vi.) Certain narcotic poisons (hydrocyanic acid, carbon monoxide § 561). (2) Those due to *internal causes*. (i.) Profuse internal hæmorrhage as with hæmatemesis, ruptured ectopic gestation. (ii.) Perforation of an abdominal viscus with extravasation of its contents into the peritoneum. (iii.) Rupture or torsion of an abdominal organ. (iv.) Very severe acute pain, as with renal or biliary colic. (v.) Sudden intestinal obstruction. (vi.) Pulmonary or other embolism. (vii.) A large spontaneous pneumothorax. (viii.) Coronary thrombosis. (ix.) Cerebral hæmorrhage. (x.) With acute pancreatitis or acute suprarenal hæmorrhage (§ 244).

(*b*) OF GRADUAL ONSET: (i.) Peritonitis and other abdominal inflammations. (ii.) Delayed hæmorrhage from trauma on the 7th–10th day, *e.g.*, from a ruptured liver, spleen or kidney, and following sudden movement or even an enema. (iii.) Profuse diarrhœa and vomiting. (iv.) Sudden and severe emotion (terror, grief, etc.). (v.) Privation and exposure to extremes of heat and cold. (vi.) Sea and air sickness. (vii.) Blast and crush injuries associated with renal failure. Other *toxic causes* include (viii.) Post-anæsthetic and post-operative shock. (ix.) An overdose of hypnotic and anæsthetic drugs. (x.) Poisoning by irritants (oxalic acid, arsenic, phosphorus). (xi.) Food poisoning. (xii.) Anaphylaxis (§§ 521, 609). (xiii.) The asthenic types of fever such as may attend typhoid and yellow fever. (xiv.) The termination of many diseases described in the chapter on debility.

Diagnosis.—When a patient is found in a state of collapse or shock, the physician has to diagnose the *cause* of the condition. After applying restoratives he should inquire: first, whether there is a history of injury, hæmorrhage, or emotional disturbance, etc.; secondly, if the patient was in good health up to the time of onset of the condition, so as to exclude group (*b*); thirdly, what food the patient has recently taken, remembering the possibility of poison. Finally, he should examine all the viscera, especially the heart and abdominal organs, beginning at the part which is or has been the seat of pain.

Etiology.—The main factors producing shock vary from case to case, and with the cause. The most important are: (i.) hæmorrhage: (ii.) circulatory failure: (iii.) severe pain: (iv.) prolonged exposure: (v.) dehydration: (vi.) the mental reactions of the patient, including loss of consciousness: (vii.) the absorption of toxic products.

The immediate *Treatment* consists in dealing with the cause: *e.g.*, blood loss must be stopped, an injured or fractured limb immobilised. When pain is severe this should be combined with an injection of morphine, and by the application of warmth with hot-water bottles and warm blankets, or by an electric cradle. The head should be lowered, the feet raised and even the legs bandaged following severe hæmorrhage. In mild cases, and if there is no abdominal injury, brandy may be given by mouth. Stimulants such as injections of nikethamide (coramine) 1–2 c.c., leptazol (cardiazol) ½–1 c.c., and adrenalin may be repeated. In severe cases isotonic blood serum or blood plasma should be given in large quantities (½–2 litres) intravenously. When blood loss has been marked, transfusion of whole blood (½–2 litres or more) is essential. In hot climates, and in the presence of dehydration, these may be supplemented by isotonic gum-saline or dextrose (5·0 per cent.) intravenously, or normal saline or isotonic glucose per rectum. Only after recovery from primary shock, and when the fall of blood pressure has been corrected, should the patient be operated on.

Shock (Collapse) and Pulse-Temperature Ratio.—In connection with the general symptoms of abdominal diseases, two facts need special mention—(1) Profound primary shock is common at the onset of acute abdominal conditions. A subnormal temperature is one of the symptoms of shock, and for this reason it is often present in the early stage of abdominal trouble, and it rarely ranges very high even in the gravest abdominal conditions. In acute peritonitis, for instance, an extensive inflammatory process affects the peritoneum, which acting alone might produce a temperature of 105° F. or more, but by reason of the secondary shock it is rarely more than 102° or 103° F. (2) *In the pulse,* however, *we find our best guide to the severity of mischief within the abdomen.* In all acute diseases, other than abdominal, we find a rough general proportion between the height of the temperature and the rate of the pulse. Thus, a temperature of 100° F. will correspond roughly with a pulse of 100, 101° with 110, 102° with 120, 103° with 130, and so on—an increase of about 10 for every 1° F. But in acute abdominal conditions this is not so. The pulse-temperature ratio is disturbed, for although the pulse rate increases with the severity of the abdominal condition, the temperature never increases proportionately. Indeed, in many of the worst cases, the temperature is one or more degrees below normal. The pulse, however, is usually a good guide, and one may say (1) that if the pulse remains under 100 nothing very serious is happening within the abdomen; and (2) that the rate of the pulse and the pulse-temperature ratio are great aids to the diagnosis, and in some sense measures, of acute abdominal disorder, especially when that disorder has reference to the peritoneum. In assessing a patient's reaction due regard must be paid also to the effects of anxiety on the pulse rate.

PART B. PHYSICAL EXAMINATION

§ **240.** In the examination of the abdomen we must proceed systematically, as in the examination of the thorax, by INSPECTION, PALPATION, PERCUSSION, MENSURATION, and occasionally auscultation; though of all these measures palpation by the educated hand is at the present time the most valuable means we have. X-RAYS assist in certain cases.

1. CAREFUL INSPECTION OF THE ABDOMEN should on no account be omitted; much can be learned in this way. The best point of view is that from the foot of the bed, or by bending over the patient's feet, so as to view the abdomen from below. The mere fact of *enlargement* may thus be verified, and whether the enlargement be generalised and uniform, or whether it be localised or asymmetrical. Notice whether the umbilicus is centrally situated, and also whether the surface presents dilated veins, such as occur in abdominal cancer, or when the portal vein or inferior vena cava is obstructed. Dilatation of the abdominal veins is met with chiefly in three conditions: (1) In liver cirrhosis, these veins being part of the collateral circulation which gradually becomes established (§ 260); (2) the veins, without being much dilated or prominent, are unduly apparent in cases of abdominal carcinoma. It is a sign of considerable value and constancy. (3) Extreme dilatation and varicosity of the superficial veins occur only when the inferior vena cava is obstructed. This is generally due to a gummatous deposit in or around the posterior border of the liver where the vena cava passes through it. The veins of the legs and testes generally share to a less extent in the dilatation. Notice also whether there is any fistula, thickening or infiltration round the umbilicus such as may occur in cancer and tuberculous peritonitis. An abdominal enlargement due to the presence of air or gas is rounded anteriorly, but when due to fluid it is usually flattened in front and the flanks bulge; when there is obstruction of the large intestine the flanks bulge; whereas in obstruction of the small intestine low down the swelling occupies the centre of the abdomen. Incidentally you may notice the presence or absence of the white lines (lineæ albicantes) left by a previous pregnancy, and of scars left by a previous operation. The presence of a hernia or of tumours of the wall (increased by coughing) may be recognised. The amount of *movement* of the abdominal wall with inspiration should be noticed, for diminished or absent movement constitutes an important sign of general peritonitis. With local peritonitis, the abdominal wall over that area may not move, whilst elsewhere abdominal respiratory movement is normal. Pulsation seen in the epigastrium is often normal, but may be due to the right ventricle or an engorged liver secondary to heart failure. Sometimes aortic pulsation is unduly visible, especially in thin neurotic dyspeptic women, or it may be transmitted by a pyloric tumour lying over the aorta. Rarely the pulsation is due to an abdominal aneurysm. *Visible peristalsis* should be looked for and should be provoked by gently flicking the abdomen or, in the case of a child, giving a feed; if present, its position and direction should be noted.

The Regional Anatomy of the Abdomen is important as a guide to the seat of disease (Fig. 67).

2. Palpation.—With practice, experience, and a knowledge of anatomy a great deal can be learned by careful palpation. The hand should be warm, otherwise the patient may flinch. Palpation may be (*a*) superficial and (*b*) deep. Superficial palpation should be carried out first; test for hyperæsthesia by picking up a fold of the skin and subcutaneous tissue from each of the four quadrants in turn. If hyperæsthesia is present the patient may complain of soreness, or show pain by his expression. Hyperæsthesia of the underlying segments of the abdominal wall is revealed by repeatedly stroking the overlying skin; where it is present the reflex is brisker and is maintained for a longer time than on the normal side. For deep palpation the hand should always be laid *flat* on the abdominal wall; then by gently dipping the fingers, by flexing the metacarpo-phalangeal joints, we ascertain (1) the presence of any tumour; (2) the boundaries of some of the solid organs. Bimanual palpation should be employed in feeling the kidneys, spleen, and pelvic organs. The patient should lie on his back with the knees drawn up and the head and shoulders supported, so as to relax the abdominal muscles. Do not use the tips, but only the pads of the fingers, for the tips stimulate the recti muscles to contract, and thus to simulate a tumour where none exists. Many patients offer considerable involuntary resistance; this must be overcome by placing them in an easy posture with the knees flexed, and distracting their attention, or asking them "to let the breath go" or to breathe deeply and regularly. Relaxation is obtained in others by an anæsthetic such as gas and oxygen or soluble hexobarbitone B.P. (evipan) in adults, and ethyl chloride in children. Much obesity is another obstacle to palpation. Palpation reveals the presence of localised resistance and tenderness which denote underlying inflammation, but it must be remembered that in severe toxæmia this reflex rigidity may be very slight. Tumours and flatulence are detected by palpation; the movement of fluid within the abdomen conveys a thrill (§ 259). The palpation and percussion boundaries of the different organs are described in later chapters.

3. Percussion of the abdomen is carried out with the same precautions as in the case of heart and lungs, and the student will now find it convenient to be able to percuss with either hand. The liver and spleen give a dull note on percussion. The full bladder or an ovarian cyst is dull with a horseshoe-shaped area of resonance above it. By this means we ascertain the presence of solid and fluid, which are dull, or of gas, which is resonant. When the fluid is free the dulness alters with the position of the patient and gives a percussion wave or thrill.

4. By measurement we ascertain the amount of increase or decrease in size. As a general rule, horizontal measurement should be taken at the level of the umbilicus, and it should be recorded for future reference. In order to ascertain whether the enlargement is symmetrical, we measure from the umbilicus to the ensiform cartilage above and the pubis below,

and from the umbilicus to the anterior spine on each side. These four measurements should be approximately equal. From these data we ascertain slight deviations from symmetry.

5. AUSCULTATION and AUSCULTO-PERCUSSION are useful in certain cases: one can thus hear peristaltic movements or gas gurgling through

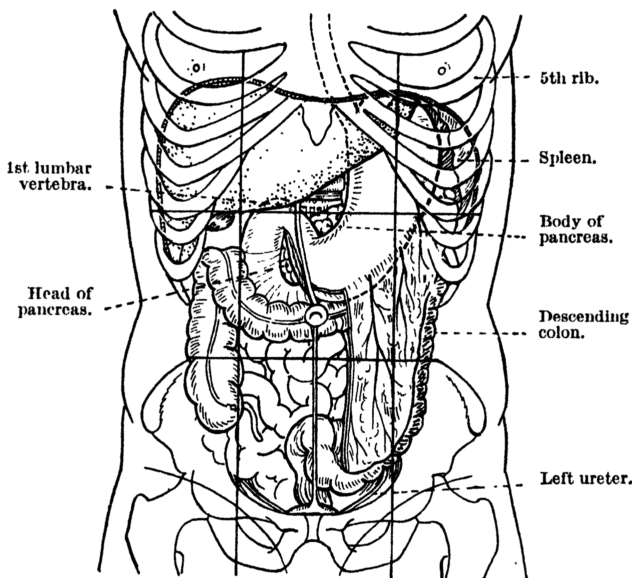


FIG. 67.—REGIONS OF THE ABDOMEN.

For purposes of convenience the abdomen is divided into nine regions. These are bounded by (*a*) two imaginary lateral vertical lines running upwards from the mid-point between the symphysis pubis and the anterior superior iliac spine below, to the ribs above; and (*b*) two imaginary horizontal lines. The upper lies mid-way between the symphysis pubis and the suprasternal notch (transpyloric plane running through the first lumbar vertebra and usually coinciding with the tips of the ninth costal cartilages): the lower crosses at the level of the iliac crests.

Their names and the organs they contain are as follows:

Right Hypochondriac.	Epigastric Region.	Left Hypochondriac.
The right lobe of the liver and the gall-bladder, upper part of the right kidney, and the right suprarenal.	The left lobe and lobulus Spigelii of the liver.	Part of the fundus and body of the stomach, the spleen and tail of the pancreas, the splenic flexure of the colon, upper half of the left kidney and the left suprarenal.
Right Lumbar.	*Umbilical Region.*	*Left Lumbar.*
The ascending and proximal part of the transverse colon, lower part of the right kidney, and some convolutions of the small intestine.	The middle and pyloric end of the stomach, the first, second and proximal portion of the third part of the duodenum, the head and body of the pancreas, the middle of the transverse colon, part of the great omentum and mesentery, and some convolutions of the jejunum and ileum.	Descending colon, part of the omentum, lower part of the left kidney, and some convolutions of the small intestines.
Right Iliac.	*Hypogastric Region.*	*Left Iliac.*
The cæcum, ovary and ureter.	Convolutions of the small intestines and the bladder in children and in adults when distended, the appendix, the pelvic colon, and the uterus during pregnancy.	Sigmoid flexure of the colon ureter and ovary.

a sphincter (*e.g.*, at the cardia or the ileo-cæcal valve); and can delimit the boundaries of an organ. Friction may be heard over liver or spleen in some cases of peritonitis and with embolism of the spleen.

6. A RECTAL EXAMINATION should always be made.

7. EXAMINATION with X-rays (with a barium meal or enema, or by cholecystography or pyelography): or the skilled use of a sigmoidoscope, cystoscope, gastroscope and œsophagoscope may assist in obscure and in chronic cases.

The FALLACIES of abdominal enlargement are: (1) *Fat in the omentum.* (2) *Phantom tumour.* See § 262. (3) *Pendulous abdomen,* so frequent in elderly women, is often thought by the patient to be a "tumour," but is due to weakness of the muscles. (4) In *rachitic children* the liver and spleen may be pushed down by the deformed costal arches, and so produce the appearance of an enlarged abdomen. (5) Extreme lordosis (and see § 262).

PART C. ABDOMINAL DISORDERS: THEIR DIAGNOSIS, PROGNOSIS, AND TREATMENT

§ 241. **Routine Procedure and Classification.**—Having *first* ascertained that the patient's leading symptom is one of those above referred to (§ 238), we *secondly* inquire into the history, and especially whether the condition came on acutely and suddenly, or is chronic and long-standing. The procedure to be adopted in acute cases, and in chronic cases, is given under their respective headings. *Thirdly,* proceed to the physical examination of the abdomen, the routine method in ordinary cases consisting of (1) Inspection; (2) Palpation; (3) Percussion; and (4) Mensuration. In any doubtful case the rectum, vagina, hernial orifices, urine, and fæces must be examined. The fallacies mentioned in § 240 must be borne in mind.

If **severe abdominal pain,** which came on **suddenly** and acutely, be the leading symptom, first turn to § 242.

If **abdominal pain** of some duration and running a **chronic** course be the leading symptom, turn to § 249.

If there be a **generalised abdominal enlargement,** turn to § 257.

If there be **localised tumour,** turn to § 263.

§ 242. **Acute Abdominal Pain,** coming on **suddenly,** or supervening on chronic abdominal pain, includes amongst its causes some of the most serious conditions with which a physician or surgeon can have to deal.

The *causes* of abdominal pain may be conveniently classified thus:

A. ABDOMINAL PAIN coming on **suddenly, with shock.**

I. Perforation of some organ or cyst (perforative peritonitis) §§ 243, 244
II. Internal hæmorrhage .. .. § 244*a*.
III. Acute intestinal obstruction (strangulated hernia, intussusception, internal strangulation, volvulus and paralytic ileus) .. §§ 244, 319
IV. Torsion of ovarian cyst; V. embolism of the mesenteric artery; VI. acute pancreatitis .. .. § 245

B. ABDOMINAL PAIN coming on **suddenly, without shock.**

In the first six the acute abdominal pain is usually ATTENDED BY SHOCK, but not in the last four. This, however, is only relative, and in any doubtful case the whole should be passed in review.

In order to ascertain which of these causes is in operation, and in view of the gravity of some of these cases, it will be desirable to consider the METHOD OF PROCEDURE in some detail.

1. Regarding the *cardinal* or *leading* symptoms, inquire carefully, as in all cases of "pain," concerning its position, character, duration and intensity. The position of the pain is not always a guide to the organ affected, for it rapidly tends to become generalised; but the direction in which it is referred is of great help in the diagnosis of the four kinds of colic. Moreover, local disease may be accompanied by generalised pain (which may later settle down locally), and widespread disease may give rise to a localised pain. Inquire about the mode of onset of the pain, its severity, its duration, its recurrence, its radiation, what relieves or aggravates it; also what other features are usually associated with it. Whenever the three symptoms, ABDOMINAL PAIN, VOMITING, and SHOCK, come on together suddenly, the condition is very probably due to PERFORATION (which will later be accompanied by PERITONITIS), INTERNAL HÆMORRHAGE or INTESTINAL OBSTRUCTION.

2. As to the *History of the Illness*, it is useful to note if there had been any illness or operation previous to the onset of the pain pointing to ulceration, dyspepsia, or other derangement of the abdominal organs. The occupation may shed some light on the cause—*e.g.*, sudden strain, working with lead. The description of the mode of onset may assist—*e.g.*, "something was felt to give way," and it should especially be noted whether the pain was acute at its onset or whether it worked up to a climax later.

3. In the *Examination of the Patient*—(i.) the *age* is an important aid in the diagnosis of the cause of the pain. In childhood there is very probably some intestinal affection, such as enteritis or colic, intussusception, strangulated hernia, or a congenital abnormality; in adolescents and young adults, appendicitis may have to be considered. In adults we think of hernia, ulcer of the stomach and tabetic crises; after middle life and in old age we think of cancer, volvulus or diverticulitis. (ii.) The *sex* may aid us, for in young females we may suspect an ulcer of the stomach even without previous symptoms; and in older women, biliary colic, salpingitis, torsion of an ovarian cyst, the rupture of an ectopic (extra-uterine) pregnancy, frequently overlooked, or gall-stones. (iii.) The presence of *rigidity*, as shown by resistance to palpation, or of *tenderness*, is

of considerable aid; they point to the existence of underlying inflammation. (iv.) *All the organs* of the abdomen must be as carefully and as thoroughly examined as circumstances will permit. Never forget to examine per rectum and vagina, because local tenderness, a pelvic abscess, hæmatoma or tumour may throw considerable light upon the case. (v.) The patient's *general symptoms* must also be carefully investigated. If the temperature *and the pulse* be normal, we may exclude inflammatory conditions. The temperature alone is not a sufficient guide in this respect (see § 239), but in general terms no serious acute abdominal condition exists without the *pulse rate* exceeding 90 or 100. If the patient is much emaciated, in adults we must bear in mind malignant disease, and in children the presence of tubercle. Examine the tongue for dryness and furring: the urine for sugar, crystals, or pus: and do not forget to examine the chest (see § 238).

If the pain, which is severe and has come on suddenly, is **attended by marked shock,** first turn to § 243. If it is **unattended by shock,** turn first to § 246. It must be remembered, however, that any severe pain will cause a certain amount of prostration.

GROUP A. I. *The patient complains of very severe* **abdominal pain,** *which has come on* **suddenly,** *followed by* SHOCK *and repeated* VOMITING *of small amounts. Later,* ABDOMINAL DISTENSION *develops. The case is one of* PERFORATION *with* PERITONITIS.

§ **243. Perforation of the Alimentary Canal, or Rupture of an Abscess, Cyst, or a Solid Organ** (which shortly develops into Perforative Peritonitis). (1) *Ulcers* of the stomach or duodenum are especially liable to perforate. Other ulcers which may perforate are: ulcer of the lower part of the ileum (due to tuberculosis or typhoid fever), ulcer of the cæcum, ulcer of the large intestine, especially the sigmoid (usually cancerous, dysenteric, or syphilitic, or from diverticulitis). (2) *Abscesses* of the appendix (§ 247), liver, gall-bladder, kidney or other organs, or of mesenteric glands. (3) *Cysts which may rupture* are hydatid or simple cysts of the liver, kidney, pancreas, or other organs, ovarian and parovarian cysts. A ruptured bladder produces similar symptoms. (4) *Rupture of an organ* may be followed by internal hæmorrhage (§ 244*a*) and causes similar symptoms.

The *immediate symptoms* of the perforation in the order of occurrence are (1) very severe sudden abdominal pain, which is the cardinal symptom, accompanied by (2) primary shock, with an ashen pallid face showing a cold clammy sweat; the temperature is subnormal and the pulse of low volume: then (3) vomiting occurs. As the patient recovers from the initial shock the inexperienced physician may be deceived until the *symptoms* of *general perforative peritonitis* set in; (4) the pain remains severe and becomes generalised; (5) toxæmia produces a condition of secondary shock with rising temperature and pulse rate; (6) vomiting of small quantities becomes incessant. Later the material becomes alkaline to litmus and even fæcal; (7) the eyes become sunken and the tongue furred and dry; (8) there is board-like rigidity of the abdomen and, a

little later, (9) constipation and moderate abdominal distension from paralytic ileus ; (10) the blood shows a marked and progressive leucocytosis.

The commonest causes are acute perforative appendicitis and perforation of a peptic ulcer. This latter may be taken as a type. We should inquire for a history of dyspepsia and other symptoms (§ 287), but in not a few cases rupture has occurred without previous symptoms of any kind whatever. On examination the thighs are flexed, there is tenderness, a board-like rigidity of the muscles, most marked in the epigastrium, and a tympanitic note over the whole abdomen. The disappearance of the liver dulness in the mid-axillary line denotes free gas, and is usually due to ruptured peptic ulcer. After a few hours there is a deceptive *latent period* during which all symptoms of discomfort are diminished. A *stage of reaction* occurs several hours later, when symptoms of secondary shock are found, with acute peritonitis (§ 244), generalised or localised. There is increased abdominal distension, vomiting and tenderness, with decreased rigidity and a rising pulse-rate. In a perforated duodenal ulcer the pain may spread to the right iliac fossa, simulating appendicitis. Three degrees of severity occur with perforation : (*a*) When there are adhesions the peritonitis may be localised or partial ; (*b*) when there are no adhesions, but a small leakage, it may be only moderately sudden in its onset ; (*c*) when the leakage is large it is extremely sudden and severe in its onset. In typhoid fever the symptoms and signs of perforation in the third week may be few (see § 493).

Perforative peritonitis may have to be diagnosed from diaphragmatic pleurisy and basal pneumonia, in which the pulse-respiration ratio is disturbed, but not the pulse-temperature ratio, and from tabetic root pain.

Treatment and Prognosis.—Laparotomy should be performed at once. If the deceptive latent period leads one to believe the patient is recovering, in a few hours general peritonitis will have set in, and operation is indicated, with or without drainage. In cases where patients have been operated upon within the first twelve hours the prognosis is good ; if after twenty-four hours, it is serious. The after-treatment depends on the cause. In the case of rupture consequent on injury internal hæmorrhage may take place with a rapidly fatal result, but even in such cases early laparotomy and blood transfusion should be performed.

§ **244. Acute Peritonitis** is an acute inflammation of the peritoneum. It is rarely a primary disease, but its onset is usually sudden, following on perforation.

Symptoms.—(1) The aspect is very characteristic ; the countenance has an anxious pinched look, the cheeks pale, and the skin cold and clammy. The posture of the patient is very characteristic, as he lies on his back with legs drawn up to fix the abdominal muscles. (2) The pain is severe and constant, but liable to exacerbations on account of the intestinal peristalsis and the passage of wind along the bowel.[1] It is

[1] Acute peritonitis, which complicates typhoid fever, is of a latent character, and unaccompanied by pain. This and puerperal peritonitis are the only exceptions.

increased by any movement, even by the respiratory movements; consequently the respiration is thoracic; (3) vomiting is persistent. (4) There is acute tenderness on pressure, so much so that the weight of the bed-clothes can hardly be borne. (5) The abdomen is rigid and immobile. (6) Pyrexia, often ushered in with sudden rigors, and attended by a small, wiry, rapid pulse of 100 to 140 per minute. The temperature is elevated only 2° or 3° F. above normal, and maintained there continuously, unless pyæmia be present, in which case there are rapid variations of wide range. In some cases—*e.g.*, perforation—it may be subnormal at first (*vide supra*). (7) Leucocytosis is found. There is marked prostration, as in all abdominal inflammations, and a great tendency to secondary surgical shock. Even from the beginning there is constipation: hiccough is often present, and if persistent it is a bad sign, as in all abdominal disorders. There is diminution and even suppression of urine. Death occurs from toxæmia, and the mind remains quite clear until the end in uncomplicated cases.

In acute localised peritonitis the symptoms are those of acute general peritonitis, but are less severe, and result in the formation of a localised abscess.

The *Causes* of acute peritonitis may be grouped under seven headings: (i.) *Acute appendicitis* is the most common (§ 247). There may be extension of inflammation from other organs in the abdomen—*e.g.*, diverticulitis, gonorrhœal salpingitis, inflammatory conditions of the intestine (typhoid, dysenteric and actinomycotic), or tuberculosis of other organs.

(ii.) *Perforation of* or *slow leakage from some part of the alimentary canal*, which had previously become thin by ulceration—simple ulcer of the stomach or duodenum (malignant ulcer rarely perforates because of the infiltration around), typhoid or tuberculous ulcer of the ileum, etc. (see Perforative Peritonitis). Slow leakage from a gastric ulcer may cause a subphrenic abscess or abscess in the lesser sac.

(iii.) *Rupture* of an organ or some abdominal cyst, such as ovarian cyst, or an abscess of the appendix, tube or liver, or rupture of the gall-bladder, etc. (§ 243).

(iv.) *Injury or Operation.*—In cases occurring in women without obvious cause, the possibility of criminal procedure for abortion should always be remembered. As regards surgical operations on the abdomen, modern experience has shown that it is not the actual injury but faulty technique, permitting the introduction of septic organisms, which produces peritonitis.

(v.) Various *Blood Infections*—*e.g.*, pneumococcal (usually in females), streptococcal, staphylococcal, and gonococcal. *Idiopathic Peritonitis* was the name formerly employed when no cause could be discovered. Peritonitis may also complicate scarlatina, dysentery, and the other acute specific fevers. *Puerperal Peritonitis* arises when septic organisms enter through the infected uterine surface. *Bacillus coli communis* may produce peritonitis either as part of a general septicæmia, or primarily.

(vi.) Any condition such as *volvulus* or *intussusception*, in which injury

of the intestinal wall has occurred, may be a cause of peritonitis, local or general.

(vii.) Local peritonitis from *Crohn's disease.*

Acute general peritonitis has to be *Diagnosed* from four diseases: (1) *Acute intestinal obstruction,* in which the constipation is absolute and no flatus is passed, even after repeated enemata; there is usually no pyrexia, and the constitutional disturbance is usually less. (2) In *colic,* although the pain is also very severe, there is an absence of rigidity, and pressure may give relief. Pyrexia and shock are absent, and the pulse is normal. (3) In *catarrhal enteritis* there is pain, and there may be vomiting and tenderness on pressure, but in this disease there is profuse diarrhœa. (4) In certain cases of *hysteria,* acute peritonitis may be very accurately simulated, though the temperature and pulse are normal, there is very little shock and no leucocytosis, and evidences of the hysterical diathesis are present.

The *Prognosis* of general peritonitis is always very serious. As regards etiology, perforative peritonitis, formerly considered the gravest, is probably now the most hopeful if promptly dealt with. Surgery has done much for the rescue of such cases, and undoubtedly the most favourable of them is that due to appendicitis. Patients with this disease, if diagnosed early and properly managed, should hardly ever be lost. The prognosis in any particular case depends therefore on (i.) the time elapsing before operation, (ii.) the cause and the severity of the shock due to toxæmia, and (iii.) adequate drainage.

Treatment.—The treatment of acute peritonitis depends upon whether it is general or local. If *general,* the only rational treatment is by operation, with drainage, immediately a diagnosis has been made. A fatal issue is almost invariable in cases not operated upon, because the condition is rarely primary, and a definite local lesion is usually present. In *local* peritonitis medical treatment is indicated in the early stages, but even then only with the co-operation of a surgical colleague. Medical treatment comprises keeping the patient in bed in the Fowler position and relieving symptoms. The diet should be fluid, consisting of fruit drinks with glucose, soups, jelly, milk, to which stimulants (*e.g.*, brandy) may be added according to the condition of the pulse. Rectal or intravenous feeding with 5% solution of glucose may be necessary. Severe cases with much vomiting are treated by continuous aspiration through a Ryle's tube in the stomach, or a Miller-Abbott tube in the duodenum, fluids being administered solely per rectum and intravenously. Local applications may give relief, especially heat in the form of fomentations. Once a diagnosis has been arrived at, morphia is a most valuable drug, for it relieves the pain, and reduces the peristalsis of the bowel, and so gives local rest. If there is any doubt as to the advisability of a surgical operation, either at once or later, morphia must be withheld, for by masking the symptoms it may lead to a continuation of medical treatment when operation is called for. It is therefore of use chiefly in local peritonitis,

or in general peritonitis where an operation is not permissible. Purgatives may be dangerous, but the lower bowel should be cleared by means of enemata. The hiccough may be relieved by giving ice to suck, by liq. iodi mitis ♏ iii in a little water, by injections of morphia or pethidine, or chloral per rectum (§ 273).

II. *The patient complains of* **sudden abdominal pain** *and vomiting, and shows* **severe shock,** *pallor, restlessness, air-hunger and subnormal temperature, with rapid running pulse of low volume—the condition is* INTERNAL HÆMORRHAGE.

§ **244a.** In **Internal Hæmorrhage** *shock* is the striking feature, and the patient may become very anæmic in a few hours. Pain is not marked, and vomiting, although present at the onset, is not diagnostic. Local tenderness may serve as a guide to the cause of the hæmorrhage. The most important causes are: (1) a ruptured ectopic pregnancy. There may be a history of a missed or abnormal last menstrual period, and on examination a boggy mass is felt in the pouch of Douglas (§ 446). (2) Injuries to the abdominal organs, and especially traumatic rupture of the spleen, liver or kidneys: hæmorrhage may follow immediately after the injury, or may be delayed to the 7th–10th day.

(3) **Acute Hæmorrhagic Pancreatitis** is a special variety of acute pancreatitis (§§ 245, 256), in which auto-digestion leads to extensive internal hæmorrhage.

(4) **Acute hæmorrhage into the suprarenal capsules** produces symptoms similar to those of acute hæmorrhage into the pancreas. There is sudden epigastric and lumbar pain, with vomiting, shock, marked dyspnœa and cyanosis. Death may occur in a few days. Or there may be delirium, convulsions or coma, or extreme muscular weakness for some days before death. It is rarely diagnosed during life. When it occurs as part of a meningococcal septicæmia with purpura, it is known as the Waterhouse-Friderichsen Syndrome. In newly-born infants it occurs as part of a hæmorrhagic diathesis.

Treatment.—Blood transfusion is usually a primary consideration. As soon as possible operation must be undertaken in order to stop the hæmorrhage. With suprarenal hæmorrhage, give injections of vitamin K and suprarenal cortical extract. In any case of internal hæmorrhage it is important to remember that the primary hæmorrhage may cease from clotting or encapsulation, but recur subsequently from disintegration of blood clot from sepsis.

III. *The patient complains of* **acute abdominal pain with shock,** *attended by* URGENT AND COPIOUS VOMITING *(at first food, then bile, and later, material which is alkaline to litmus, and finally fæcal). There is* ABDOMINAL DISTENSION *and* INABILITY TO PASS FLATUS *even after repeated enemata—the condition is* ACUTE INTESTINAL OBSTRUCTION.

Acute Intestinal Obstruction—*i.e.*, obstruction coming on suddenly, is always a matter of serious importance, and every practitioner should be thoroughly acquainted with its several causes. The diagnosis and the various causes are fully dealt with under Intestinal Disorders in § 319.

§ **245.** *The patient complains of* **acute abdominal pain,** *with more or less*

shock ; *the temperature is probably normal or subnormal, but the symptoms do not quite conform to any of the preceding*—some of the **rarer causes** are probably in operation, such as the following :

IV. **Torsion of an Ovarian Cyst** is known when the signs of such a cyst are associated with the onset of sudden pain and tenderness of the cyst.

V. In **Embolism of the Mesenteric Artery,** a cause of embolism, such as endocarditis, is present. It is rarely diagnosed during life. The absence of symptoms pointing to the other causes, and the presence of melæna, may lead one to suspect embolism. Embolism of the spleen may also cause severe symptoms.

VI. **Acute Pancreatitis** is due to regurgitation of infected bile into the pancreatic duct, due to thickening or spasm of the sphincter of Oddi, and often in association with gallstones. (1) The pain here is very sudden, persistent and severe, usually in the upper part of the left side of the abdomen and extending to the back ; (2) constipation and severe vomiting (never fæcal) are usually present ; and (3) there is usually tympanitic abdominal distension with epigastric tenderness and rigidity ; (4) cyanosis, circulatory collapse and profuse sweating, are associated with a subnormal temperature ; (5) a brownish-green discoloration around the umbilicus, or in the lumbar area, when present, aids the diagnosis (§ 256).

GROUP B. VII. *The patient, while apparently in good health, complains of* **acute abdominal pain,** *which has come on* **suddenly, without** *definite* **shock ;** *the pulse does* NOT EXCEED 100 ; *there may be* VOMITING *and constipation.* The case is probably one form of COLIC, though APPENDICITIS, ROOT OR REFERRED PAIN, and some OTHER AFFECTIONS may start in this way.

§ **246. Colic** is a somewhat vague term applied to spasmodic paroxysmal pain situated in the abdomen. There are **intestinal, biliary, renal** and **appendicular colic.** All have the following features in common : (1) The pain is extremely severe (in the first three, less so in appendicular colic), and sudden in its onset ; (2) not infrequently there is reflex vomiting from the severity of the pain ; (3) the face is pale and " anxious," and in severe cases the pulse is rapid and feeble, though it practically never exceeds 100 ; (4) *the temperature is neither above nor below normal ;* (5) the physical signs in the abdomen are negative, and the pain may even be relieved by pressure ; (6) the patient is in a " cold sweat," " doubled up " with pain, restless, or rolling about.

(*a*) **Intestinal Colic** is due to distension and spasm of the bowel. The colic of the small intestine is characteristically twisting, paroxysmal, and is referred to the epigastrium or umbilicus ; colic of the colon is referred to the hypogastrium. In intestinal colic a hardening of the bowel may be appreciated by the palpating hand. It is relieved by pressure, which distinguishes it from peritonitis. The abdomen may be distended with flatus. Sometimes it is followed or accompanied by diarrhœa and vomiting, as in gastro-enteritis, or by constipation, as in lead colic. Colic may be the first sign of lead-poisoning, accompanied by a slow, hard pulse, with other signs of plumbism, such as a blue line on the gums ; a history of working with lead may be obtainable (§ 553). Colic is a frequent early symptom of *diverticulitis* (§ 321). The *heat cramp* (§ 508) of miners and workers in stokeholds may resemble abdominal colic. Cramp may also be experienced in high or low atmospheric pressures.

(*b*) **In Biliary Colic,** due to the passage of a gall-stone into the bile ducts, the pain starts in the right hypochondrium : it often radiates round the 9th segment to the angle of either scapula, or reflexly it occurs along the root of the neck or at the tip of the shoulders. A dull pain continues during the intervals between the spasms and may be felt in the right iliac fossa. After lasting a few hours or a day or two it may be followed by jaundice and bile in the urine. A history of previous attacks assists the diagnosis.

(*c*) **Renal Colic** is due to the movement of a calculus, crystals, muco-pus, or blood-clot in the pelvis of the kidney or along the ureter. The pain starts in the loin or in the upper lateral abdomen, and radiates *downwards* to the groin and testicle of the same side, which is often retracted. It may last for a day or two. Sometimes pain is referred to the opposite kidney. During the attack there is rigidity in the loin and often some tenderness over the kidney; micturition is frequent; sometimes there is hæmaturia or strangury. For some time after the colic an exaggerated cremasteric reflex persists. There is probably a history of previous attacks, or of gravel, blood or pus in the urine.

(*d*) **Appendicular Colic** is due to an obstruction in the appendix, by a concretion kink or stenosis from a previous attack; distal to the obstruction acute inflammation may develop. The pain occurs in the right iliac fossa, is never very severe, and is accompanied by some rigidity and localised tenderness over MacBurney's point; in children it may be referred to the epigastric or umbilical regions.

TABLE XIV.—DIAGNOSIS OF COLIC.*

	Character and Distribution of Pain.	*Associated Symptoms.*	*Age and Sex of Patient.*
Intestinal.	Twisting, around umbilicus, paroxysmal; relieved by pressure.	Constipation (or diarrhœa). No jaundice.	Any age or sex. Sometimes evidence or history of plumbism.
Biliary.	In right hypochondrium, shooting upwards to right or left shoulder, constant, but also in paroxysms.	Jaundice may supervene. Other hepatic symptoms may be present.	Stout married women over forty.
Renal.	In loin shooting down to groin and testicle or labium of same side.	Crystals or other urinary change, pus or hæmaturia. No jaundice. Sometimes frequent micturition or strangury.	Usually male. Children and adults.
Appendicula colic.	In right iliac fossa.	May be vomiting; local tenderness and rigidity.	Any age; both sexes.

Pyloric Spasm, especially with a duodenal ulcer, can give attacks of acute paroxysmal pain in the upper abdomen, and more rarely **Spasm of the Cystic Duct** can cause acute pain arising from the gall bladder.

The *Diagnosis* of the forms of colic is given above. An X-ray examination should be made when more than one attack occurs.

Prognosis.—The course of an attack of colic is short and severe.

Treatment.—For all forms of colic, local applications of hot fomentations, a kaolin poultice, or a hot bath, and hypodermics of morphia (gr. $\frac{1}{6}$ to $\frac{1}{4}$), and atropin (gr. $\frac{1}{60}$), of pethidine (50–100 mgm.) or of ephedrine (gr. $\frac{1}{2}$–1) may be necessary to alleviate the extreme pain. Large draughts of warm water should be taken. Especially with intestinal colic an enema should be given, followed by suitable purgatives. For lead-poisoning, see § 553. Hepatic colic is dealt with under gall-stone (§ 353) and renal colic in § 408.

VIII. *The* **Abdominal Pain** *is constant, but liable to exacerbations, especially after exercise; there is* NAUSEA *or* VOMITING, *with some elevation of the temperature; there is* RIGIDITY *and* TENDERNESS *in the right iliac region; the pulse is rapid. The disease is probably* ACUTE APPENDICITIS.

§ **247. Acute Appendicitis** may consist simply of (*a*) a catarrhal inflammation of the vermiform appendix, which is relatively benign: or (*b*) a virulent form with ulceration, gangrene and local or diffuse peritonitis.

Symptoms.—In a typical *acute attack of appendicitis* there are six symptoms which, occurring in this sequence, point to appendicitis—pain, vomiting, tenderness, local rigidity, a quickened pulse, and leucocytosis. (1) The chief symptom is *pain*, coming on acutely, first referred to the umbilicus or epigastrium, and later becoming localised to the right iliac fossa. (2) *Vomiting* may be urgent at the onset of an attack; when it continues for many days the prognosis is unfavourable. (3) On examination, the most marked features are tenderness, rigidity, and later a local swelling. The *tenderness* may be manifest as cutaneous tenderness on picking up the skin in the right iliac fossa between fingers and thumb. There is also deep tenderness on palpation, particularly well marked at "MacBurney's point," *i.e.*, at the junction of the outer and middle thirds of a line joining the right anterior superior iliac spine and the umbilicus. A third point of tenderness is often found on rectal examination in the right anterior wall of the rectum, particularly with the pelvic position of the appendix. The *rigidity* causes the abdomen as a whole to show a poor respiratory excursion: on palpation there is guarding of the muscles, particularly of the lower right segment of the rectus abdominis. (4) There may be a local *swelling* or an indefinite tumour with dulness to percussion. These are due to local peritonitis or to abscess formation; they may also be found on rectal examination. (5) The *pulse* is quick and thready and its rate forms the best single indication of the acuteness of an attack. The temperature is rarely above 99°–100°, and this disturbance of the pulse-temperature ratio is an important diagnostic aid (§ 89). The disease is rarely ushered in by a rigor. The fever often falls and the pain goes with the onset of gangrene or with spreading peritonitis, but the pulse, except in rare cases, remains rapid. (6) The tongue is almost always coated. Constipation is usually present, so that the case may be mistaken for intestinal obstruction; but sometimes the attack is ushered in with

diarrhœa. The urine is scanty; with pelvic appendicitis the bladder is irritable, and often there is diarrhœa. (7) On listening with a stethoscope no gurgling sounds are heard, owing to spasm of the ileo-cæcal sphincter associated with inflammation around. (8) Leucocytosis of 15,000 to 20,000 per cu. mm. occurs. (9) Paralytic ileus is usually a late event.

Types of Acute Appendicitis.—(i.) *Catarrhal* inflammation is mild and localised to the appendix. It may subside completely, but usually some degree of inflammation remains which causes local discomfort and vague dyspeptic symptoms. (ii.) Recurrent attacks of this nature cause fibrous thickening in the wall and retention of secretion forming a *mucocœle* of the appendix. Then (iii.) a subsequent attack of inflammation produces an abscess in the tip of the appendix. (iv.) *Ulceration and gangrene* of the appendix are due to a more virulent infection, arising behind the obstruction of a stercolith, in a mucocœle, or by obstruction of the lumen by external adhesions. Gangrene is usually due to septic thrombosis of the blood supply to the appendix. (v.) An *abscess* which forms in the lumen may perforate. If the reaction of the peritoneum is vigorous and the organisms not too virulent, a local peritonitis results: the abscess can resolve and the inflammation give a mass of local adhesions. (vi.) In other cases the abscess enlarges and may finally rupture into the bowel or bladder, may descend into the pelvis or point externally above Poupart's ligament (when it is often mistaken for a psoas abscess). (vii.) Sometimes the inflammation extends to the cæcum (typhlitis) or to the surrounding tissues (perityphlitis). (viii.) When the organisms are very virulent, a fulminating general peritonitis is likely to arise.

Aberrant types.—With a retrocæcal appendicitis pain may be referred to the loin, or down the right thigh, in each case leading to flexion of the hip from psoas spasm. If the appendix happens to be a long one with the tip lying in the left side of the pelvis, there may be pain, tenderness and rigidity entirely confined to the left iliac fossa.

Course and Prognosis.—With an acute attack there are three possible events—recovery, local abscess formation, or general peritonitis. (1) In a favourable case the temperature falls about the third day, the swelling disappears, pain and other symptoms subside, and the patient may be well in ten days. In other cases slight fever persists for a few weeks, and there is left an indurated swelling due to the omentum. The patient may go about for months or years with chronic appendicitis, and suffer only vague pains, general malaise and dyspeptic symptoms. At any time, however, the acute symptoms may recur. (2) When the general symptoms show no improvement by the second day, and the local swelling progressively increases, it is probable that an abscess is forming. (3) Perforation, with generalised peritonitis, may occur at any time. The general symptoms in such cases are much more severe, vomiting persists, and the abdomen is distended and motionless by the second or third day. There is no disease in which it is more dangerous to hazard a prognosis. An apparently convalescent case may develop general peritonitis and die

within twenty-four hours; on the other hand, a case presenting every sign of a large and extending abscess may clear up entirely and prove free from any subsequent attack. Apart from the great improvement in the prognosis when immediate operation is performed, the only indications of value when forming an opinion are the condition of the patient as regards shock, collapse, and age. The younger the subject, the more grave the prognosis. In pregnancy, appendicitis is serious. *Complications.*—Apart from local and general peritonitis the complications most to be feared are the formation of a subphrenic, perinephric or pelvic abscess: or implication of the liver by spread along the vessels and lymphatics leading to portal pyæmia. Any previous appendicular inflammation which has not been treated surgically may act as a focal point of infection, causing arthritis, iritis, etc.

Etiology. Two main types of appendicitis are often recognisable. In the first there is obstruction to the lumen of the appendix and the stasis leads to inflammation. In the second there is a blood stream infection of the appendix, often from a catarrhal condition of the naso-pharynx or tonsils.

Treatment.—Rest in bed in the Fowler position and fluid diet are essential. Hot fomentations locally are useful for the pain. Opium in small doses (short of causing drowsiness) is also admissible for the relief of pain after the diagnosis is established. Neither opium nor heroin should be given for long. Other analgesics such as omnopon and pethidine may be employed.

The *question of operation* requires careful consideration, and a surgeon should be early in touch with the case. The largest proportion of recoveries is recorded in cases operated on within six hours of the onset of symptoms which enabled a diagnosis of appendicitis to be made. The subsidence of symptoms is not necessarily a contra-indication to operation. The onset of gangrene, in particular, may cause a sudden subsidence of all signs of acute disorder; even the pulse rate may return to within normal limits, and if seen first at this stage the diagnosis may be very difficult. The most valuable sign, in the absence of clinical indications, is the presence of a leucocytosis. If this goes above 20,000, or is found to be rising when two or more estimations are made at intervals, there is so strong a presumption of pus formation that immediate operation is indicated. If, by this or by other means, the presence of pus is diagnosed, operation must not be delayed. Delay for even a few hours, as, for instance, when the patient or his friends are unwilling that he should be removed to a hospital or home "until the morning," has on many occasions proved fatal from the onset of collapse of such severity that the patient's strength was not sufficient to carry him through even the shortest operation.

§ 248. Among the **rarer causes of acute abdominal pain** without shock are:

IX. Of various OBSCURE ORGANIC AFFECTIONS of the abdomen, evidenced at first only by pain, two may be mentioned: PANCREATIC CALCULUS and OBTURATOR HERNIA, in both of which the only symptom for some time is pain coming on SUDDENLY without shock. In the former the pain may be extremely severe, and of a paroxysmal character, situated just below the umbilicus; later on it can be associated with fat in the fæces, emaciation, and glycosuria.

Attacks of KETOSIS in children are associated with pyrexia, headache, abdominal pain and vomiting (§ 384). URÆMIA and PYELITIS can give similar symptoms.

DISLOCATED or FLOATING KIDNEY may be attended by a constant (chronic) pain, or give rise to severe attacks (Dietl's crises, § 253), hardly distinguishable from intestinal or renal colic.

DIVERTICULITIS may cause attacks of acute abdominal pain in the left iliac fossa (§ 321).

INTESTINAL WORMS (§ 316) can cause abdominal pain, pyrexia and constitutional symptoms which must not be confused with acute appendicitis, especially in children.

TORSION OF AN UNDESCENDED TESTIS should be suspected when a testicle is found to be absent from the scrotum.

OSTEOMYELITIS OF THE ILIUM or OF THE SACRAL VERTEBRÆ shows persistent pain, pyrexia and leucocytosis.

In SPLENIC EMBOLISM the pain is generally sudden in onset, but is not usually very severe or lasting, and is referred to the splenic region. Its most common cause is acute or subacute endocarditis, evidences of which are present.

HENOCH'S PURPURA and angio-neurotic œdema may have acute recurring attacks of colic simulating intussusception. For differential features see § 584.

ENLARGED GLANDS may cause symptoms resembling appendicitis. They may be tuberculous, or associated with typhoid or glandular fever, or with streptococcal throats.

In most obscure organic affections the pain comes on gradually, and is of a chronic character. Acute pain occurring in attacks of varying duration is met with in cases of membranous or MUCOUS COLITIS, OVARITIS and PANCREATITIS (as with mumps): also with ANEURYSM OF THE ABDOMINAL AORTA, with a FLOATING RIB and in CROHN'S DISEASE. DIABETIC COMA is sometimes heralded by pain, usually in the epigastrium, which may be very severe (§ 238). Exaggerated abdominal breathing is a useful diagnostic aid.

The causes of abdominal pain which originate from organs outside the abdomen are mentioned in § 238.

X. In ROOT PAIN or REFERRED PAIN abdominal pain may come on suddenly and acutely, and may be for a long time the only symptom.

1. *Nervous dyspepsia* is one of the most typical forms of referred pain. The pain is severe, periodic, but usually relieved rather than aggravated by food or by pressure. The skin may, however, be very sensitive to the flick of a handkerchief.
2. The gastric and vesical crises in association with *tabes dorsalis*.
3. At the onset of *acute poliomyelitis*, pain may be referred to the abdominal wall.
4. *Spasm* or *colic* of any hollow viscus may occur without organic derangement or discoverable nervous cause, especially in nervous subjects. The commonest type is colospasm (§ 252).
5. The neuralgia which accompanies or follows *herpes zoster*.
6. *Coronary thrombosis* causes pain to be referred more to the abdomen than to the chest, but is recognised by the circulatory disturbances (§ 52).
7. *Basal pneumonia, diaphragmatic pleurisy* and *blast injuries to the lung* can cause abdominal pain with rigidity.
8. *Migraine* is certainly met with, alternating with abdominal pain.
9. *Acute glaucoma* is an occasional cause (§ 855).

§ 249. Chronic Abdominal Pain comes and goes at first, then BECOMES CONTINUOUS with PERIODIC EXACERBATIONS. Here we do not deal with pain which points definitely to lesions of the stomach, liver, spleen or intestines: these are considered in their respective chapters. Abdominal pain is the leading or only symptom in the following conditions:

The history must be thoroughly investigated, and every organ carefully examined. Three features may afford us important clues:

1. The POSITION, character, degree, and constancy of the *pain*, and the presence of *tenderness* must be observed. (i.) If the pain and tenderness be *generalised*, one may suspect Tubercle or Cancer of the Peritoneum. (ii.) If they be situated chiefly in the *lower abdomen*, one may suspect Appendicitis or disease of the Colon, Bladder, Ovary, Fallopian tubes, or Uterus. (iii.) If the pain be chiefly in the *upper abdomen*, Gastric, Duodenal, Liver or Gall-bladder disease. Thorough and REPEATED EXAMINATIONS of the *abdomen*, *rectum*, and *vagina* are nearly always necessary. The *urine* also should be repeatedly examined for blood, pus and crystals, and the *fæces* (§ 303) for gall-stones. Occult blood (§ 303) and chemical changes pointing to disease of some organ may be detected by expert examination of the fæces. If there be general abdominal enlargement, turn to § 257; if a localised tumour, turn to § 263. X-ray or special instrumental examinations (§ 240) may yield important information.

2. The AGE of the patient, and the history and duration of the illness should be inquired into. In *children* perhaps the commonest of the obscure causes of chronic abdominal pain are constipation, dietetic errors, intestinal worms, tuberculosis of the peritoneum and Meckel's diverticulum; in the *aged* cancer of some organ.

3. The STATE OF THE BOWELS, both previous to and at the time of examination. In I., II., and III. above there is constipation, while in some of the other causes there is diarrhœa or irregularity of the bowels.

I. **Chronic Appendicitis** occurs in two typical forms: Chronic, and Recurrent or Subacute Appendicitis. (*a*) In CHRONIC APPENDICITIS (1) the chief symptom is pain starting from the right iliac fossa, or radiating from the umbilicus or epigastrium to this region. It occurs particularly after food, but the typical time-relationship of gastric or duodenal ulcer is absent, and strict dieting affords only partial relief. The pain is characteristically aggravated by over-exertion. (2) Hæmatemesis may occur from an acute gastric or duodenal ulcer, secondary to the appendicular sepsis. (3) Nausea may occur, apart from vomiting, and sometimes there is alternating diarrhœa and constipation, and (4) a history of general malaise. (5) X-ray will reveal tenderness, fixation or deformities and defects of filling, with prolonged retention of barium in the lumen. One form of chronic appendicitis is due to malignant disease, tuberculosis or actinomycosis of the cæcum or appendix. Another is due to stricture of the lumen with formation of mucocœle of the appendix.

(*b*) RECURRENT APPENDICITIS has recurring subacute attacks. Here the course of the disease is essentially chronic, and is often associated with colitis. The patient may have months of apparent health, but in most

cases a fresh attack of inflammation occurs sooner or later. It is wise to operate if circumstances permit.

II. *In addition to* **chronic abdominal pain,** *there is a history of* CONSTIPATION, *steadily increasing to* COMPLETE STOPPAGE *of the bowels and* DISTENSION. VOMITING *gradually becomes more severe.* The case is probably one of CHRONIC INTESTINAL OBSTRUCTION *with supervention of acute symptoms.*

In CHRONIC INTESTINAL OBSTRUCTION (§ 320) the abdominal pain is more or less generalised and intermittent. The constipation may at first have alternated with diarrhœa, but after a time it is so complete that not even flatus can be passed. Vomiting, at first of food, and later of alkaline or even fæculent matter, a rapid pulse, and other constitutional symptoms ensue if the condition is not relieved. The commonest causes are Malignant Stricture, Simple Stricture, Peritoneal bands, Diverticulitis, Pressure of a Tumour, Volvulus, and Impacted Contents.

III. *The* **abdominal pain** *is* **chronic** *and* GENERALISED ; *it is attended by* CONSTITUTIONAL SYMPTOMS, *and some* ABDOMINAL ENLARGEMENT *or other local signs.* The disease is probably CHRONIC PERITONITIS.

§ 250. **Chronic Peritonitis** runs a slow and chronic course, and is usually attended by a certain amount of generalised pain. There is a simple or idiopathic chronic peritonitis, but three more frequent forms are: (*a*) That due to **tubercle,** and (*b*) that due to **cancer**—two conditions which, by the way, are most frequently met with at the opposite extremes of life, and present a marked contrast both in their clinical and anatomical features; (*c*) Rupture of a papilliferous ovarian cyst (§§ 243, 261).

CHRONIC TUBERCULOUS PERITONITIS is known by (1) the patient is young; (2) pain and tenderness; (3) localised hard masses or a general doughy feeling; (4) often fluid, and (5) always emaciation and fever. Hence the disease is fully discussed under the heading of emaciation in § 557.

CHRONIC CANCEROUS PERITONITIS (Cancer of the Peritoneum) is always attended by much pain, constant, and also in paroxysms. There is a great tendency to the rapid formation in the abdominal cavity of fluid which is nearly always tinged with blood. It arises only in late middle or advanced life. Its recognition is easy in typical cases on account of the age, acute pain, and ascites (under which heading it is described, § 260). SARCOMA of the peritoneum is rare.

CHRONIC PERITONITIS of the simple or idiopathic type is very difficult to diagnose in the majority of cases, because of the extreme variability and vagueness of the symptoms. (1) Pain and tenderness, sometimes localised, are present, worse at times and with exertion; (2) dyspepsia, often constipation, sometimes vomiting; (3) malaise with pyrexia from time to time; (4) palpation may detect localised thickenings and areas of resistance which convey a doughy sensation on palpation; (5) ascites is present in some cases; in other cases it is absent, and the abdomen is quite flat.

Etiology.—(1) After an attack of acute peritonitis; (2) inflammation of any organ may cause localised peritonitis; (3) after paracentesis without strict asepsis; (4) idiopathic, due to unknown causes. It may occur with chronic nephritis, with cirrhosis of the liver, and with other general conditions, in which two or more of the serous

membranes (pleura, pericardium) become simultaneously affected (polyorrhomenitis or polyserositis).

The *Diagnosis* has often to be made by a process of exclusion, especially when there is no history of acute peritonitis nor of inflammation of any organ. Sometimes it is indistinguishable from tuberculous and cancerous peritonitis. Abdominal pain simulating colic may be due to peritoneal adhesions. When ascites reappears after repeated tappings peritonitis is usually present.

The *Prognosis* as to life is good in mild cases, though chronic invalidism is apt to ensue. Subacute attacks are liable to occur, and there may be great exhaustion and emaciation from involvement of some part of the alimentary canal, or from the formation of local abscess. Adhesions may lead to intestinal obstruction. When associated with advanced hepatic or renal disease, the prognosis is grave.

Treatment.—Rest and supporting belts may give relief. Inunction with blue ointment or applications of Liq. Iodi Mit. (B.P.) (1 in 3 of water) are useful. Paracentesis and surgical treatment may be required.

IV. § 251. **Visceroptosis** (Synonyms: Glénard's disease, enteroptosis) is a condition with ptosis or downward displacement of one or all of the abdominal organs. It is more common in the thin nervous woman, and has various causes. When ptosis is accompanied by membranes superimposed on the lengthened mesentery, obstructions and "kinks" tend to form, which give rise to varied symptoms. Progressive loss of intra-abdominal supporting fatty tissue, and weakness of the abdominal muscles are other factors which hasten the onset of symptoms. *Symptoms* are: (1) Pain or dragging and "sinking" feelings in the abdomen or back, palpitation and dizziness; (2) dyspepsia, sometimes nausea and vomiting severe enough to imitate a gastric ulcer; (3) constipation, often alternating with diarrhœa; (4) intestinal stasis, causing symptoms of toxic absorption, such as lethargy, headache, skin pigmentation, fatigue, and nervousness; (5) anæmia is common.

The *diagnosis* from gastric ulcer and appendicitis in young women, and malignant disease in old people, may be very difficult; but when palpation reveals a gurgling cæcum, and prolapse of kidneys and stomach, medical treatment can be safely tried. X-ray examination of a pronounced case shows a prolapsed stomach, the lower border reaching the pelvis, a mobile and elongated duodenum, a dilated and prolapsed cæcum, the transverse colon low in pelvis, and an elongated pelvic colon. Lane's chief "kinks" are obstructions in the terminal coil of the ileum and of the ascending or sigmoid colon due to adhesions causing pressure and narrowing of the lumen of the intestine. When X-ray examination is not available, the condition is usually detected by percussion and palpation of the abdomen, and inspection in the upright position.

Treatment.—Prevent toxic absorption by aiding free elimination, and by giving food which causes less toxic residue. The first is accomplished best by paraffin and bassorin preparations together with aperients; the second, by a lacto-vegetarian and vitamin-containing diet, with restriction of meat and abundance of fresh fruit and vegetables. A well-fitting and correctly applied abdominal belt, such as the Curtis belt, aids mechanically by holding in better position the dropped viscera, and relieves the

pain and dragging sensations. Fattening the patient and treating anæmia act similarly. Massage, electricity (rhythmic faradic or sinusoidal currents) and exercises afford support by developing the abdominal muscles. Rest with the foot of the bed raised and other measures to aid the nervous symptoms are necessary; a modified Weir-Mitchell treatment is often useful. Operations are better avoided.

V. § 252. **Spastic Colon** (Syn.: Colospasm) occurs in young or middle-aged adults.

Symptoms.—(i) Pain over the line of the colon, usually of a nagging or burning character, is most marked in the descending or sigmoid colon: often more of a severe continuous discomfort than a true pain, it is liable to colicy exacerbations, which may be most marked *after* a bowel evacuation. (ii) The colon is felt as a tender contracted tube in spasm. Often the patient accurately delineates the colon along its whole course. (iii) Constipation results from the spasm. (iv) The stools are often thin and pencil-like in form, and may be passed in several small portions during the day. (v) Between the bouts of constipation diarrhœa may result from aperients taken to relieve the condition. (vi) Frequency of micturition and eczema of the umbilicus may co-exist.

Etiology.—Colospasm probably results from an overaction of the parasympathetic nerves, and usually occurs in overactive persons, especially when worried or fatigued, or in cold weather. It is unusual to find any other organic disease of the colon, but secondary spasm may follow chronic or mucous colitis. Diverticulosis and diverticulitis are recognised complications.

Treatment.—It is difficult to free the patient permanently from this condition. A warm flannel belt should be worn at all times; the general health must be attended to, with adequate holidays and the avoidance of fatigue. Irritating foods as well as irritant purgatives must particularly be avoided; phenolphthalein seems to be especially detrimental. Liquid paraffin, without or with agar-agar, small regular doses of belladonna, eumydrin, or phenobarbitone, and small enemata may be used.

VII. § 253. **Movable Kidney** (Dropped, Dislocated, or Floating Kidney, according to the degree of mobility).—This condition is by no means uncommon and does not usually give rise to symptoms unless the degree of mobility is considerable.

The *Physical Signs* can only be discovered by palpation of the abdomen, with the patient lying down. The method of palpating the kidneys is given in § 394. With the patient in the erect or sitting posture, the kidney comes down more during inspiration than when lying down. After a little practice the patient will be able to lean forward and relax the muscles, which is an important aid to the observer. The left kidney rarely falls below the umbilicus, but the right one may be displaced into the iliac fossa, and even into the pelvis.

Symptoms.—In a few cases two kinds of pain may be experienced: (*a*) A constant dull, dragging pain in the back, or perhaps only an uneasiness in the loin, radiating down to the groin and inner side of the thigh, relieved by rest; (*b*) Attacks like renal colic may be followed by the passage of urine in large quantities, occasionally with albumin and blood, due to vascular engorgement of the kidney—"Dietl's crises" (§ 414). Sometimes hydronephrosis results. Neurasthenia often follows, with mental depression or symptoms of dyspepsia, vertigo, diarrhœa, or constipation.

Etiology.—A much larger percentage of women than of men have movable kidney: it often follows pregnancy. A fall or strain will also displace the organ, and that is why it is advisable for those with spare abdominal muscles to wear a belt when at work in the gymnasium. It occurs more often in tall, narrow-chested than in short people. Rapid loss of fat, or lowering of the intra-abdominal pressure, such as occurs after delivery, are frequent causes.

Treatment.—Bromides and rest will relieve the patient for a time, and any concurrent dyspepsia must be remedied; but the best treatment consists in wearing a proper form of belt and improving the health. The abdominal belts supplied by

instrument makers are not always successful, but an apparatus is designed for applying additional pressure inside the belt; and in some cases pads over the kidney can be introduced between the belt and the abdominal wall. Fattening the patient, abdominal exercises, and sleeping on an inclined plane with the foot of the bed raised, often relieve symptoms.

VIII. § **254. Pain Following Previous Abdominal Operations.**—After previous operations intra-abdominal adhesions may form or nerves supplying the abdominal wall itself may be involved in the scar tissue and give rise to persistent pain. The latter variety may be differentiated by using a local anæsthetic to block the intercostal nerves before they reach the abdominal wall. If this relieves the pain, a more permanent effect may be obtained by the subsequent blocking of nerve impulses with a few c.c. of 90 per cent alcohol: a few applications of abdominal diathermy softens scar tissue and gives relief.

IX. § **255. Incipient or Obscure Visceral or Spinal Disease.**—(*a*) In cases of chronic pain GENERALISED OVER THE ABDOMEN, and in the absence of constipation, diarrhœa, or any of the causes mentioned under § 248 onwards, one might suspect cancer of the intestines, of the pancreas, or of the kidney, cancer or tubercle of the suprarenals (*i.e.*, Addison's disease, in which pain in the epigastrium is a constant symptom), "rheumatism" of the abdominal muscles, visceroptosis, or movable kidney. In many cases the pain is an expression of a psychoneurotic anxiety state. Children may suffer from recurrent attacks of abdominal pain due to worms: but often no cause can be found. Such cases should be treated as incipient intussusception—that is to say, avoid purgatives and give digestible foods and enemata.

(*b*) In various spinal affections the pain is frequently referred to the FRONT OF THE ABDOMEN, and among the more obscure causes may be mentioned abdominal aneurysm pressing on the spine, and cancer or caries of the vertebræ. The first of these occurs mostly in male adults, the second in the aged, and the third (Pott's disease) in children. In the latter the child frequently refers to the pain as "stomach-ache," worse after sneezing or running about. The girdle pain due to a spinal cord tumour, chronic and acute myelitis and the prodromal stages of the exanthemata should also be borne in mind.

(*c*) If the patient complains of PAIN SITUATED CHIEFLY IN THE LOWER ABDOMEN, one might suspect diseases of the intestine or rectum, appendicitis (§§ 247, 249), cancer or other disorders of the bladder, psoas abscess, lymphatic gland enlargement, and pelvic peritonitis (in which the pain shoots down the legs), extra-uterine pregnancy, pyosalpinx, dysmenorrhœa and all its causes, pelvic displacements, tubercle or cancer of the prostate or testes, sacro-iliac arthritis and obturator hernia. The fatigue pains of debilitated women may be referred to one or other iliac region.

(*d*) PAIN SITUATED CHIEFLY IN THE UPPER ABDOMEN may be due to various affections of the stomach, duodenum, liver or gall-bladder, and spleen. In lesions of organs in this region pain is often referred to the scapula or the root of the neck. Among the painful affections of the *stomach* may be mentioned gastric (or duodenal) ulcer, gastritis (acute or chronic), cancer of the stomach, which in its most usual form, scirrhous of the pylorus, is commonly difficult to diagnose in its early stages. Among the painful affections of the *liver and gall-bladder*, perhaps passive congestion, perihepatitis, cancer, gall-stones and chronic cholecystitis are the commonest; hydatid is one of the obscure conditions, though it is rarely painful. Abscess of the liver should be suspected in those who have resided in tropical countries. Painful affections of the *spleen* are not common, the chief being infarction, but the capsule is sometimes the seat of perisplenitis: enlargement of the organ aids diagnosis.

X. § **256. Diseases of the Pancreas** are fortunately rare, for they are often unrecognisable during life. When definite physical signs of tumour are present, the diagnosis is not so difficult. The **symptoms** commonly present are: (1) Abdominal pain is present in a proportion of cases, and is deep-seated in the epigastrium, often with associated pain in the upper lumbar region; (2) progressive loss of weight commences

early; (3) digestive disturbances are vaguely related to meals and include nausea, anorexia, vomiting, diarrhœa or constipation; (4) general debility and depression; (5) the passage of large pale, bulky stools containing a large amount of unsplit fat; (6) sugar tolerance is usually reduced. Certain *tests* for normal secretion of the pancreatic juice may be applied. (1) *Examination of the Fæces.* If pancreatic digestion is deficient, proper digestion of all three classes of food substances will be deficient; (*a*) creatorrhœa, or the passage of undigested meat fibres, with striæ visible under the microscope; (*b*) steatorrhœa, or the passage of large quantities of *unsplit* fat in the stools (if only biliary obstruction is present, large quantities of split fat are passed in the stools in the form of soaps and fatty acids, see § 303); (*c*) large quantities of undigested starch granules. (2) Increase of *diastase* in the urine and blood, with decrease in the stools. Diastase is the ferment which changes starch into sugar. Normally, an average of 20 units per c.c. is excreted daily in the urine. In cases of obstruction of the pancreatic duct, it may be increased to 300–400 units. When renal activity is impaired, there is less diastase in the urine but more in the blood.

The test measures the amount of starch digested in a given time by a definite quantity of urine. A twenty-four-hours' sample of urine is required, and is made just acid to litmus, also (i.) 0·1 per cent. solution of soluble starch; (ii.) 0·9 per cent. solution of NaCl; and (iii.) a weak solution of iodine. Ten test-tubes are numbered 1 to 10. With 1 c.c. pipette, place in tube No. 1, 0·9 c.c. urine; and then in tubes 2–5, 0·6 c.c., 0·4 c.c., 0·2 c.c. and 0·1 c.c. respectively. For further dilutions of the urine dilute some of the original urine ten times with the saline solution, and into tubes 6–10 place 0·9 c.c., 0·6 c.c., 0·4 c.c., 0·2 c.c. and 0·1 c.c. of the diluted urine. Fill each tube with saline solution to 1 c.c. The tubes now contain 0·9, 0·6, 0·4, 0·2, 0·1, 0·09, 0·06, 0·04, 0·02, and 0·01 c.c. of the original urine. Add to each tube 2 c.c. of the starch solution, shake, and incubate or stand in a water bath at 37° for half an hour. Then remove the tubes to cold water and immediately add 2–3 drops of the iodine solution to each tube, and shake. Notice when the change occurs from the blue to the slight pink tinge. The first tube showing this contains just enough diastase to digest the starch. Divide 2 (the number of c.c. of starch) by the number of c.c. of urine in that tube. Suppose it was tube No. 7. Then $\frac{2}{0{\cdot}06} = 33$ units of diastase. (3) In cases where there is deficiency of the *production of insulin*, glycosuria and lowered sugar tolerance are found.

Less reliable tests are (4) Sahli's test: capsules of gelatin hardened in formalin so that they are digested only by the trypsin of a healthy pancreas, and containing a drug which is readily detected in the urine: (5) Loëwi's test: three drops of 1 in 1000 adrenalin dropped on the conjunctiva, and repeated five minutes later. If the pupil dilates in half to one hour, one can conclude that there is irritability of the sympathetic, which is frequent with pancreatic disease.

1. Pancreatic Cysts are of two types: (*a*) the rare true cyst which contains pancreatic ferments: (*b*) the false cyst containing no ferments. True cysts are due to obstruction or obliteration of the duct by pancreatic calculi, or cicatricial contraction. False cysts are due to injury or follow acute pancreatitis. The swelling appears between the stomach and the colon, and does not move with respiration. Fatty diarrhœa is rare. In the true cyst the fluid will emulsify fat, convert starch into sugar, and digest fibrin.

2. Pancreatic Calculi are small concretions consisting chiefly of calcium carbonate. They are visible on X-ray examination, a diagnostic feature which distinguishes them from the majority of biliary calculi.

3. Acute Pancreatitis is met with in three forms: (*a*) *Acute Hæmorrhagic Pancreatitis*, which sets in suddenly with agonising pain, and often results in death in one to four days (§§ 244, 245). (*b*) *Acute Suppurative Pancreatitis* begins suddenly with pain and irregular pyrexia, and may lead to death in three or four hours, but may become chronic, and last some months. There may be a large abscess in the lesser sac. (*c*) *Gangrenous Pancreatitis*, with necrosis of the organ, is very rare. (*d*) *With*

Mumps. At the end of the first week the temperature rises again, with headache, nausea, vomiting, epigastric pain and tenderness, backache and often profuse sweating. It runs a favourable course and the symptoms subside in 4 to 5 days.

4. CHRONIC PANCREATITIS is a fibrosis of the organ which mostly runs a latent course. It may be associated with diabetes. The onset is insidious; discomfort and distension in the epigastrium are felt after meals, with drowsiness. Borborygmi and offensive stools, anæmia, and emaciation follow. Paroxysmal pain may be complained of above and to the right of the umbilicus, and tenderness can be elicited there. The pain may be referred to the left scapula. Later, the common bile duct is compressed by the pancreas and produces obstructive jaundice, with dilatation of the gall-bladder, and thus resembles cancer of the head of the pancreas. Later still, there may be pressure on the duodenum and inferior vena cava.

The diagnosis is difficult in early stages, and requires expert analysis of the excreta (see above). Later, the stools are fatty and so characteristic that the condition can be diagnosed by the naked eye.

PANCREATIC DIABETES. The most common type of diabetes mellitus is that due to disease of the islets of Langerhans in the pancreas (§ 416).

5. CANCER OF THE PANCREAS may be primary or secondary, and is a rare condition. The symptoms are: (1) Pain in the epigastric and lumbar regions is often present. At first it occurs in paroxysms, then becomes constant, and runs a chronic course. (2) Loss of weight is early and persistent. (3) Digestive symptoms, with anorexia, flatulence and periodic diarrhœa or vomiting are not directly related to meals. (4) Obstructive jaundice, intense and persistent from the pressure on the bile-duct, is present in 70 per cent. of the cases, and sometimes pain like biliary colic accompanies this. The gall-bladder is then enlarged. (5) Other symptoms as above described. (6) Later on a tumour is found in the epigastrium or in the umbilical region, with little or no mobility, deep-seated, and hard to define. (7) Œdema and phlebitis of the legs, from pressure on the inferior vena cava, and ascites may occur. (8) The spleen and liver may enlarge when their venous return is obstructed. Metastases usually occur late in the disease.

The *Diagnosis* of cancer and other tumours of the pancreas is difficult. A tumour of the liver, pylorus, or transverse colon, is more mobile; pancreatic tumours do not move with respiration. Much indicanuria points to an intestinal rather than to a pancreatic tumour. No great stress can be laid on the presence of fat in the fæces, or on glycosuria, but abundant undigested muscle fibre found in the fæces is more characteristic of pancreatic disease. Increase of diastase in the urine and steatorrhœa point to pancreatic disease.

Prognosis.—In cancer of the pancreas death usually occurs soon after the onset of jaundice, or within six weeks after ascites sets in. The complications are: (i.) Symptoms due to pressure on the neighbouring organs—intestine, pylorus, or portal vein; (ii.) sudden hæmorrhage into the alimentary tract or the peritoneal cavity; (iii.) pulmonary embolism.

Treatment is mainly symptomatic. Starches and sugars should be limited. Milk and casein are the most digestible forms of protein in pancreatic disease. The administration of pancreatic extracts may aid the digestion. Duodenal catarrh may be allayed by bismuth salicylate; and hexamine disinfects the biliary passages. In pancreatitis the jaundice has been relieved by cholecyst-gastrostomy. Other surgical measures are employed for the several diseases of the pancreas.

GENERALISED ABDOMINAL ENLARGEMENT

Difficulty in the diagnosis of the cause of abdominal enlargement can often arise in cases of obesity, constipation, pregnancy, venous congestion, atony or ptosis of the abdominal organs. And see Fallacies, § 240, for the less common sources of error.

§ 257. **Classification.**—Generalised abdominal enlargement occurs under four conditions:

I. Gas in the intestines (tympanites), or occasionally in the peritoneum	§ 258
II. Fluid free in the peritoneum (ascites)	§ 260
III. A cystic collection of fluid in the abdomen	§ 261
IV. Solid abdominal tumours	§§ 262, 263

The **Routine Procedure,** as previously described (§ 241), should be by Inspection, Palpation, Percussion, Auscultation, and Mensuration.

If a **hard tumour** can be felt in any part, turn first to § 262.

If the abdomen is quite **soft to palpation** and **resonant** all over, turn first to § 258.

If the abdomen is **dull to percussion** in the flanks, and presents a fluid thrill, turn first to § 260.

If the abdomen is **resonant in the flanks** and **dull in front,** turn first to § 261.

The abdomen is **uniformly enlarged** ; *it is* SOFT *and yielding to palpation ; percussion, systematically conducted over the whole area, gives a* RESONANT *note.* The swelling is probably due to TYMPANITES.

§ 258. **Tympanites** is the term employed for a flatulent distension of the stomach and intestines by gas. It should be remembered that flatulent distension may accompany and render obscure a *small quantity of fluid in the peritoneum.*

The *Causes* of tympanitic enlargement are as follows:

I. Atonic and other forms of DYSPEPSIA and AEROPHAGY (air swallowing) are the most frequent causes of flatulent abdominal distension. It is usually intermittent, and generally greatest after meals (§ 283).

II. In ATONY OF THE COLON the bowels are constipated, and the patient is liable to "colicky" pains; and the constitutional symptoms are few except when there has been prolonged toxæmia (§ 317).

III. In TUBERCULOUS PERITONITIS there is a tendency to the formation of intestinal adhesions and *flatulent distension.* Moreover, the distended abdomen has a doughy feel and here and there a patch of dulness on percussion, which is quite characteristic (§ 557).

IV. "PHANTOM TUMOUR" may assume the shape of a generalised more or less resonant enlargement, but it more often resembles a localised tumour (§ 262). It disappears during anæsthesia.

V. In INTESTINAL OBSTRUCTION there is considerable abdominal distension, accompanied by pain, vomiting, and other general constitutional disturbance (§§ 319 and 320).

VI. PARALYTIC ILEUS causes general distension of the abdomen (§ 319).

VII. ACUTE DILATATION OF THE STOMACH causes distension in the left hypochondrium and persistent copious vomiting (§ 295).

Gas in the Peritoneal Cavity gives much the same signs as tympanites, but there is extreme distension, and hyper-resonance all over to such a degree that the normal dulness of the liver and spleen is obscured. It

is met after perforation or rupture of some part of the alimentary canal. The patient is shocked, and presents all the symptoms associated with perforation (§ 243). A few hours after the occurrence of the perforation a delusive lull occurs in the shock and other symptoms, only to be succeeded by a fatal exacerbation. Perforation of a peptic ulcer is the commonest cause, and one of the diagnostic features of this condition is the loss of the normal area of liver dulness.

There is **uniform abdominal enlargement,** *which is soft and yielding to palpation and* DULL TO PERCUSSION *in parts; a* FLUID THRILL *is present.* There is FLUID WITHIN THE ABDOMEN.

§ 259. When there is **Fluid in the Peritoneal Cavity,** either free or encysted, the abdomen is soft to palpation, dull to percussion in parts (either in the flanks or in front), and measurements show it to be enlarged.

When the fluid is in any quantity, two special signs can be elicited. (1) *Fluid thrill.*—A thrill can be transmitted from one hand to the other, through the surface of the fluid. Place the left hand over one side of the dull portion, and tap sharply with the fingers of the right hand over the opposite side; an impulse or thrill will be felt by the left hand when fluid is present. To prevent the wave or impulse from travelling across the abdominal wall, instead of through the fluid, an assistant should place the edge of his hand vertically on the umbilicus. (2) In some cases of free fluid in the peritoneum, on suddenly dipping the fingers over a solid organ (*e.g.*, the liver), a characteristic sensation, due to displacement of fluid, can be felt. Neither of these signs can be elicited with a gaseous enlargement or a solid tumour. In *obese persons* considerable difficulty arises in the detection of fluid.

The fluid may be either (*a*) FREE in the peritoneal cavity, when it is termed ascites; or (*b*) enclosed in a CYST, such as an ovarian cyst.

(*a*) If FREE in the peritoneal cavity, it will obey the law of all fluids, and *shift with the position of the patient.* Thus in ascites (§ 260) when the patient lies on his back both flanks are dull to percussion, and the epigastric region is resonant; then, if the patient turns on one side the uppermost flank which before was dull is now resonant, while the epigastric region, if there is much fluid, is dull (shifting dulness). Much may be learned from the character of the fluid withdrawn by a cannula. Ascitic fluid is usually straw-coloured, with much albumen. Hæmorrhagic fluid usually means cancer (§ 919).

(*b*) If the fluid is ENCYSTED—*e.g.*, ovarian cyst, we can still elicit the fluctuation and the percussion tests just referred to, but the level of the dulness will not alter with the position of the patient (§ 261). In many cases fluctuation can be felt on bimanual examination. In every case a catheter should be passed to avoid overlooking a distended bladder.

There is a **generalised uniform enlargement** *of the abdomen, which gives all the* SIGNS OF FLUID, *and the fluid* ALTERS ITS LEVEL *with the position of the patient.* The condition is ASCITES.

§ 260. **Ascites** is a term applied to an effusion of fluid within the peritoneum. The physical signs of fluid have been described above. It is sometimes difficult to detect a very small quantity of fluid in the peritoneum, but its existence is rendered probable (i.) by the dulness on percussion of the umbilical region with the patient on his hands and knees; (ii.) by finding that when the patient turns from one side to the other, the flank which was dull is now resonant. On rectal examination fluid may be detected at an early stage when it has gravitated to the pelvis, and it may be detected here when it is insufficient to give other signs.

Ascites may have to be *Diagnosed* from any of the cystic conditions mentioned below (§ 261), but certainly the most frequent and important source of difficulty is *ovarian cyst* (Table XV, p. 315). Occasionally peritoneal adhesions (especially cancerous) may confine the fluid to one part of the abdomen, and then the fluid does not shift with the position of the patient. A greatly distended urinary bladder may simulate ascites, but the passage of a catheter readily excludes this fallacy.

The other *Symptoms* which accompany ascites belong to two categories: (1) Those due to pressure within the abdomen—*e.g.*, œdema of the feet and legs, from pressure on the inferior vena cava and its branches; later on dilatation of the surface veins of the anterior abdominal wall may occur from the same cause; albuminuria from pressure on the renal veins, and dyspnœa from undue elevation of the diaphragm (and often an accompanying pleural effusion). (2) There are evidences of the condition which has caused the ascites, and of all the causes by far the commonest is peri-portal cirrhosis of the liver. The temperature is generally normal, except in chronic peritonitis.

The *Causes of Ascites* are six in number. In reference to the diagnosis of these causes, if there be any œdema of the ankles, it is important to ascertain whether this œdema or the ascites came first. For instance, when PORTAL OBSTRUCTION is in operation, the dropsy of the feet will have started subsequently to the ascites; in HEART or LUNG disease it will have preceded the ascites; whereas in RENAL DISEASE they would have started about the same time. ASCITES with well-marked JAUNDICE in an old person is extremely likely to mean CANCER OF THE LIVER or peritoneum. ASCITES with SALLOWNESS of the skin in a MIDDLE-AGED person is most probably due to ALCOHOLIC CIRRHOSIS of the liver. For Ascites due to TUBERCULOUS PERITONITIS see § 557.

I. **Portal Obstruction** is the commonest cause of well-marked ascites. This is recognised in two ways: (*a*) By a history or presence of the *symptoms* of portal obstruction (of which ascites is only one); and (*b*) the presence or a history of one of the *causes* of portal obstruction.

(*a*) The SYMPTOMS of portal obstruction, in the order in which they usually appear, are as follows: (1) A liability to attacks of flatulence and of gastric and intestinal catarrh, as evidenced by pain in the stomach, flatulent dyspepsia, alternating diarrhœa and constipation, and the vomiting of mucus streaked with blood, especially in the early morning before

breakfast. (2) Hæmorrhoids and enlarged œsophageal veins. (3) Hæmorrhage, sometimes in very large quantity, from hæmorrhoids or from the stomach. (4) Congestion, and therefore enlargement of the spleen. (5) ASCITES is one of the later results. (6) Enlargement of the veins of the abdominal wall from the establishment of a collateral circulation. (7) Œdema of the legs also appears subsequent to the ascites, and is due to pressure on the large veins in the abdominal cavity by the ascitic fluid. (8) Albumen in the urine may arise in the same way, or from concurrent disease of the kidney; in the former case the albuminuria may disappear after paracentesis.

(*b*) The CAUSES of portal obstruction may be grouped into (*α*) diseases within the liver, or (*β*) diseases outside it.

(*α*) *Diseases within the Liver.*—*Cancer* is the chief cause; it produces portal obstruction usually by the pressure of the enlarged glands in the fissure, or by masses protruding from the liver. *Chronic Interstitial Hepatitis* (*Atrophic cirrhosis*) is often due to alcoholism, there being a history of this and of alcoholic dyspepsia. Simple ascites without marked jaundice or other obvious symptoms is presumptive of cirrhosis. A large *gumma* at the portal fissure may obstruct the portal vein. *Perihepatitis* sometimes produces ascites by thickening of the capsule (sugar-loaf liver). Ascites only very rarely accompanies *hepatic congestion,* and never fatty liver, hydatid, or abscess.

(*β*) The causes of portal obstruction *outside the liver* are: (1) *Cancer* of the stomach or pancreas, and various other tumours pressing on the vein. (2) Enlargement of the *glands* in the fissure of the liver (cancer, tubercle, syphilis or lymphadenoma). (3) *Thrombosis* of the portal vein is rare.

II. In **Heart Disease,** either primary (*e.g.*, mitral disease and cardiac dilatation) or secondary to lung mischief, the ascites is generally part of the dropsy affecting the cellular tissues and other serous cavities of the body. Here dropsy of the feet *will have preceded the abdominal dropsy,* and there will be a previous history of palpitation, dyspnœa, and perhaps cough. Examination of the heart will reveal the nature of the disease.

III. In **Kidney Disease** ascites may be part of a General Dropsy affecting the face, limbs, peritoneum, pleuræ, and pericardium. The fact that the dropsy started in all of these situations about the same time reveals this cause. Albuminuria is frequently enough a consequence of the pressure of the ascitic fluid, but the presence of epithelial casts almost certainly indicates that the renal disease was primary. It usually takes the form of acute or subacute parenchymatous nephritis, rarely waxy or granular kidney.

IV. **Chronic Peritonitis** is another cause of fluid in the peritoneum. An idiopathic form of chronic peritonitis is sometimes described, but it is practically never met with apart from a deposit of tubercle (in the YOUNG), § 557, or of cancer (in the AGED), § 250.

V. Chylous Ascites, or the collection of chyle in the peritoneal cavity, occurs as the result of obstruction of the thoracic duct, or it may occur after trauma, or in spleno-medullary leukæmia. In tropical countries it is more often due to *Filaria bancrofti*.

The *Prognosis and Treatment of Ascites* are very largely those of the causal condition. The *Prognosis of Ascites due to portal obstruction* depends very much on the nature of the intra- or extra-hepatic lesion which has produced it, as given above and in Chapter XII. The degree of the obstruction is measured by the amount of ascites and other symptoms present, and still better by the amount and frequency of the hæmorrhage that has taken place from the stomach or intestines. Life may be prolonged for many years even when a considerable amount of ascites has accrued, provided it has come on slowly, and time has thus been afforded for the gradual establishment of the collateral circulation through the surface veins of the abdomen and other collateral channels. It is in this sense that repeated tappings are good, for in this way time is gained for the establishment of collateral circulation. In cases of alcoholic cirrhosis the habit must be abandoned, otherwise the patient cannot live longer than six to twelve months, for ascites indicates an advanced condition of cirrhosis; cases treated early may recover.

The *Treatment of Ascites*, like its prognosis, must depend upon its cause (*q.v.*). The treatment of *ascites due to portal obstruction*, and to some extent that of other forms, is as follows: (1) Diuretics may be successful in causing fluid absorption and excretion. Urea (gms. 15–30 b.d.), diuretin, mersalyl or theocine are often effective diuretics, but they must be used cautiously. (2) Salt-free diet often helps to prevent re-accumulation of fluid. (3) Paracentesis is generally necessary sooner or later. Some physicians say it should be put off until it is called for by the urgency of dyspnœa. In cancer this is certainly a good rule, but in cirrhosis of the liver it is best to operate at once in all cases where there is much fluid, unrelieved by medicine. Often diuretics which were useless before, are efficacious after the operation, because the kidneys are relieved from pressure. Sometimes recovery takes place after repeated paracentesis, because time is thus afforded for the establishment of the collateral circulation. It is best to use a small trocar with the tube conducted to a pail, so that the peritoneum may gradually empty itself. With a large trocar leakage may remain, or peritonitis ensue. By the Talma-Morison surgical method the omentum is transplanted between the layers of the abdominal wall to facilitate anastomosis between the vessels of the portal and the systemic circulations.

There is a **generalised abdominal enlargement** *which gives all the* SIGNS OF FLUID (§ 259); *but the fluid does* NOT ALTER ITS LEVEL *with the position of the patient.* There is ENCYSTED FLUID IN THE ABDOMEN.

By far the commonest of such cystic tumours is an OVARIAN CYST. Other and less common cystic abdominal tumours are PREGNANCY WITH HYDRAMNIOS, CYSTIC FIBROMA of the uterus, HYDRO- and PYO-NEPHROSIS,

PANCREATIC or MESENTERIC CYST, a large HYDATID, a MUCOCŒLE of the GALL-BLADDER, and an ENCYSTED ASCITES.

§ 261. I. **Ovarian Cyst** is centrally situated, and grows from below upwards. It is attached to the pelvic organs, it can be moved laterally but not upwards. The enlargement is fairly uniform, and gives all the signs of fluid (§ 259). But the level does not alter with the position of the patient; and whereas the umbilical region is dull on percussion (" horse-shoe shaped dulness "), the flanks are resonant. On palpation it is tense and elastic, and in malignant ovarian cysts nodules can be felt in the walls. Ballottement between the two hands on combined abdominal and pelvic examination is often a most useful sign.

TABLE XV.

DIFFERENTIAL DIAGNOSIS OF ASCITES AND AN OVARIAN CYST.

	Ascites.	*Ovarian Cyst.*
Inspection.	Flanks bulge, front flat.[1]	Flanks flat, front bulges.
Percussion.	Flanks dull, front resonant. On turning, upper flank becomes resonant.	Flanks resonant, front dull. No alteration of dulness on turning.
Measurement.	Umbilicus to xiphoid greater than umbilicus to pubes. Circumference at umbilicus greater than slightly below. Navel to iliac spine same both sides.	Umbilicus to xiphoid less than umbilicus to pubes. Circumference at umbilicus less than slightly below. Navel to iliac spine greater one side than the other.

The features associated with it are (1) a history of it having grown upwards from the pelvis, and (2) these tumours (unlike encysted ascites) may be of very rapid growth, and reach quite a large size in three or four months. (3) There have usually been menstrual irregularities, though by no means always. (4) The cyst may be clearly felt by bimanual examination of the pelvis. There may have been no general symptoms of any kind, but generally some pain and local discomfort have been complained of. Often when the cyst contains pus there is little or no fever. When there is a history of attacks of pain, it generally indicates adhesions, an important matter to the operator. An examination of the uterus usually reveals nothing. A malignant papilliferous cyst is indicated by (1) the presence of nodules in the walls; (2) the age of the patient, a history of emaciation, and severe pain; (3) later ascites and œdema of the legs.

Diagnosis.—In the *earlier stages* the diagnosis of an ovarian tumour is sometimes difficult. It is an elastic, movable, and globular swelling; the uterus is not enlarged and can be defined as quite separate from the tumour. In this stage it may have to be diagnosed from *hydro-* or *pyosalpinx. Pelvic peritonitis* and *cellulitis* and *pelvic hæmatocele* form a swelling which is very firmly fixed in the pelvic cavity and accompanied by constitutional symptoms. In *extra-uterine fœtation* there would be

[1] Bulging in front may occur in cases with large and acute effusion.

morning sickness, a patulous os uteri, and other symptoms of pregnancy, with an empty uterus, and a positive Zondek-Aschheim Test.

In the *later stages* ovarian cysts have to be diagnosed from all the conditions mentioned below.

II. PREGNANCY WITH HYDRAMNIOS and a thin uterine wall is sometimes very difficult to diagnose from an ovarian cyst, for both develop very rapidly. Experienced clinicians have been known to fail in the differentiation. The symptoms of pregnancy (see § 447), the exactly central position of the tumour, and the softened cervix, may aid. The test for pregnancy and, later, an X-ray examination, settle the diagnosis. *Hydatid mole* presents similar difficulties, but it is fortunately rare.

III. LARGE CYSTIC FIBROID of the uterus, especially of the subperitoneal (pedunculated) variety, may produce the signs of a fluid tumour. It is recognised by (1) its connection with the uterus, which is enlarged; (2) its slow growth, which may extend over many years; and (3) menorrhagia in some cases.

IV. A LARGE HYDATID CYST of the spleen or liver, a HYDRO- OR PYONEPHROSIS, a dilated GALL-BLADDER, a large PANCREATIC, OMENTAL, OR MESENTERIC CYST, or a large PERITYPHLITIC ABSCESS, may on rare occasions produce the appearance of a general fluid enlargement of the abdomen, and may require to be diagnosed from ovarian cyst; but they are nearly always *asymmetrical.* They grow from, and their percussion dulness is continuous with, the organs whence they rise; they are referred to among Abdominal Tumours (§ 263).

V. ENCYSTED ASCITES is not common. It may result from previous peritonitis, of which there will probably be a history. More frequently, perhaps, it results from tubercle or cancer of the peritoneum (§ 250). In all of these there is a want of symmetry in the enlargement and in the fluid, an absence of the associated symptoms of ovarian tumour, and a history or other evidences of the cause in operation.

VI. PNEUMOCOCCAL PERITONITIS in children may form an encysted swelling, but this is accompanied by a swinging temperature.

The *Prognosis* of ovarian tumour is always serious, though in the non-malignant form it may be quiescent for some years. If not treated, a cyst may (1) rupture and produce peritonitis; (2) it may become infected; (3) the pedicle may become twisted; (4) hæmorrhage may take place into its cavity; (5) occasionally it bursts into the bowel with sinus formation.

The *Treatment* is entirely surgical. The earlier the cyst is removed the better. It is well to do this before the occurrence of attacks of pain indicate inflammatory adhesions.

ABDOMINAL TUMOURS.

§ 262. **Method of Procedure.**—We now turn to the second group of abdominal enlargements—namely, those in which the enlargement has originated in, or is localised to, one part—*i.e.*, Abdominal Tumours. It is only by repeated and careful examination that mistakes can be avoided in the diagnosis of abdominal tumours. The same methods are adopted here as in general enlargement (§§ 241, 257). (1) *Inspection* in the recumbent, and sometimes in the erect, posture *should never be omitted*; (2) *Palpation* to determine its size, position, borders, mobility and texture. This requires a flat hand previously warmed and with the patient's abdominal muscles thoroughly relaxed by a suitable posture; (3) *Percussion*, to define the resonance or dulness of the tumour; (4) Careful *Measurement* made and

recorded, both for the comparison of one part with another, and to note the progress made by the growth; (5) *Auscultation*, which is especially useful in the diagnosis of late pregnancy; and (6) *Examination under an anæsthetic* is sometimes required.

Fallacies of Abdominal Tumours.—(1) *Obesity* may offer a serious obstacle to the examination of abdominal enlargements or tumours. In these cases the umbilicus is usually depressed. The only way to arrive at a correct decision is to place the hand flat upon the abdomen and then dip the fingers suddenly and forcibly inwards.

(2) The presence of *fluid* within the abdomen, together with a solid tumour, may prevent our discovering or examining the latter thoroughly. It is best to re-examine after paracentesis has been performed.

(3) *Pregnancy* and a *distended bladder* are frequent sources of error.

(4) *Gas in the intestine* causes enlargement in the lower abdomen, with marked resonance to percussion.

(5) *Fæcal accumulations* may simulate malignant and other tumours, though they can generally be indented by the fingers. They are always situated in some part of the large bowel. Give a course of castor oil or other purgative, or repeated enemas.

(6) A "*phantom tumour*" is a swelling (usually tympanitic, sometimes dull), produced by irregular muscular contraction of one or both recti muscles, and it is wonderful how precisely it may simulate a solid tumour. It is apt to appear and disappear suddenly, hence the name. The condition is met for the most part in young hysterical women, and is usually beyond the control of the patient. It is a frequent cause of error in diagnosis. Spasm of the diaphragm may produce a generalised abdominal enlargement by pushing the viscera down. The patient should be placed in a position of perfect ease for the relaxation of all the muscles of the body, with the knees drawn up and the neck slightly bent. Sometimes an anæsthetic is required in order to establish the diagnosis.

(7) The *liver* occasionally presents an extra lobe (§ 263. I.). Displaced or movable organs may be mistaken for tumours.

Having excluded these fallacies, and being satisfied as to the existence of an abdominal tumour, there are five points to which our attention should be directed:

1. The first and most important question is the *locality of the tumour*, in which region is it situated, or where did it start?

2. To ascertain with *which organ it is connected*, consider what organs are located in the region occupied by the tumour, and then see if it be structurally continuous by palpation and percussion with one of these.

3. If it *moves with the breathing* of the patient we know that it must be connected with the diaphragm, or some organ depressed by it during respiration, such as the spleen, liver, gall-bladder, stomach, intestines, kidney, or omentum. If fixed, it is a tumour of the pancreas, aorta, lymphatic glands, or some other organ unaffected by respiration, or bound down by adhesions.

4. Inquire for a *history of any disease* or functional disturbance of the abdominal organs—*e.g.*, in the case of the kidney, whether the urine contains, or has contained, blood or pus; or perhaps there has been jaundice, pointing to hepatic mischief. Inquire also whether the tumour is constantly present or appears intermittently.

5. The diagnosis of the *nature of the tumour* depends very largely upon its history and the age and sex of the patient. Tense cystic tumours are extremely difficult to differentiate from solid growths, but we can try to obtain the percussion and fluctuation tests (§ 259). There is also another question which very frequently presents itself for consideration —viz., is the tumour benign or malignant? The general symptoms of malignant disease (cancer) are discussed in § 555; but the age of the patient, and the rapid course and lethal tendencies of the disease, are the chief means of differentiating it.

§ 263. *If there is a visible or palpable* **tumour,** *in the abdomen, ascertain which* REGION *the tumour chiefly* OCCUPIES *or* ORIGINATED *in, and refer to that region in the following summary. Having identified* ITS ORIGIN *in this way, reference must be made to the diseases of the organ affected to ascertain the* NATURE *of the tumour.*

I. RIGHT HYPOCHONDRIUM.—The commonest tumours in this position are tumours of the *liver*, especially cancer and enlargement of the organ. The features which HEPATIC TUMOURS present in common, in addition to their position, are: (1) They are not covered in front by resonant bowel, and their dulness is continuous with that of the liver; (2) they move with respiration; and (3) there are ascites, jaundice, or other evidences of liver derangement. It must not be forgotten that hepatic tumours may be simulated when the liver is pushed down by pleural, pericardial or subphrenic effusions; or that it may be puckered by contraction of the capsule, and so simulate a tumour or enlargement (Diagnosis of Hepatic Enlargements, § 343 *et seq.*); Riedel's lobe (see below) is another fallacy. A distended GALL-BLADDER (*e.g.*, by cholecystitis) is recognisable as a tense pear-shaped swelling below the ninth costal cartilage. There is only occasionally a history of biliary colic but often a history of "chills" (biliary fever, § 354). It is distinguished from the kidney by the fact that the colon passes over the kidney. Tumours in this region may also be connected with the *duodenum* or *right kidney* (see II. and IV.).

Riedel's Lobe of the Liver.—In certain cases, sometimes associated with gall-stones retained within the gall-bladder, a tongue-shaped process projects downwards from the right lobe of the liver. It may reach as far as the iliac crest, or even to the iliac fossa. In hardly any of the cases has the condition been correctly diagnosed before operation. It has most often been mistaken for floating kidney, and has also been taken for distended gall-bladder, hydatid cyst, renal or omental tumour. It is sometimes tender, its shape more or less that of a pear. Under anæsthesia its connection with the liver may possibly be made out. By X-ray its shadow may obscure the pyelogram of a normal right renal pelvis, leading to the mistaken diagnosis of renal tumour.

Suprarenal Tumours become manifest in the right or left hypochondrium, and are difficult to distinguish from tumours of the liver, gall-bladder, kidney and spleen

respectively. The symptoms consist of: (i.) Pain radiating across the abdomen and to the back; (ii.) pain referred to the shoulder tip; (iii.) emaciation, with nervous depression, and digestive disturbance; (iv.) a tumour felt beneath the costal margin, at first movable with respiration, but soon becoming fixed; it can be felt posteriorly in the costo-vertebral angle; (v.) absence of urinary and gall-bladder symptoms. Early diagnosis is aided by excretion urography, the tumour depressing the corresponding kidney and giving filling defects in the renal pelvis. The injection of air into the loin, followed by a skiagram, has also revealed tumours of the adrenal—a procedure to be undertaken only by experts.

Suprarenal Tumours arise in the medulla and in the cortex. The important *medullary* tumours are (*a*) the pheochromocytoma which is associated with hypertensive properties, and (*b*) the neuroblastomata. The latter arises chiefly in children; a striking feature is their extensive metastases. In the *Hutchinson syndrome* they arise in the left adrenal and form metastases in bones, especially the ribs, skull, lungs and liver; exophthalmos and ecchymosis of the left eye may be the first sign. In the *Pepper syndrome* the tumour arises in the right adrenal; metastases occur mainly in the liver and become enormous. *Cortical* tumours cause local pressure effects; some lead to sexual manifestations; in children, precocious growth and sexual development, which must be distinguished from similar conditions due to hypothalamic tumours. In female adults, carcinoma of the adrenal may give rise to symptoms similar to Cushing's disease; in male adults, signs of feminism may develop. Adrenal hyperplasia (bilateral) is associated with *virilism*, which is more marked when symptoms arise before than after puberty. There is a general masculinisation of the female: hirsutics, muscular development, changes in voice, and diminished female characters, *e.g.*, deficient mammary glands, amenorrhœa, either primary or secondary, or other menstrual disturbances: such tumours are characterised by a positive Ponceau-Fuchsin staining reaction (Vines).

II. In the Epigastric Region tumours may be connected with the liver (*vide supra*); but the first tumour which would occur to one's mind would be Cancer of the Stomach—*i.e.*, a hard, irregular swelling attended by vomiting, "coffee-ground" in character. The commonest form of malignant disease of the stomach, however, is scirrhus of the pylorus, in which visible peristalsis, copious vomiting at long intervals, and other gastric symptoms appear before any swelling can be detected (§ 294).

Pancreatic cysts may cause a fluctuating swelling in the epigastrium, but their detection is extremely difficult. There may be a history of pain, and symptoms of pancreatic disease (see § 256). Cysts of the *small omental sac* present a similar swelling. *Pulsation in the epigastrium* may be due to hypertrophied right ventricle but is usually normal; rarely it is caused by abdominal aneurysm.

III. In the Left Hypochondrium tumours of the spleen originate and sometimes attain an enormous size (§ 357). They move with respiration, and they make their way forward in *front* of the colon towards the umbilicus. A splenic tumour can generally be moved forwards by getting the hand behind it, a fact which distinguishes it from tumour of the left kidney, and it presents the characteristic splenic notch. It resembles tumour of the left lobe of the liver, but the latter cannot be displaced downwards by the hand. Other tumours in this position may be connected with the *stomach*, *pancreas*, *liver*, *kidney*, and *sigmoid flexure*.

IV. The Lumbar Region may be the starting place for Renal Tumours, which are characterised by four features: (i.) Their comparative fixity during respiration. (ii.) Dulness in one flank, and, unless both

kidneys are involved, resonance in the other. (iii.) They are *always resonant in front*, because as they make their way forwards and downwards they push the colon in front of them; and (iv.) there is no resonant part between the dulness of a renal tumour and the spine, as there would be in the case of a splenic tumour. In many the rounded and reniform shape of the kidney is retained. They are distinguished from hepatic tumours by the dulness in the flank not being continuous with that of the liver, and by the presence or history of blood, pus, or other urinary changes. The commoner forms of renal tumours are hydro- and pyonephrosis, congenital cystic kidney, renal sarcoma (commonest tumour in children), and perinephric abscess. A perinephric abscess tends to point backwards. *Pyo-* or *Hydronephrosis* are cystic tumours, containing urine *with* or *without* pus respectively (§ 424). Hydronephrosis may be almost painless, not tender, and unattended by subjective or constitutional symptoms; pyonephrosis is always tender, and attended by hectic fever (unless the abscess is chronic). Hydatid of the kidney may only be evidenced by swelling; sometimes it gives a thrill on percussion. Other tumours starting in the lumbar regions may be connected with the *ascending* and *descending colon.*

Movable or *Floating Kidney* is one of the most frequent of abdominal tumours, especially on the right side. It descends with inspiration, slips back into position during expiration, and may be found as low as the iliac fossa. Its mobility and rounded or reniform shape, are characteristic, but not always easily detected. There is a characteristic pain of a dull, aching, or dragging character in the back, increased by exertion (see § 253).

V. The LEFT ILIAC REGION may be the seat of a tumour caused by CANCER of the SIGMOID FLEXURE, and this is the most frequent position in the bowel for cancerous growth. Cancer and other *tumours of the large intestines* are distinguished generally by their free mobility (unless fixed by adhesions). They are, when cancerous (far the commonest neoplasm of the intestines), attended by irregularity of the bowels, generally alternating constipation and diarrhœa. The commonest starting-point for primary cancer of the bowel is the sigmoid flexure; but before a cancerous swelling can be detected in the left iliac region the patient will have been troubled with recurrent diarrhœa and pain, sometimes melæna. These symptoms are followed in course of time by œdema of the leg or sciatica. In *cancer* of the peritoneum the intestines may become matted together, and although fluctuation may be detected, there may be little fluid in the peritoneal cavity. Sarcoma of the *small intestines* is rare and usually only diagnosed by laparotomy. The prognosis of cancer is given in Chapter XVI. So-called "colloid cancer" of the peritoneum is a remarkable exception in regard to duration of life; it may last for years. Treatment is not very hopeful. See "Emaciation." While diverticulosis may occur in any part of the alimentary canal, *diverticulitis* may show a swelling due to adhesive peri-diverticulitis or abscess formation which is difficult to distinguish from cancer. (See § 321.)

VI. The RIGHT ILIAC REGION is the position in which APPENDICITIS

is usually manifested; it is fully described under "Abdominal Pain" (§ 249). *Intussusception* of the bowel, which occurs mostly in childhood, generally arises in this region, but the tumour is most commonly felt under the liver (§ 319). *Pelvic peritonitis* may form a firm swelling in either iliac region. Its other features are (i.) vaginal examination reveals a tender swelling in the corresponding fornix, pushing the uterus to the opposite side; (ii.) there is a history of acute pain and fever at the onset, frequently following childbirth or abortion. *Gumma, tubercle* and *cancer of the cæcum*, contrary to what we might expect, often constitute a *movable* tumour in the iliac region, and are apt to be mistaken for a mass of fæces. Cancer or *actinomycosis* of the cæcum may be attended by suppuration, so giving rise to abscess with pyrexia. The history of such cases may run a long course, and resemble appendicitis. Enlarged *glands* and *Crohn's disease* may be mistaken for appendicitis. Iliac *abscess* in Pott's disease may point in this region. A right movable kidney may simulate a tumour.

VII. The UMBILICAL REGION is the starting place of tumours connected with the pancreas, duodenum, mesenteric glands, and aorta, all of which are *immobile during respiration;* though a tumour in this position is far more often connected with the stomach, liver, or transverse colon, which *move with respiration.* Enlargement of the *mesenteric glands* may be sometimes detected in spare subjects by grasping the two sides of the abdomen either between the two hands or the finger and thumb of one hand. When large enough to form a tumour, they are fixed and matted together.

Aneurysm of the Abdominal Aorta is a pulsatile and expansile swelling, immobile during respiration. In thin subjects a thrill may be felt, and a murmur heard. In auscultating the abdominal aorta we must be careful not to produce a murmur by pressure of the stethoscope. It is attended always by a severe fixed neuralgic pain in the spine, and sooner or later breathlessness and cardiac signs. It is these latter symptoms which distinguish true aneurysm from "pulsatile aorta" (see below), and from a swelling in front of the vessel to which the pulsation has been communicated. An endeavour should be made to grasp the swelling on each side, so as to confirm the expansile nature of the tumour.

Pulsating Abdominal Aorta (throbbing in the belly).—Dyspeptic subjects and nervous females are often troubled with marked pulsation of the abdominal aorta, which is sometimes obvious both to the patient and the doctor. There is in this affection great local discomfort, and even pain, with marked pulsation, obvious to both inspection and palpation. The diagnosis from aneurysm rests partly on the fact that the pulsation is not limited to any part of the aorta, and partly, that such rapid and violent action of the heart is not common in aneurysm.

VIII. The HYPOGASTRIC REGION is the situation whence BLADDER, UTERINE, OVARIAN and TUBAL TUMOURS grow. *Ovarian tumours* (which are nearly always cystic) are usually characterised in the *early stages* by their free mobility, unless they are malignant, and their rapid growth (§ 261). *Tumours of the bladder* are usually rendered sufficiently obvious by changes in the urine and by passing a catheter. *Tumours of the uterus* are similarly revealed by uterine symptoms, excepting perhaps some subperitoneal fibroids. These may reach a large size without any

symptoms at all; their origin and relations are detected by bimanual examination. *Pregnancy* causes a symmetrical enlargement, starting from the hypogastric region about the third month of gestation. Among the rarer tumours are pelvic hydatid and pelvic hæmatocele.

The Nature, Prognosis, and Treatment of these various abdominal tumours are discussed under the organs with which they are connected.

§ 264. **Flattening or Recession of the Abdomen** is not a sign of any great importance. "Ventre plat, enfant il y a," is a French expression signifying that the abdominal wall slightly recedes during the first two or three months of pregnancy. It is met with in abstinence from food, and in wasting disorders, such as with dehydration, cancer and tubercle. It may be present also in intestinal, hepatic, and renal colic, and as a consequence of excessive purging or vomiting. A hollow or "boat-shaped" abdomen is often characteristic of meningitis in infants. It may also occur when acute general peritonitis is present, especially in children.

CHAPTER X

THE STOMACH

IT is to be noted that the alimentary tract, apart from the mouth, pharynx and rectum, is not subject to direct examination by ordinary methods. Much progress has followed the use of test meals (§ 279) and examination by X-ray; and in expert hands the œsophagus and stomach, and the sigmoid may be brought into direct vision by œsophagoscopy, gastroscopy and sigmoidoscopy. In ordinary practice we are largely dependent upon subjective symptoms in the investigation of disorders of the stomach. However, the patient's sensations before and after meals are not necessarily related to his stomach, for gastric symptoms are frequently not of gastric origin, but associated with disease of the heart, kidney, lungs, gall-bladder, duodenum, appendix or internal secretory glands. On the other hand, derangements of the stomach produce widespread effects in the general economy. The nutrition, of course, fails; but, apart from this, sufferers from gastric disorders are liable to prostration and depression. In chronic disorders of the stomach the functions of the nervous system may be so profoundly disturbed by neurasthenic and other symptoms that the physician may overlook the primary cause of the mischief—namely, mal-assimilation of food. The stomach and digestion are influenced by two sets of nerves—the sympathetic and vagus; their relationship and equilibrium may be disturbed by (1) reflex conditions, (2) asthenia of the nervous system, (3) endocrine secretions, and (4) emotions.

PART A. SYMPTOMATOLOGY

The symptoms which reveal disorders of the stomach may be **local** (viz., epigastric pain or discomfort, nausea or vomiting, hæmatemesis, dryness or bad taste in the mouth, thirst, flatulence, heartburn, hiccough, water-brash, altered appetite); or **general** and **remote** (viz., cardiac symptoms, various nervous derangements, skin symptoms, and emaciation).

Among the **Local Symptoms** of gastric disorder, PAIN OR DISCOMFORT AFTER FOOD, and NAUSEA OR VOMITING, are the most constant and important—*i.e.*, the cardinal symptoms. HÆMATEMESIS is less frequent, but more serious. Other local symptoms are also of value in diagnosis.

§ **270. Gastric Pain,** or discomfort, in diseases of the stomach, is a most important *local* feature. Although it is not in every case sufficiently constant in its characters to enable us to establish the diagnosis, nevertheless it merits the closest study. In some cases it is altogether absent (even when simple ulcer or malignant disease exists), but when present,

the features which should be noted are its *position*, its *character*, its *degree*, its *constancy*, and above all, its *relation to the taking of food*.

Its *Position* is usually over the epigastrium, but pain is very frequently complained of between the shoulders, and very severe pain in the back may also occur. A localised pain with tenderness occurs with ulcer. In its *character* it varies considerably. Sometimes it is like a dull weight or a feeling of distension, such as occurs in nervous dyspepsia and chronic gastritis; or it may be of a burning character, as in hyperchlorhydria; or it may resemble abdominal cramp, as in spasm of the pylorus (§ 246), or in some cases of nervous dyspepsia. Sharp or lancinating pain of a persistent character usually attends ulcer or cancer of the stomach.

Its *Relation to Food* is by far the most important feature of the pain in gastric diseases: (*a*) *It comes on at once* and lasts a variable time in nervous (atonic) dyspepsia, in acute and chronic gastritis and in ulcer (simple or malignant). In simple ulcer pain may come on soon or as long as two hours after the meal, varying with the site of the ulcer; the pain is at once relieved by vomiting—a characteristic feature; and solids usually give more pain than liquids. In gastric ulcer the sequence generally is food, ease, pain, ease till food is taken again. (*b*) When pain *comes on an hour or more after food*, it is due to excessive acidity, either from hypersecretion or fermentation (organic acids). In hypersecretion, pain is relieved by taking food and alkalies. Pain coming on late after food is common in duodenal ulcer (hunger pain), and the sequence tends to be food, ease, pain lasting until the next meal. A similar pain may be caused by chronic appendicitis or gall-stones. (*c*) Pain *coming on without time relation to food* is characteristic of nervous dyspepsia, and is met with in carcinoma of the stomach. If deep pressure *over the seat of pain* relieves it, the condition is probably functional, not organic.

Fallacies.—Pain of the acute type may be mistaken for *biliary colic*, but in that condition the pain is greater on the right side, and is sometimes followed by jaundice. In *hepatic* disorders, pain is more often limited to the right hypochondrium. The spine should always be examined for *caries*, especially when stomach pain is complained of by children. The pain in such cases is referred to the terminations of the intercostal nerves. The gastric crises of *tabes dorsalis* may be mistaken for simple gastritis. Pain in the *chest* (§ 33) must not be mistaken for abdominal pain. *Diaphragmatic hernia* (§ 109) and other causes of *dysphagia* must be thought of. *True angina pectoris* and *coronary thrombosis* might be mistaken for that type of dyspepsia in which the stomach is distended with gas and hampers the heart's action. In acute *pancreatitis* there is extreme pain of sudden onset in the left hypochondrium, and the case usually terminates fatally in a few days (§ 245). Other pancreatic diseases are also attended by pain in the situation of the stomach.

§ **271. Nausea or Vomiting** is, after pain, the most frequent and most definite symptom of stomach disorders. Its causes may be grouped under three headings: (*a*) Local, (*b*) Nervous, and (*c*) Toxic. Waterbrash

(§ 273) is sometimes spoken of by the laity as "vomiting," but is not true vomiting. Regurgitation from a dilated œsophagus or œsophageal pouch is another fallacy; the food returns easily and is not acid in reaction. Prolonged coughing may induce vomiting; patients may complain of vomiting, and the physician may be led in consequence to treat the stomach instead of the lungs.

(*a*) LOCAL CAUSES of vomiting include: (1) *Errors of diet*, such as shell-fish, infected food, excess of alcoholic, fatty, and other irritating foods. Under these circumstances the vomiting of the peccant material occurs soon after ingestion. (2) *Irritant and corrosive poisons* and *emetics* also speedily give rise to vomiting. The diagnosis of this cause is aided by (i.) an examination of the vomit, which should *always be preserved;* it may smell of phosphorus (which is luminous in the dark), or of carbolic, or other acids. (ii.) An examination of the mouth for any corrosive action. (iii.) The occurrence later of the toxic effects peculiar to the several poisons; and (iv.) a history of poisoning obtained from the patient or his friends. (3) *Fermentation* of the contents of the stomach, such as that met with in dilatation due to pyloric obstruction, when the vomiting may occur at very considerable intervals, sometimes of a day or two; the vomited matter is copious, frothy, and contains sarcinæ and yeasts. (4) *Diseases* such as acute gastritis, cancer, and simple ulcer are usually accompanied by vomiting. In chronic gastritis of alcoholic origin mucus is vomited chiefly in the early morning. (5) *Acute dilatation of the stomach* may come on more or less suddenly in early life, or in states of general weakness and toxæmia, as after operation or in pneumonia, with repeated vomiting and symptoms of collapse, resembling intestinal obstruction. It is a serious condition, often fatal unless relieved by continuous gastric suction.

(6) *Acute duodenal ileus*, an obstructive condition of the third part of the duodenum due to compression by the superior mesenteric vessels, occurs with dilatation of the stomach, profuse vomiting, epigastric distension, and later severe prostration. It must be treated on the same lines as acute dilatation of the stomach, with continuous gastric suction by a tube left in the stomach, and with the foot of the bed raised. (See also § 319, IV.)

(7) Persistent vomiting and marasmus in young infants are the two chief symptoms of **Congenital Hypertrophic Stenosis of the Pylorus.** The symptoms commonly begin about the end of the second week of life—(i.) projectile vomiting, which cannot be stopped; (ii.) progressive emaciation; (iii.) constipation; and later (iv.) visible peristalsis of the stomach. (v.) A small hard nodule (the hypertrophied pylorus) may be palpated under the upper part of the right rectus. Careful feeding, lavage, and methyl atropin nitrate (eumydrin), beginning with 0·5 c.c. and increasing by 0·5 c.c. to 2·5–3·0 c.c. of 1/10,000 solution half an hour before meals q.i.d., often effect a cure. Toxic symptoms, such as abdominal distension, bouts of fever, dilatation of the pupils, indicate a reduced dose. Rammstedt's operation gives good results, but it must not be left as a last resource.

(*b*) VOMITING OF NERVOUS ORIGIN may be classified under two groups —cerebral and reflex.

That due to a CEREBRAL CONDITION. 1. *Hysterical Vomiting* may

follow any or every kind of food, no matter what its quantity or quality may be; or perhaps digestible articles like milk will cause vomiting, while indigestible foods like pickles may be retained. Sometimes this vomiting resembles a simple regurgitation or possetting, as compared with the urgent vomiting of organic disease, the symptoms of which are wanting.

2. In *Migraine* and *Bilious Headache* the patient perhaps awakens with a headache, and vomits only mucus and bile (merely an indication that the vomiting is urgent, or that the stomach is empty).

3. Another important cause of vomiting is *Organic Cerebral Disease* (§ 827)—*e.g.*, tumour, early meningitis, abscess, Ménière's disease. This is recognised by: (i.) The vomiting occurs without relation to food; (ii.) it is urgent and projectile; (iii.) there may not be nausea; (iv.) the vomiting may be excited by simple change of posture; (v.) the presence of other cerebral symptoms, such as vertigo and perhaps optic neuritis (§ 850). Vomiting may also attend the gastric "crises" of *locomotor ataxy*. It occurs at intervals, and is usually severe. It is recognised by the absence of the ankle and knee jerks and the presence of other symptoms of the disease (§ 817). Vomiting may be associated with *glaucoma* (§ 855), which is easily overlooked.

4. *Mountain* and *aviator's sickness* is due to *anoxæmia*.

Reflex vomiting from *visceral irritation* may be met in a great many abdominal disorders, such as peritonitis, pancreatitis, intestinal, biliary, or renal colic; in all stages of intestinal obstruction, in strangulated hernia, and with intestinal new growths. In the last named the attention of the physician is often drawn from the true source of trouble. It occurs also with pregnancy, uterine and ovarian disorders. Pharyngeal irritation, especially in alcoholics and smokers, leads to prolonged hawking often followed by vomiting.

(*c*) Toxic Causes are uræmia and jaundice and the onset of some of the acute specific fevers. The vomiting of Addison's disease, hyperthyroidism, and pernicious anæmia comes under this heading. Certain cases of vomiting in pregnancy are due to toxæmia. After anæsthetics vomiting may be urgent; sometimes this is due to blood in the stomach, and will cease when it is expelled or washed out.

The *Treatment* of vomiting must be directed to its cause, but there are certain measures which can be applied to relieve the symptom. The patient should be kept at rest in the horizontal position, and without food, or only given milk in small quantities at a time, and iced water. Milk diluted with barley-water, whey, or citrated milk are given where ordinary milk is not retained. Among the remedies which may be employed are effervescing mixtures, alkalies, hydrocyanic acid, bismuth, minim doses of ipecacuanha or liq. iodi (in a teaspoonful of water), opium, and acetanilide (especially in the vomiting after anæsthetics), sod. bicarb. gr. 60 to Oi water, seidlitz powder (if the vomiting be due to constipation) or calomel. In some cases champagne is helpful. Bromides and valerian aid nervous vomiting; a mustard-leaf applied to the epigastrium

may also be useful. Washing out the stomach with warm water or normal salt solution often gives relief, especially in acute dilatation. For *Sea-sickness*, navigan, cafinal, chloretone and hyoscine gr. $\frac{1}{100}$ by mouth are recommended.

Cyclical or Recurrent Vomiting, "acidosis," is a not uncommon condition in children. The attacks may occur at regular intervals of 2, 4 or 8 weeks. Predisposing causes: (i.) there is often a family history of the same condition or of allergy or migraine; (ii.) thin, highly-strung, lordotic children are much more susceptible. Precipitating causes: (i.) constipation, over-eating of fatty foods, eggs, chocolates; (ii.) over-fatigue, excitement, over-strain at school, riding in cars and trains; (iii.) the onset of any infection, commonly in the throat or at the onset of one of the specific fevers; when arising in the appendix the differential diagnosis may be difficult and a surgeon should be consulted. The condition is associated with defective function of the liver. An attack comes on suddenly, with headache, pallor, repeated vomiting and retching, followed by abdominal pain, some pyrexia, drowsiness, and if the vomiting persists, dehydration and a rapid thready pulse; the breath smells sweet from the presence of acetone, and acetone and diacetic acid are found in the urine (Ketosis, § 384). Cases have been mistaken for meningitis and for acute abdominal conditions.

Treatment.—The child should be kept at rest in a darkened room, and the bowels freed with grey powder or an enema. Frequent small sips of glucose in water or in a fruit drink (1–2 oz. to 1 pint) must be given by mouth, with small doses of alkaline carbonates and citrates. In severe cases rectal or intravenous glucose may have to be used. Any associated infection must be treated. To prevent attacks, the above-mentioned predisposing conditions must be dealt with.

§ **272. Hæmatemesis** (Vomiting of Blood).—Bleeding from the stomach, unless in slight quantity, is usually accompanied by nausea and vomiting. In the first place, it is important to decide whether the blood really comes from the stomach and œsophagus.

Sources of Fallacy.—(1) Blood from the *lungs* may be mistaken for blood from the stomach (see Hæmoptysis, § 104). (2) *Epistaxis*, the blood running down the gullet and being vomited, is a common fallacy in children, in whom the blood is apt to be swallowed. This may follow operations on the tonsils or teeth. Epistaxis is recognised by making the patient blow his nose: there are no abdominal symptoms. (3) Blood from the *fauces* or *gums*, especially when the gums are spongy, or when pyorrhœa alveolaris exists, may give rise to a sanguineous vomiting or expectoration, the cause of which is very apt to be overlooked even by competent observers; but the blood is mixed with saliva, and is rarely large in amount. (4) Blood from a fracture of the base of the skull and from œsophageal disease may also be swallowed and vomited. On the other hand, *hæmorrhage from the stomach* is (i.) preceded by a feeling of faintness and nausea, and (ii.) followed by melæna (tarry stools).

(iii.) Blood from the stomach is mixed with food, and mostly brown ("coffee-grounds"), though it may be red if the quantity is large (*e.g.*, in ulcer) or if food has been brought up before the blood. (iv.) There is no history or local sign of pulmonary disease, and there may be a previous history of disease or derangement of the stomach or liver.

The *Causes of Hæmatemesis* may be roughly divided into (*a*) those which produce a slight or protracted hæmorrhage, and (*b*) those which give rise to a large quantity at one time.

(*a*) **Slight or Protracted Hæmorrhage** occurs chiefly in Chronic Gastritis and Cancer. A temporary irritation or congestion of the stomach produced by irritating articles in the food or by urgent vomiting (*e.g.*, with migraine), may be attended by *streaks* of blood in the vomit. A smaller hæmorrhage may occur in cases described in group (*b*) below.

I. CHRONIC GASTRITIS is known by (i.) vomiting in the morning—often viscid mucus streaked with blood—or at other times. (ii.) It may be accompanied by, and due to, disease of the liver (cirrhosis), or advanced cardiac disease, and is found especially in alcoholic subjects (see § 284): syphilis is a rare cause.

II. CANCER OF THE STOMACH OR ŒSOPHAGUS is recognised by: (i.) The patient is usually beyond middle age; (ii.) pain is complained of—severe, constant, and generally worse after food; (iii.) the blood vomited is rarely copious, but typically "*coffee-ground*" in character, and may recur for weeks; (iv.) the hæmatemesis is followed by melæna unless the blood is scanty, and occult blood is usually present in the fæces; (v.) there is progressive cachexia; (vi.) an abdominal tumour or evidence of cancer elsewhere, *may* be found (see also § 294).

(*b*) A **Large Hæmorrhage** at one time may occur in Ulcer of the Stomach or Duodenum, Portal Cirrhosis of the Liver, Splenic Anæmia, Gastrostaxis, Purpura, Chronic Nephritis, or after taking Chemical Irritants.

III. PEPTIC ULCER, OF THE STOMACH OR DUODENUM, OR OF THE JEJUNUM AFTER GASTROENTEROSTOMY.—(i.) The bleeding is copious, the vomit is often bright red, after being brown at first, and melæna follows; (ii.) there is usually a history of indigestion, sometimes of operation; but hæmatemesis may occur in the previously healthy.

IV. ATROPHIC CIRRHOSIS of the liver (by causing portal obstruction) (§ 342). The hæmatemesis may be slight, but it is more often very copious—the most copious met with, as it is of venous origin, from ulceration of varices of the lower œsophagus.

V. OTHER CAUSES OF PORTAL OBSTRUCTION (see § 260)—*e.g.*, tumour pressing on the portal vein. This, as with cirrhosis, is known by the other symptoms of such disease—*e.g.*, increasing ascites, splenic enlargement, jaundice, and diarrhœa. Thrombosis of the portal vein is rare and hard to diagnose. It gives rise to sudden onset of signs of portal obstruction.

VI. ANEURYSM OF THE AORTA, or of one of its branches, leaking into the œsophagus or third part of the duodenum. This is known by (i.)

possibly a previous history of aneurysmal symptoms (§ 80); (ii.) the blood is copious; (iii.) sudden death usually occurs; but in certain other cases there is a small recurrent leakage from the aneurysm for a few days or weeks preceding death.

VII. Gastrostaxis.—Under this title are included cases of hæmatemesis, occurring usually in young anæmic women, due to capillary oozing. Such cases were formerly thought to be due to gastric ulceration, but more frequent operations and post-mortem examinations have shown that no ulcer is present. Hypertrophic gastritis may be seen with the gastroscope.

VIII. Morbid Conditions of the Blood, such as nephritis, yellow fever, malignant forms of the specific fevers, purpura, leukæmia, and hæmophilia.

IX. Splenic enlargement in the early stage of splenic anæmia, even before the liver is involved.

X. Chemical Irritants (*e.g.*, arsenic, strong alkalies, and mineral acids), or mechanical injuries from articles which have been swallowed. In susceptible individuals acetylsalicylic acid (aspirin) can produce an acute gastric erosion, especially if swallowed in small lumps which lodge between folds of mucous membrane.

In the *Differentiation* of the causes of hæmatemesis (1) examine the stomach and duodenum for ulcer; (2) examine the liver, especially for cirrhosis; (3) examine the chest for aneurysm or other mediastinal growths which may have perforated the œsophagus; (4) ascertain the approximate quantity of vomited blood, and then review the case, remembering the possibility of simulation in neurosis.

Prognosis.—Hæmatemesis is usually a serious symptom, but its gravity depends upon the cause. As regards the lesion, aneurysm is the most grave of the causal conditions; then, in order, cancer, morbid blood states, cirrhosis, and peptic ulcer. In chronic ulcer of the stomach and duodenum the mortality from hæmatemesis may be 10 per cent. Grave prognostic signs are marked pallor, air hunger, rapid thready pulse, and repeated vomiting or melæna.

Treatment.—The indications are: (i.) to stop the hæmorrhage. The patient must be kept absolutely at rest in the horizontal position. An ice bag should be placed over the epigastrium. Morphia hypodermically is the best hæmostatic and relieves anxiety: it must be repeated to allay restlessness. Thromboplastin or calcium salts may be injected. Adrenalin 30–60 min. in 1 oz. ice-cold water by mouth, repeated each 1–2 hours, is the best local hæmostatic. (ii.) To combat shock and allay distressing thirst, rectal glucose-salines and axillary salines are essential. In profuse hæmorrhage, when shock is severe, blood transfusion by the drip method is necessary. Intravenous gum-saline should only be used if blood is not available. The fall in hæmoglobin of the blood is not a good measure of the necessity for transfusion, since in acute hæmorrhage, although the blood volume falls, dilution of the blood by absorption of tissue fluids occurs later. It is inadvisable to give more than 2 pints of blood at a time for

fear of restarting the hæmorrhage./ (iii.) By the mouth nothing is given until 24 hours after major bleeding has ceased: some allow sips of iced water or ice wrapped in muslin to be sucked. Then iced citrated milk may be started. Meulengracht has advised much more liberal feeding (§ 297. III); and some give 3 pints of citrated milk each 24 hours to prevent starvation and dehydration and to keep gastric acidity low. Opinions still differ as to which method is preferable. (iv.) Aperients or enemas must never be given until after the lapse of several days. Liquid paraffin by mouth or rectal washouts are permissible. (v.) General treatment must include scrupulous care in the toilet of the mouth, to prevent parotitis, and the subsequent administration of iron. (vi.) If hæmorrhage recurs in spite of treatment, surgical treatment is probably advisable.

§ **273.** The **other Local Symptoms** of gastric disorder are of considerable diagnostic value.

1. BAD TASTE IN THE MOUTH, most noticeable in the morning, and DRYNESS OF THE LIPS are often complained of in gastric disorders. Sleeping with the mouth open must be excluded.

2. HALITOSIS (foul breath) may be due to bronchiectasis: recent work suggests the importance of the absorption via the small intestine of offensive volatile products of fat digestion—a condition cured by reducing the fat intake to 40–60 G. a day. A *tainted breath* may also be due to dental caries, pyorrhœa and septic tonsils (and see § 201).

3. THIRST occurs in dyspeptic conditions with acute dilatation of the stomach, inflammatory stomach lesions, and in all cases of persistent vomiting.

4. FLATULENCE is a distension of the stomach or intestines by gas, which may be brought up by the mouth or passed by rectum.

Symptoms.—Flatulence causes *local* symptoms of discomfort and distension, and *remote* symptoms such as palpitation and cardiac irregularity.

Etiology.—Gastric flatulence is a common symptom in chronic gastritis, gall-bladder dyspepsia, and in some nervous individuals without gastric derangement in whom there is repeated swallowing of air and subsequent "belching" of gas. Intestinal flatulence may be the result of swallowed air, of excessive fermentation of starches and sugars, and of constipation or diarrhœa; paralytic ileus, cœliac disease and sprue give a flatulent distension of the abdomen often without any complaint from the patient.

Treatment necessitates removing the cause. Carminatives such as brandy, spir. ammon. aromat., ginger, peppermint and phenol may aid: magnesia helps the flow of bile as well as emptying the small and large intestines, and so Gregory's powder is particularly helpful. Charcoal aids intestinal flatulence.

5. "HEARTBURN" and ACID ERUCTATIONS are usually met with together. Heartburn is a burning sensation passing up from the epigastrium to the pharynx, and sometimes mouthfuls of acid fluid are brought up at the same time. It is due to superacidity and partial regurgitation of the gastric contents into the lower end of the œsophagus.

Causes.—Superacidity, or "acid risings," may be of two kinds. (*a*) *Organic acids* are met with in diseases where there is *deficient* gastric secretion—some forms of atonic dyspepsia, chronic gastritis, cancer, and dilatation of the stomach. HCl is a germicide, and when from any cause it is absent, bacteria flourish; fermentation ensues within a *few hours* after food, and is accompanied by pain in the epigastrium. The three principal acids are: butyric, lactic, and acetic.

(*b*) Hyperchlorhydria, or *excessive secretion of HCl*, is met with in one form of acute dyspepsia, and is usually present with duodenal ulcer. Here, the pain or "gnawing" occurs *before* meals, and is temporarily relieved by food (see also § 285).

(*c*) In pregnancy it may be a persistent symptom, and is often relieved by acid hydrochlor. dil. 15 ♏., spir. chlorof. 10 ♏. in water after food.

The *treatment* of 3 and 4 is discussed in § 285.

6. Hiccough.—Normally the opening of the glottis synchronises with the contraction of the diaphragm, and consequently there is no hindrance to the free entry of air. Hiccough is caused by a spasm of the diaphragm which occurs at irregular intervals and sometimes at the moment of closure of the glottic aperture. The characteristic cough is then heard. The important causes of persistent hiccough are: (1) Reflex stimulation of the phrenic nerves by gastric or colonic flatulent distension or irritation after hot, peppery foods and with hepatic disease. (2) Irritation of the peritoneum, as in peritonitis, general or local, near an inflamed abdominal organ, or in typhoid fever. (3) Disease of the thoracic viscera, especially diaphragmatic pleurisy. (4) Toxic blood states, notably uræmia. (5) Neurosis. To this cause are assigned certain cases for which no more adequate reason is apparent. (6) Hiccough may also occur as a symptom of hysteria, of cerebral tumour and meningitis. *Encephalitis Lethargica* may show itself first with persistent hiccough. (7) Persistent hiccough may also arise from central or peripheral irritation of the phrenic nerve by spinal tumours. (8) Epidemic Hiccough, probably an infection of the central nervous system, clears up spontaneously, without sequelæ. Severe cases should have morphine, when the hiccough prevents sleep. The patients are usually intensely alarmed and need reassurance.

Prognosis.—Hiccough is not as a rule a serious symptom. In abdominal disease it is of grave import. In the terminal stages of uræmia, meningitis, or cerebral tumour, persistent hiccough may herald exitus. Epidemic hiccough may resist all treatment; it exhausts the patient, and may be the immediate cause of death.

Treatment.—The simplest forms of treatment are those directed to producing definite physiological contractions of the diaphragm. These are such well-known methods as sipping water and holding the breath, or inhaling CO_2. Anything which gives rise to a feeling of suffocation may cause a forcible contraction of the diaphragm, and so stop the spasm; for this reason tickling the nares and taking snuff have been tried, often with success. The hiccough due to dyspepsia is readily cured with bicar-

bonate of soda and peppermint, and that of colonic distension, by colonic lavage. If these measures fail, or if the hiccough recurs frequently, a thorough investigation is called for. When no causal condition can be found and the hiccough continues to be severe, one may give sedative drugs by the mouth, or, if necessary, by the rectum ; the bromides, the acetanilide group, amyl nitrite and tinct. opii or $\frac{1}{10}$ gr. apomorphine (subcutaneously) are successful. Peripheral stimuli, such as blisters to the epigastrium, pinching the lobe of the ear, forcible pulling forward of the tongue, and digital pressure on the vagus in the neck, may be tried ; and the abdomen may be bound tightly with a bandage or with adhesive strapping. A general anæsthetic may have to be administered.

7. "WATER-BRASH" (Pyrosis) is the name given to a clear alkaline fluid expelled from the mouth in gushes, most often in the morning. Sometimes it is expelled without any kind of straining, but more often it is attended by retching. It is probably a reflex hypersecretion of saliva swallowed during the night, due to irritation in the stomach. It is met in many dyspeptic conditions, and fairly often with *peptic ulcer*.

8. ANOREXIA (Loss of Appetite) is not always an indication of stomach disease, as it is present in many general constitutional disturbances, such as infectious fevers, tuberculosis and malignant disease. Its chief clinical importance lies in its presence in the early stage of *gastric cancer*. In *cancer* and *chronic gastritis* there is sometimes no appetite before a meal or a premature feeling of fulness after a few mouthfuls. In *ulcer* there is sometimes a fear of taking food. HYSTERICAL ANOREXIA (Anorexia Nervosa) is known by : (i.) general failure of appetite or refusal to eat ; (ii.) pronounced loss of weight ; (iii.) constipation ; (iv.) slow pulse ; (v.) growth of downy hair on limbs and face ; (vi.) it occurs mostly in young females, in whom there is amenorrhœa, depression and restlessness ; (vii.) careful investigation reveals no organic condition (see §§ 554, 888).

INCREASED APPETITE is often met, as Shakespeare pointed out, in gastric disorders. It is found in some cases of chronic gastritis and dilated stomach, in acromegaly, pregnancy, and during convalescence. A FALSE APPETITE which is satisfied with the first few mouthfuls of food is sometimes met in subacute and chronic gastritis, owing to irritation of the mucous membrane. BULIMIA or ravenous appetite is seen in diabetes, in neuroses of the stomach, after acute gastritis, in wasting disorders such as sprue, in phthisis, intestinal worms, and Graves' disease. PERVERTED APPETITE, excessive fondness for acids and sweets, or desire to eat objects such as chalk, pencils, or hair, may occur in hysteria and pregnancy.

§ 274. **General** or **Remote Symptoms** are usual.

1. GENERAL MALAISE and a sense of ill-health and incapacity for work are among the earliest and most constant accompaniments of all derangements of the digestion, whether functional or organic. The dark rim beneath the eyes, and the sallow "earthy" complexion, so frequently associated with town-dwellers, are quite as often due to dyspepsia, just as this latter is often due to defective teeth or to the insufficient use of

them. EMACIATION is not common in gastric disorder, though in chronic cases there is some loss of flesh. It appears early in cancer of the stomach, and is severe in anorexia nervosa.

2. The CARDIAC SYMPTOMS met with in dyspepsia are palpitation, pain in the region of the heart (angina innocens); dyspnœa, syncope, and vertigo; intermission of the cardiac rhythm. Cough may occur, due to pharyngeal catarrh or reflex irritation. Collectively, these symptoms may give rise to the impression that the case is one of cardiac valvular disease, although the heart may be structurally healthy (Roemheld's syndrome).

3. FUNCTIONAL DISTURBANCE OF NERVOUS SYSTEM.—*Headache and depression of spirits* are frequently met in all forms of dyspepsia. A sense of general ill-health and irritability of temper out of all proportion to the local mischief attend most gastric disorders, and, where stomach symptoms are not prominent, may lead the physician away from the true cause. Many of the symptoms of *neurasthenia* may result from gastric disorder.

4. DIARRHŒA may accompany stomach disease when the gastric contents are of an irritating nature, and when achlorhydria is present (gastrogenous diarrhœa). CONSTIPATION is usually found with simple ulcer, cancer, and chronic gastritis. But a more usual condition is an IRREGULARITY of the bowels, accompanied by borborygmi (rumbling in the bowels).

5. The URINE invariably exhibits signs which reveal disturbances of metabolism. The commonest of these is an excess of URATES; in other cases PHOSPHATES and OXALATES are found. In these circumstances dyspepsia is a predisposing cause of renal and vesical calculus.

6. SKIN SYMPTOMS.—General *pruritus* may accompany some forms of gastric derangement. *Flushing* of the face after meals is met in gastric disorders, especially in women. Acne rosacea is common with dyspepsia. The face may be swollen so that the case appears like one of acute nephritis; but the sudden onset, without much constitutional disturbance, and early disappearance on curing the indigestion, distinguish it from that disease. *Urticaria* occurs in certain individuals after eating indigestible articles, and with several forms of gastric disorder (§ 609).

PART B. PHYSICAL EXAMINATION

Disorders of the stomach are investigated by Inspection, Palpation, Percussion, Auscultation, X-ray examination after an opaque meal, the Gastroscope, Examination of matters vomited or withdrawn from the stomach by a tube, by Test Meals and by Fæcal Analysis.

§ 275. Inspection.—(1) The *Teeth* in all cases must be closely examined. Common causes of indigestion are bolting the food, defective or absent teeth, septic tonsils and other forms of oral sepsis. See § 204 and § 209.

(2) The *Tongue* and its diseases have been described (§ 212). At one time the tongue was thought to indicate the state of the stomach, but it is a more certain indication of the patient's general condition. But

even in this, allowance has to be made for certain variations—namely: (i.) a coated tongue is normal to some, even in health, and a clean tongue in others may be associated with disease; (ii.) certain diets—*e.g.*, milk—produce a coated tongue; and (iii.) certain habits—*e.g.*, smoking and alcoholism—also coat the tongue. The mouth may show signs of poisoning by corrosive acids.

(3) Inspection of the epigastric region may reveal a tumour, or the peristaltic movements of a dilated stomach. Aortic pulsation may be transmitted by a pyloric tumour, although no bulging is visible.

(4) In skilled hands the flexible gastroscope may be employed to examine the interior surface of the stomach. Alterations in the mucous membrane, mucus secretion, hæmorrhages, ulceration or neoplasm may be demonstrated.

§ 276. **Palpation** requires considerable experience. The patient's shoulders should be supported and his arms relaxed by the sides, and he should be instructed to open his mouth, to draw up his knees, and to "let his breath go." [1] Talking to him is useful to distract his attention. The warm hand should be laid quite *flat* upon the abdominal wall. Thus one can detect the presence of a tumour, tenderness, or other abnormality. Sometimes it is helpful to have the patient supported on hands and knees, and to palpate upwards.

Gastric Succussion or *Splashing* is made out by placing one hand on each side of the stomach, and suddenly pressing inwards the finger-tips of each hand alternately: listening over the stomach at the same time with a stethoscope materially aids this sign. Otherwise it can be detected by vigorously rolling the patient from side to side. Splashing can be *normally* elicited during the process of digestion—*i.e.*, during the first hour or two after a meal, especially if much fluid has been taken. But if succussion can be elicited after that time, it suggests that there is delayed emptying of the stomach.

§ 277. **Percussion** of the stomach is not very satisfactory or precise.

Surface Anatomy of the Stomach. (Fig. 67 and § 240.) The cardiac orifice lies behind the seventh left costal cartilage 2½ inches from the mid-line. The fundus occupies the left dome of the diaphragm and lies behind the apex of the heart. As this part of the stomach always contains gas, it is resonant (Traube's space). The body of the stomach is vertical, and turns sharply into the pyloric antrum. The pylorus lies opposite the first lumbar vertebra in the transpyloric plane just to the right of the mid-line. The greater curvature is extremely variable and depends on the state of filling; it may reach below the umbilicus in normal conditions. The rough outline of the stomach resonance may be defined after giving successively the two portions of a seidlitz powder dissolved separately. In this way dilatation (§ 295) may be distinguished from gastroptosis (§ 296).

[1] Some say it is better to have the legs extended loosely; general anæsthesia may be necessary in obscure cases.

§ 278. The **Motor Functions** of the Stomach and Intestinal Tract are most accurately investigated by X-ray examination after an opaque meal.

There is considerable individual variation. Delay in the alimentary canal may be tested by giving a teaspoonful of charcoal the night before a test breakfast. Charcoal so given should appear in the fæces in thirty-six to forty-eight hours. If it does not appear on the second morning, an enema should be given. The presence of charcoal in the returned enema shows delay in the lower colon; if it is not present, the delay is higher up. This test is not very accurate.

X-RAY EXAMINATION is carried out with the fluorescent screen after giving 3 ounces of barium sulphate suspended in a suitable sweetened medium. Radiograms taken can be studied afterwards. The *barium meal* is seen passing down the œsophagus and any obstruction or diverticulum is noted. The outline, position, tone and the rate and character of the peristaltic movements of the stomach are observed, the time at which it is empty, and the passage through the pylorus and duodenum. Irregularity of outline may be seen and local tenderness felt if there is a growth of the stomach, and if the barium lodges in the crater of an ulcer. During the filling or emptying of the stomach, the folds of mucous membrane are defined, and give valuable information. The normal shape of the duodenal cap is characteristic, and is altered by ulcer, adhesions or pressure from without, as by a distended gall-bladder. The position and mobility of the lower ileum and cæcum are observed, and the appendix may be seen filled. The passage of the barium through the colon is watched at intervals. Normally the stomach empties in three-and-a-half to five hours; the terminal ileum and cæcum begin to fill about the same time. The terminal ileum should be clear of material four hours after the stomach is empty, and the colon should be clear in seventy-two hours. Abnormal appearances of the stomach, duodenum and colon are seen in Figs. 68 to 73. With a *barium enema*, the colon is observed filled and after evacuation, and if necessary with air inflation.

§ 279. **Examination of Stomach Contents.**[1]—First, as to the CHEMISTRY OF DIGESTION, and the practical information to be derived from clinical examination of the stomach contents. *Four processes* normally take place in the stomach: (1) The conversion of starch into sugar, begun in the mouth, is carried a stage further; (2) proteins are changed into peptones; (3) fat globules are set free from their envelopes; (4) milk is curdled. Delay in digestion may be caused by (1) deficient peristalsis of the stomach walls, (2) deficient quality or quantity of the gastric juice, (3) the consumption of indigestible articles, or (4) the dilution of the gastric juice by drinking too much fluid at meal-time.

The gastric juice contains HCl, water, pepsin, rennin, mineral salts, a *little* mucus, and Castle's intrinsic factor (§ 539). Pepsin and rennin exist in the secretory cells only as zymogens, which, on secretion into the stomach, become active ferments or enzymes. In the healthy state, as the result of digestion, about 30 c.c. of fluid should be obtained from the stomach one hour or so after a test-meal (*vide infra*), straw-coloured, without much odour, without organic acid, and with about 0·2 per cent. of free HCl.

As regards *hydrochloric acid*, much depends on the time of examination. *Hyperchlorhydria* has come to be somewhat loosely used for "excessive acidity," and thus to be confused with the acidity of fermentation (due to organic acids). On the other hand, after a meal, a negative result on testing for HCl would indicate the absence of peptic activity, as an acid is required for the normal digestive action of pepsin. Excess of HCl is distinctive of pyloric or duodenal ulcer. HCl is diminished in catarrhal conditions of the mucous membrane, in many anæmias, in the majority of cases of malignant disease, during pregnancy, and in states of nervous exhaustion.

Three organic acids are met in the presence of fermentation in the stomach, *lactic acid*, *butyric acid* and *acetic acid*. *Lactic acid* is most easily recognised on testing with Uffelman's reagent, and is the only one of diagnostic importance. It is normally absent in the gastric juice after digestion has proceeded for one hour, but traces may

[1] It is not possible here to give more than a brief outline of this important subject.

be found, due to the ingestion of lactic acid in certain foods, or to fermentation in the mouth. Fermentation occurs when HCl is deficient or when there is delayed emptying of the stomach: lactic acid is most frequently found in cases of gastric cancer with achlorhydria.

The secretion of *pepsin* is not interfered with, unless there be destruction of the glands of the stomach. An acid secretion without peptic activity does not occur.

Renninogen and Rennin are diminished or absent in the later stages of gastritis and cancer. The amount of rennin is directly proportional to the quantity of pepsin.

Examination of Gastric Contents after a Test-meal is a useful method of investigating the secretion and emptying of the stomach. The gastric contents should be tested in all doubtful cases of digestive disturbance.

The **fractional test-meal** yields information as to the gastric secretion, the emptying of the stomach and the neutralisation of excessive acid by the reflux of alkaline duodenal contents. A soft rubber tube (Ryle), with an oval perforated bulb at the end, is swallowed, any resting contents are withdrawn with a small glass syringe, and then a pint of test gruel is drunk with the tube in position. (The gruel is made with two tablespoonfuls of breakfast oatmeal mixed with one quart of water, and boiled down to one pint and strained.) The tube is kept in position whilst the patient reads quietly. During the next two to three hours, at intervals of a quarter of an hour, about 10 c.c. are drawn up and placed in a numbered test tube. If three or four tubes have shown no acid with congo-red indicator, histamine may be injected subcutaneously (0·25 mg. histamine or 1 mg. histamine acid phosphate), in order to excite secretion. The contents of the test tubes are separately examined and a curve plotted. Record the appearance, smell, consistency, and presence of excess of mucus, bile, or blood in each specimen. MICROSCOPICALLY, we can detect fat globules, starch cells, vegetable and muscle fibres, residues of delayed emptying, cells of the mucous membrane, torulæ cerevisiæ or sarcinæ, and pus cells. Epithelial cells may be in excess in carcinoma. The Oppler-Boas bacillus may sometimes be seen on examination under the high-power lens. CHEMICALLY, the stomach contents are normally acid, although 4 per cent. of otherwise normal people have no acid in the gastric juice. The normal acidity is due to *free hydrochloric acid*, much of which is loosely combined with proteins. In the absence of free HCl, the acid present is due to organic acids, such as lactic and butyric acids produced by fermentation in the stomach (*e.g.*, in gastric carcinoma), and this is combined with protein. The sum of the free acid and the combined acid gives the *total acidity*.

To estimate the *free hydrochloric acid*, titrate 5 c.c. of the filtered gastric contents with N/10 solution of caustic soda, using a 1 per cent. solution of dimethylamido-azobenzol as the indicator, and add the alkali till the pink colour is discharged. Then add phenolphthalein as an indicator, and add more NaOH till the red colour of the phenolphthalein is developed. The amount of alkali added in the first instance is a measure of the amount of free HCl present, and the total amount of alkali added is a measure of the free + combined acid, *i.e.*, the total acidity. The results are usually expressed in terms of c.c. N/10 NaOH per 100 c.c. gastric juice, and in the fractional method of gastric analysis, may be plotted in the form of a graph.

Lactic acid may be detected by adding Uffelmann's reagent (made by mixing a little 5 per cent. solution of carbolic acid with a few drops of liquor ferri perchloridi). The blue colour is discharged by lactic acid and a yellow colour is produced.

PART C. DISEASES OF THE STOMACH, THEIR DIFFERENTIATION, PROGNOSIS AND TREATMENT

§ 280. Routine Investigation. FIRST: We must identify the patient's LEADING SYMPTOMS as being referable to gastric disorder (see Part A).

SECONDLY: Inquire as to the HISTORY, and especially whether the symptoms came on *acutely* and recently, or whether, as is more usual,

the illness came on insidiously, and has run a *chronic* course. Much depends on the skill and method with which the history is elicited. Inquire particularly as to pain or discomfort and its relation to meals, and as to the other symptoms mentioned in Part A.

THIRDLY: Proceed to the PHYSICAL EXAMINATION, and ascertain whether there be any localised tenderness and pain, and whether any tumour or other abnormality be present.

If the patient's symptoms have come on gradually, and lasted a considerable time, turn to **Chronic Disorders** of the Stomach (§ 283).

If, on the other hand, his symptoms have begun somewhat suddenly and recently, the case is probably one of the two **Acute Disorders** of the Stomach: I. ACUTE DYSPEPSIA; or, II. ACUTE GASTRITIS.

I. *The patient—whose temperature is normal—complains of* NAUSEA, GASTRIC DISCOMFORT, *headache, and depression, which have come on suddenly; there is a little epigastric tenderness.* The disease is probably ACUTE DYSPEPSIA.

§ **281. Acute Dyspepsia** ("Bilious Attack," "Congestion of the Liver") consists of a sudden disturbance of the digestion in a previously healthy person, such as occurs in association with surfeit, high living or other errors in diet.

The *Symptoms*, which come on suddenly, are: (1) Pain, or a feeling of oppression or distension in the epigastrium, occasionally accompanied by slight tenderness on pressure. (2) Nausea and vomiting often follow. (3) Headache, depression, anorexia, coated tongue, constipation, scanty urine loaded with urates. (4) The illness is sometimes preceded and accompanied by drowsiness, and there is often a history of previous similar attacks.

The *Diagnosis* is easy, the only similar condition being acute gastritis, in which the constitutional symptoms are more apparent, the duration of the illness considerably longer, and the *tenderness much more marked.* Irritant poisoning comes on more suddenly with urgent vomiting (§ 271). Similar symptoms may usher in certain infectious diseases.

Etiology.—(1) Too large a meal, especially after previous fatigue. (2) Errors in diet, such as excess of alcohol (which retards digestion), fats, ice, and many other articles which vary with the idiosyncrasy of the individual.

Prognosis and Treatment.—Acute dyspepsia usually passes off in two or three days. (1) If pain be present, assist vomiting by mild emetics, such as copious draughts of salt and water, tickling the fauces, etc. Violent emetics aggravate the condition. (2) Three grains of calomel or blue pill, and milk diet for a day or two, generally relieve. (3) Bismuth and tonics may be given during convalescence.

II. *The patient complains of considerable* PAIN *or discomfort, and* TENDERNESS IN THE EPIGASTRIUM, *with nausea or vomiting, all of which have come on somewhat suddenly.* The disease is probably ACUTE GASTRITIS.

§ 282. **Acute or Sub-acute Gastritis** is relatively a much more serious disorder than the foregoing. It consists of a sudden derangement of digestion due to inflammation of the mucosa of the stomach.

Symptoms.—(1) Pain, intense and burning, or a feeling of distension in the epigastrium, coming on directly after food, and accompanied by tenderness on pressure. (2) Vomiting, not always immediately after a meal, of undigested food, sometimes with streaks of blood. (3) Malaise, anorexia, slight pyrexia, headache, depression, and other constitutional symptoms may be present, attended sometimes by great prostration, thirst, furred or coated tongue. (4) Diarrhœa may ensue after a day or two.

The *Diagnosis* may have to be made from acute dyspepsia (§ 281), and from other causes of vomiting (§ 271).

Etiology.—(1) In the majority of cases simple acute gastritis is caused by errors in diet, or by decomposing (or infected) meat; alcohol or an excessive quantity of normal food also causes it. (2) Irritant poisons (*e.g.*, arsenic, antimony, phosphorus, etc.). In long-continued vomiting, without apparent cause, poisoning should be suspected, and the vomited matters examined. (3) In some cases, gout and other constitutional conditions predispose to or determine an attack. Heart, lung, and liver diseases are predisposing causes.

Prognosis.—Recovery generally takes place in about three to six days, the affection rarely lasting longer than eight or ten days. It may go on to chronic gastritis. Death rarely takes place, excepting from irritant poisoning or in cases of membranous gastritis.

Treatment.—The indications are: (1) To remove any irritant that may be present in the stomach. This can be done by promoting vomiting, especially if the epigastric pain continues. The stomach may be washed out with saline or a weak solution of bicarbonate of soda. It may be desirable to give a purgative, such as 3 grains of calomel (if there is vomiting, ½ grain doses hourly), followed by a seidlitz powder next morning. Hot fomentations or a mustard leaf to the epigastrium may relieve the pain. (2) The second indication is rest to the stomach, which is gained by twelve or twenty-four hours' abstinence from food, followed by fruit juice and glucose, and then milk in small quantities. Later on, bismuth combined with opium is the best treatment. The milk diet should be supplemented only very gradually.

CHRONIC DISORDERS OF THE STOMACH

§ 283. *The patient, whose temperature is normal, complains of* "**Chronic Indigestion,**"—i.e., *pain or discomfort in some way connected with his food, which has probably come on gradually, and may have lasted a long time.*

Note the relationship of the discomfort or pain to food and examine for tenderness. Guidance may be obtained from the following summary.

TABLE XVI.

The patient complains of SUBSTERNAL PAIN on SWALLOWING FOOD	Dysphagia	§ 219
The discomfort FOLLOWS MEALS, and *is* RELIEVED by *vomiting and belching*	Chronic gastritis ..	§ 284
There is DISCOMFORT and PAIN, RELIEVED by *alkalies and by food*	Acid gastritis	§ 285
There is PAIN, AGGRAVATED BY FOOD and there is TENDERNESS *on the left*	Gastric ulcer .. .. or gastro-jejunal ulcer	§ 289 § 291
The pain is RELIEVED BY FOOD and there is TENDERNESS *on the right*	Duodenal ulcer ..	§ 290
The pain, 3–4 hours after food, is associated with NAUSEA, VOMITING, and *distension on the right side*	Duodenal ileus ..	§ 292
There is CONSTANT DISTENSION in the epigastrium and *attacks of pain on the right side* ..	Duodenal diverticulum	§ 293
There is pain, which is constant and AGGRAVATED BY FOOD; there is FLATULENCE and NAUSEA, UNRELIEVED *by* BELCHING	Cholecystitis	§ 354
There is DISCOMFORT following meals, variable in position, with FLATUS which passes downwards	Colitis	§ 310
There is PAIN which FOLLOWS EFFORT	Angina pectoris ..	§ 51
There is severe and INTERMITTENT PAIN, *not connected* with FOOD or EFFORT	Gallstones and tabetic crises	§§ 353, 817
Pain is more or less CONSTANT, with DISTENSION and FLATULENCE:		
Cancer of the stomach		§ 294
Chronic dilatation of the stomach		§ 295

Many disorders **unconnected with the stomach** may give rise to symptoms of chronic indigestion; among these the following may be mentioned: Pulmonary tuberculosis (of which dyspepsia is often the earliest symptom), Appendicitis, Colitis, Anæmia, Abdominal Tumour, Cardiac, Hepatic, Renal or Uterine Disease, various Nervous Disorders, and Pancreatic Disease (rare).

I. *The patient complains of* CHRONIC INDIGESTION, *and the epigastric pain or discomfort comes on* SOON AFTER A MEAL. The disease is probably CHRONIC GASTRITIS.

§ **284. Chronic Gastritis** is the commonest form of chronic dyspepsia. It was formerly called atonic or nervous dyspepsia. The *symptoms* are: (1) Pain or distress, usually in the epigastrium, coming on immediately or very shortly after food—especially fried and greasy food. The pain may be in the back or shoot up to the shoulders; or there may be no definite pain, only a feeling of weight or distension. It may be accompanied by tenderness and is often relieved by eructations of wind. (2) The appetite is usually diminished; it may be good, but ceases quickly after beginning the meal. Often breakfast is well taken, lunch not so well, and later meals worse. (3) There is a bad taste in the mouth. The tongue is flabby, dry and indented by the teeth. (4) There is a tendency to eructation and heartburn; nausea, even vomiting may occur, but not frequently. (5) There are languor, headache, depression, disturbed sleep, ready fatigue, and general discomfort and drowsiness after meals. There may be palpita-

tion, dyspnœa and other cardiac symptoms: sometimes acne rosacea and urticaria.

There are three stages: First, a simple *congestion*, in which the hydrochloric acid is diminished. The second stage is one of *mucous catarrh*, in which there is a large secretion of mucus, and hydrochloric acid is almost completely absent. In the third stage there is *atrophy* of the



FIG. 68. *Right.*—Hypertrophic gastritis of greater curvature, showing the feathered appearance indicated by arrows. *Left.*—The same with the stomach filled.

mucous membrane; both hydrochloric acid and pepsin are now absent. Pernicious anæmia may be associated with this stage; some believe malignant disease may follow in after years.

Etiology. (1) Errors of diet, including deficiency of vitamin B; (2) defective teeth and inadequate mastication; (3) acute and chronic febrile diseases, sepsis, anæmia, infected teeth or tonsils. Dyspepsia is often the earliest symptom met with in pulmonary tuberculosis. (4) Abuse of tobacco and alcohol. (5) Circulatory diseases, early hypertension, nephritis, and various abdominal disorders—*e.g.*, appendicitis,

chronic colitis, gall-bladder disease, abdominal or pelvic tumour. (6) It may be part of organic disease of the stomach, such as ulcer, syphilis or early pyloric cancer.

Diagnosis. The subjective symptoms are not characteristic, and chronic gastritis is usually secondary to disease elsewhere in the body. Some forms of chronic gastritis do not give rise to digestive symptoms, the patient complaining only of weakness and exhaustion. Test meal, gastroscopic and X-ray examinations are necessary for the diagnosis of the state of the stomach. There is excess of mucus and usually diminution of hydrochloric acid, while the strial picture of the stomach shows thickening or thinning of the mucosal folds. Alcoholic and acid gastritis are special varieties (see II and III below). The important stomach conditions to be differentiated are *gastric ulcer* in the young, and *cancer of the stomach* in the middle-aged and old (see Table XVII, p. 345). *Achylia gastrica* is the late stage of chronic gastritis and may be accompanied by acne rosacea, visceroptosis and ileal stasis: it also occurs in Addisonian anæmia.

II. *In addition to other symptoms of* CHRONIC INDIGESTION, *the patient has much nausea, and* VOMITS MUCUS IN THE MORNING, *occasionally streaked with blood. The disease is probably* ALCOHOLIC GASTRITIS.

Alcoholic gastritis is produced by persistent dietetic errors, especially alcoholic excesses, and is aggravated by the venous congestion arising from cirrhosis of the liver.

III. *The patient complains of* BOUTS OF INDIGESTION, *in which the discomfort does* NOT *come on* SOON AFTER A MEAL, *is followed by* VOMITING OF ACID FLUID, *and is* RELIEVED BY FOOD *and by alkalies. The discomfort consists of* SINKING FEELINGS *in the epigastrium, or* HUNGER PAINS. *The disease is probably* ACID GASTRITIS.

§ **285. Acid Gastritis** (Acid Dyspepsia, Hyperchlorhydria) is due to causes which bring about directly or reflexly excessive secretion of gastric juice, or retention with pyloric spasm. Among these are nervous strain and worry; alcohol, tobacco and condiments; colitis, appendicitis, cholelithiasis, gastric or duodenal ulcer, and duodenal diverticulum. In *achylia gastrica* there may be acid eructations, but these are due to organic acids formed by fermentation.

The *Prognosis of Gastritis* depends on the cause and the duration of the symptoms. It is never fatal, but often renders life wretched for the sufferer. If met early, treatment should be thorough; if untreated, dilatation of the stomach, general malnutrition and neurasthenia may develop. Simple alcoholic gastritis soon recovers. The outlook is more grave when due to general toxic states or when there is irremovable venous obstruction.

Treatment of Gastritis.—(1) Remove the cause. Correct faulty habits of chewing and bolting the food, remove infected teeth and provide efficient dentures, treat any catarrh of the nose or infection of the tonsils. Reduce or prohibit alcohol and tobacco. (2) Local treatment: Gastric lavage with water or sod. bicarb. (gr. 60 to ℥i); hydrogen peroxide (♏ 30 to ℥i) may be used. (3) Substitution therapy with hydrochloric

acid, ℳ xv with bitters before meals in suitable cases; in achylia give ℳ 60 of dilute hydrochloric acid in a tumbler of diluted orange juice to be sipped with meals. (4) Diet does not depend on the character of the gastric secretion (§ 297. I and II). Small dry meals of simple but varied foods, avoiding condiments as a rule, are best. Give vitamin B as wheat germ or marmite. (5) Symptomatic treatment: For the pain, bismuth or magnesium carbonate, dilute hydrocyanic acid; for fermentation and acidity, sulphocarbolate of soda, kerol and alkalies two or three hours after a meal. Mucous vomiting is relieved by draughts of hot water, with alkalies, before breakfast. For flatulence, 20 grains of sodium bicarbonate in a cupful of hot water, with 20 drops of tincture of ginger, give great relief. Some find helpful pepsin, lactopeptine, takadiastase, or other artificial digestives. (6) General measures. Attention to the general health is necessary. A holiday from work, with regulated exercise and diet, and the treatment of sleeplessness, may be required at the beginning. Abdominal massage, electricity and exercises to improve muscle tone are important curative measures. Rest before and after meals is excellent in nervous cases. In acid gastritis, olive oil ℳ 60 before meals or atropine gr. 1/150 inhibits secretion.

IV. *The patient complains of* NAUSEA *and* ERUCTATIONS, *having no definite relation to the taking of food, and careful investigation reveals* NO STRUCTURAL DISORDER OF THE STOMACH. *The case is probably one of* NERVOUS DYSPEPSIA.

§ 286. **Nervous Dyspepsia** was formerly a frequent diagnosis, but modern investigations have shown that these symptoms are usually due to gastritis or to disease elsewhere in the abdomen. The constant discomfort and distress of gastric disorders bring about a neurasthenic and depressed state. On the other hand, flatulence is common in anxiety neurosis. Anxiety and worry cause spasm, and therefore delay. By means of the fractional test meal and X-ray examination it has been discovered that with strong emotional states digestion may stand still for the first hour or more; then if the cause be suddenly removed, digestion proceeds rapidly from that moment. But it is not wise to diagnose gastric neurosis until all clinical and special investigations have been carried out to exclude organic disease. Air swallowing and rumination are not gastric diseases, but bad habits, and are to be treated by explanation and psychotherapy.

V. *The patient complains of* INDIGESTION. *The* PAIN COMES ON DAILY, WITH CONSTANT RELATION TO FOOD. *It is* RELIEVED *by* LIQUID FOOD *and by* ALKALIES. *There is* TENDERNESS ON PRESSURE. *The disease is probably an* ULCER.

§ 287. **Simple** or **Peptic Ulcer** may be acute or chronic, and may be situated in the stomach, or the duodenum as far as the ampulla of Vater. The ulcers probably arise by peptic digestion of areas of mucous membrane which have been injured by toxins swallowed from the mouth or pharynx, or absorbed from septic foci elsewhere in the body. They tend

to heal readily unless there is gastric stasis and superacidity, when they become chronic, erode the wall of the viscus and may invade adjacent organs.

V*a*. *The patient complains of severe* PAIN, PRODUCED BY FOOD *and* RELIEVED BY VOMITING, *the vomit sometimes containing a quantity of blood.* The disease is ACUTE ULCER OF THE STOMACH.

§ 288. **Acute Peptic Ulcer** is less common than formerly. It occurs in the second and third decades of life. The ulcers are usually small and multiple. There are three very characteristic features, to which the symptoms of chronic dyspepsia may be added:

(1) *Pain* of an intense boring character usually limited to one spot, (2) *aggravated by food*, and accompanied by tenderness. A small, very tender area is sometimes present, and is characteristic. It is usually situated in the epigastrium. (3) The pain is *relieved by vomiting*, which comes on soon after food. The vomited matter contains an excess of hydrochloric acid. (4) Hæmatemesis, which may be profuse, may come on suddenly from time to time. (5) The appetite is usually normal or increased, but the patient avoids food because of the pain it produces. There is generally constipation and anæmia, and often a history of inadequate food and lack of fresh air. In some cases there may be no symptoms until profuse hæmorrhage or perforation suddenly occurs.

The *Diagnosis* is not difficult if pain, an area of tenderness, and hæmatemesis be present. The last, which was thought to be the most characteristic symptom, is now known to be very profuse in gastrostaxis (§ 272). Chronic appendicitis may simulate the disease. See Table XVII.

Treatment of Acute Ulcer.—In all but the mildest cases the patient must rest in bed. Treatment of hæmorrhage is given in hæmatemesis (§ 272). In cases of perforation immediate laparotomy is the best treatment. Where there is recent hæmorrhage or intractable vomiting, no food is allowed by the mouth, but ice may be sucked, and feeding is solely per rectum. Suitable diets are given in § 297. III. All foci of infection—teeth, tonsils, appendix, gall-bladder—must be removed later. Chronic inflammation of the appendix must be remembered.

V*b*. *The patient who is* MIDDLE-AGED *and is enfeebled by illness or anxiety, complains of* INDIGESTION *at* REGULAR INTERVALS AFTER FOOD, *which is* RELIEVED BY *taking* LIGHT FOOD AND BY VOMITING. *The disease is probably* CHRONIC GASTRIC ULCER.

§ 289. **Chronic Ulcer of the Stomach** is usually single. It occurs more frequently in men than in women. The patient is often thin and miserable and may complain of (1) "chronic dyspepsia." (2) Attacks of pain in the epigastrium, left upper abdomen or back, come on half an hour to two hours after a meal and pass off before the next meal. (3) The pain is often described as an aching or gnawing pain, and is located to a definite area of the upper abdomen. (4) The appetite is poor or may be restrained, the patient being afraid to take solid food and feeling better when resting and on light food. (5) Nausea and vomiting may occur, and the latter is

sometimes induced by the patient to obtain relief. (6) Antacid mixtures and powders, almost invariably relieve the pain. (7) Hæmatemesis is not common. Constipation is usual.

Physical signs.—Particularly during a bout of pain, deep pressure in the mid-line of the epigastrium usually indicates a definite local area of



FIG. 69.—GASTRIC ULCER ON LESSER CURVATURE.
Lower figure shows ulcer nearly healed after three weeks' treatment.

tenderness. In some cases, there is guarding of the left upper rectus abdominis, and deep tenderness may be elicited over the actual site of the ulcer.

Etiology.—The primary condition is probably one of gastritis; superimposed on this are other factors such as fatigue, overwork, worry, oral

sepsis, or irregular meals, which cause the inflamed gastric mucosa to ulcerate. Two groups of patients may be found: (1) Young or middle-aged women debilitated by overwork in poor surroundings, poor food or after infectious diseases. They are usually anæmic, thin and easily tired. The ulcer may be part of a general gastritis. (2) Elderly men who have pyorrhœa or have lost their teeth. In these cases, also, overwork or excess of tobacco smoking may contribute. In some instances the ulcer symptoms may be masked by tiredness and a sense of exhaustion.

The *diagnosis* is not difficult when the characteristic pain with epigastric tenderness is present, but pain after food, relieved by emptying the stomach, with occasional vomiting of blood, may occur in other diseases. The pain may be continuous if there are local complications such as adhesions to surrounding organs or chronic perforation. Every case of "chronic dyspepsia" should be investigated. The test-meal and efficient

TABLE XVII.

	CHRONIC GASTRIC ULCER.	MALIGNANT DISEASE.	CHRONIC GASTRITIS.
Pain . . .	1½–2 hours after food.	Constant discomfort.	Immediately after food.
Aggravated by	Large meals and condiments.	Meat.	Fried foods.
Appetite . .	Usually good.	Anorexia often marked.	Poor.
Duration of symptoms .	For months or years.	Recent, and not responding to treatment.	Often long-standing.
Vomiting .	Not frequent; relieves pain.	Often large quantity every few days.	Morning vomiting of mucus, especially with alcohol.
Hæmatemesis	Occasional but profuse; therefore bright red.	A continuous oozing; therefore "coffee-ground" in character.	Rare; and only streaks, unless in the venous congestion due to heart disease.
Tumour . .	None.	Present, though may not be palpable; secondary deposits may be recognisable in liver, peritoneum, glands, etc., later.	None.
Age . . .	Thirty to fifty.	Usually men over forty.	Any age.
Course . .	Recovery if well treated; with relaxation of régime, relapses occur.	Fatal in one or two years if not removed.	Liable to pass on to a chronic dyspepsia.

X-ray examination are the chief means of coming to a diagnosis. The stomach contents usually show increased free hydrochloric acid and total acidity, except where an atrophic gastritis is present. In the X-ray examination the crater of the ulcer projecting into the wall of the stomach may be filled by the barium emulsion and there is often spasm of the circular muscle which produces an incisura opposite the ulcer; at the filling and emptying stages of the meal there is a characteristic spider-form of the striæ of mucous membrane converging towards the ulcer. In the hands of experts the ulcer may be shown with the gastroscope (Table XVII, p. 345).

The *prognosis* is usually favourable if treatment is carried out early, and the general condition of the patient medically and socially attended to. If untreated, perforation into the peritoneal cavity or hæmorrhage may occur; in healing cicatrisation may lead to distortion or stricture of the stomach (hour-glass) or of the pylorus (stenosis). Death occasionally results from hæmorrhage and in a small proportion of cases of chronic ulcer of the stomach cancer may develop in the site of the ulcer.

Treatment is described in § 290.

Vc. *The patient is a healthy-looking*. ACTIVE MAN, *who* FOR YEARS HAS HAD ATTACKS OF ACIDITY *after overwork, worry or indigestible food: he* DEVELOPS PAIN 3–4 HOURS AFTER FOOD *or in the night:* RELIEVED *by* TAKING FOOD OR ANTACIDS. *The disease is* CHRONIC DUODENAL ULCER.

§ **290. Chronic Duodenal Ulcer** is eight times as frequent in males as in females. The *symptoms* often begin at the age of 20–35, and tend to come in attacks after dietetic errors, overwork, worry or exposure. (1) Epigastric pain, sometimes intense, and usually of nagging or gnawing type, comes on when the stomach is empty 3–4 hours after food—the so-called "hunger-pain," which frequently wakes the patient at about 2 a.m. and is almost immediately relieved by food. The pain tends to come at a regular time each day or during the night. (2) Vomiting is sometimes complained of, especially in a particularly severe bout of pain: it is due to pylorospasm, and after the stomach is emptied, relief is immediately obtained. (3) The pain may radiate to the back, and become more constant when the ulcer burrows into the head of the pancreas. (4) Sudden intestinal hæmorrhage may occur, evidenced by melæna, and sometimes preceded or accompanied by hæmatemesis.

Physical signs are always more marked during one of the recurrences of symptoms. There is often rigidity, or resistance to palpation, in the right upper quadrant of the abdomen. Deep tenderness is usually present in the mid-line just above the umbilicus, and/or locally over the site of the ulcer in the first part of the duodenum. *X-ray examination* reveals either pyloric spasm and delay, or the stomach contents rush through with rapid emptying of the stomach; a series of radiograms, rapidly taken, may reveal characteristic irregularity of the duodenal cap (Fig. 70). Hyperchlorhydria is usual.

Diagnosis.—In typical cases, the characteristic pain makes the

diagnosis easy. Chronic gastric ulcer, stone in the gall-bladder or kidney, and chronic appendicitis are to be differentiated. The diagnosis is confirmed best with the X-rays. A fractional test-meal and the discovery of occult blood in the fæces may help in obscure cases. Similar symptoms, but with left-sided pain, may accompany ulceration occurring after gastro-enterostomy (see V*d*).

Prognosis.—Medical treatment is usually successful, but it must be adequate. Insufficient treatment is the cause of non-success, as the ulcer readily heals superficially, but tends to relapse easily unless time is



FIG. 70.—A DEFORMED DUODENAL CAP WITH AN OPAQUE SPOT IN THE CENTRE.
The right-hand illustration shows the same duodenal cap (in the second left oblique view). The ulcer (see arrow) can be seen as a spur on the posterior surface, with opposing spasm.

given to allow healing to take place throughout. Perforation, hæmorrhage or recurrence after thorough medical treatment are indications for surgical interference. Operation should always be followed by careful medical treatment.

Treatment of Chronic Gastric and Duodenal Ulcers. The indications are to (1) rest the patient, (2) give a sufficiency of nourishing food which will call for little digestive effort, (3) reduce the superacidity of the gastric juice and (4) eliminate all sources of sepsis. (1) It should be stipulated that the patient be in bed 4–6 weeks, followed by slowly getting up and not returning to work for at least 3 months. (2) The food given will be

such as will neutralise the stomach acid, will excite little secretion and of a bulk which will not distend the organ. Diet in the initial stages consists chiefly of milk of which 3–4 pints are consumed in the 24 hours: it may be diluted, citrated or mixed with egg as unboiled custard: feeds should be given at least every 2 hours during the day, with 1–2 feeds during the night, and many prefer that the milk be sipped continuously all the waking hours, or be administered by a continuous nasal drip directly into the stomach (§ 231). In 1–2 weeks, depending on the cessation of symptoms, cereals, bread and butter, biscuits, rusks and butter, steamed white fish, tender or minced meat or chicken, well-boiled rice and milk puddings are gradually added; and at the end of 3–4 weeks a light invalid diet is reached (and see § 297. III). All mechanical and chemical irritants in the diet should be avoided: to prevent deficiency of vitamin C, small amounts of orange juice or grape-juice are necessary. From the first, olive or arachis oil 1–2 teaspoonsful are given several times a day: this may be flavoured with peppermint water. (3) The superacidity of the gastric juices and thirst are remedied by sips of alkaline water ($\frac{1}{2}$ teaspoonful of bicarbonate of soda to 1 pint of water). Antacids neutralise the gastric secretion: teaspoonful doses of a powder containing bismuth carbonate 1 part, sodium bicarbonate 2 parts, magnesium carbonate 1 part, calcium carbonate 1 part, can be given half an hour after a meal: the proportions of bismuth carbonate and magnesium carbonate are varied so that constipation is avoided. It must be remembered that if too much sod. bicarb. is used for too long a period, alkalæmia and even uræmia may occur. The triple phosphates of magnesium and of calcium, magnesium trisilicate and colloidal aluminium hydroxide are also powerful antacids. Olive oil, cream and tincture of belladonna ♏ 3–5 twice or thrice daily restrict the acid secretion. Alcohol and smoking are forbidden until the ulcer has healed, and even then only allowed in moderation. (4) All foci of infection, *e.g.*, septic teeth, infected tonsils, or chronic appendicitis, must be treated. Adequate mastication must be insisted on and artificial teeth fitted where necessary. The progress of healing may be determined by repeating the barium meal each 4–5 weeks, or by observation with the gastroscope.

Certain *complications* are liable to occur: any return of symptoms is an indication to resume a fluid diet. For pain or vomiting due to pylorospasm inj. atropine gr. $\frac{1}{150}$ and tinct. belladonnæ should be given: opiates are not advisable. Constipation is avoided by varying the composition of the antacid powder, by mist. magnes. hydrox., or by liquid paraffin or a plain paraffin emulsion. Anxiety and restlessness are treated by small doses of phenobarbitone, and sleeplessness by a moderately quick-acting barbiturate. Hæmorrhage requires opiates and complete rest; it is best to omit food by mouth for twenty-four hours, thirst being relieved by rectal fluids—water or hypotonic saline. After vomiting ceases diluted milk is given and the diet described above gradually adopted (§ 297 III). Perforation should be treated by omitting all food and medicine by

mouth, and usually by operation (§ 243), though occasionally perforation with adhesion to the posterior abdominal wall subsides without operation. Anæmia may require transfusion at the outset, followed by iron therapy.

Treatment should continue for 3 months on the strict lines laid down above, followed by a careful diet with regular 2-hourly, small, easily digested meals for 6 months or more (see post-ulcer diet, § 297. III). Part of this time should be spent on holiday before returning to work.

The indications for operation in peptic ulcer are (i.) perforation; (ii.) obstinate cases recurring after full medical treatment; (iii.) repeated hæmatemesis; and (iv.) pyloric obstruction and hour-glass stomach. Operation should always be followed by careful medical treatment and dieting for at least two years. Recurrence is by no means uncommon, even after excision of the ulcer or gastro-enterostomy has been performed: for this reason partial gastrectomy is often preferred.

§ 291. V*d*. **Gastro-jejunal ulcer** may occur at the site of gastro-enterostomy. The ulcer is situated adjacent to the anastomosis. The *symptoms* resemble those of duodenal ulcer, but the pain is on the left side and may be referred to the left flank. X-ray or gastroscopy confirms the presence of an ulcer. Perforation may occur into the peritoneal cavity, but more usually the ulcer spreads into the gastro-colic omentum and if neglected a gastro-colic fistula may result. Hæmorrhage may give rise to hæmatemesis and melæna. Rest and diet as for peptic ulcer will usually relieve, but surgery may be required in complicated cases, when the gastro-enterostomy may be undone and after resection the stomach and duodenum restored to continuity.

§ 292. V*e*. **Chronic Duodenal Ileus** occurs especially in visceroptotic patients who have lost weight or who lack abdominal muscle tone; it may follow a wasting illness, or a complicated child birth. *Symptoms* are in many ways similar to those of a duodenal ulcer (which may co-exist). Pain 3 to 4 hours after food, distension, nausea, and repeated vomiting occur. Anorexia, malaise, depression, migrainous headaches and other nervous symptoms may be present. *Diagnosis* is usually only possible by a barium meal, when the duodenum, lying to the right of the mid-line, is seen to be distended, to show to and fro peristalsis, and much delay in emptying. *Treatment.* A period of rest in bed (with the foot of the bed raised, or lying on the face) is necessary. Abdominal exercises, massage, and faradism to the abdominal muscles; small, frequent, nourishing meals, a supporting abdominal belt, and later, a change of environment help (and see § 251).

§ 293. VI. **Duodenal Diverticulum.**—Duodenal diverticula occur either as dilatations of the terminations of the pancreatic ducts in the wall of the duodenum, or pouches formed by the contractions of adhesions resulting from previous inflammation. (i.) The patient is usually past middle age; (ii.) he complains of symptoms resembling those of chronic duodenal ulcer, but without periods of well-being; (iii.) attacks of severe bursting pain in the right hypochondrium may occur as the result of inflammation of the pouch; (iv.) hæmorrhage with sometimes severe melæna may occur. The *diagnosis* is made by X-ray examination. *Treatment* consists in giving a bland diet containing no hard or indigestible pieces; liquid paraffin (♏ 60) two or three times a day, preferably before meals, to which bismuth oxychloride (gr. 15) may be added if there is pain. In severe cases surgery may be indicated. Good results have followed, but the operation is serious because the pouches are usually embedded in the head of the pancreas.

PAIN *is more or less* CONSTANT *with* DISTENSION *and* FLATULENCE; *the disease is probably* CANCER *or* DILATATION *of the* STOMACH.

VII. *The patient, who is in middle or advanced life, presents more*

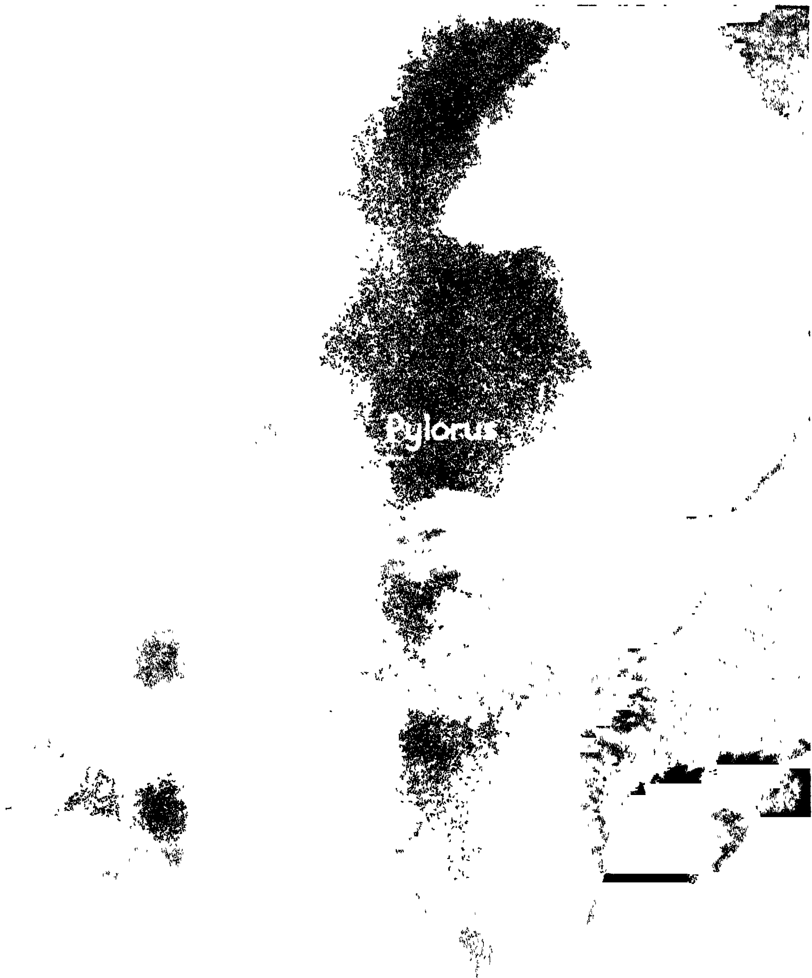


FIG. 71.—DUODENAL DIVERTICULUM; large mushroom-shaped diverticulum in the third part of duodenum.

CACHEXIA *than could be accounted for by dyspepsia, and vomits from time to time* "COFFEE-GROUND" MATERIAL. There is probable MALIGNANT DISEASE OF THE STOMACH. Gastric symptoms beginning in a patient of middle age or over should always be regarded as suspicious of cancer.

§ 294. Cancer of the Stomach (Synonym: Carcinoma ventriculi).—The clinical history rarely extends beyond one or two years. The *Early*

Symptoms depend largely on the situation of the disease. (i.) Loss of appetite, especially for meat and bulky foods, is usual. (ii.) A sense of epigastric discomfort, flatulence and fullness occur during the meal, or immediately afterwards, and may be associated with belching foul-smelling gas. (iii.) The pain is situated in the epigastrium or back, radiates in different directions, and is usually accompanied by tenderness but no rigidity. It is increased rather than diminished by taking food, and is sometimes continuous and independent of meals. (iv.) Vomiting may occur early or late, and usually indicates obstruction in some part of the stomach. Generally it takes place some time after the ingestion of food, the interval depending on the position of the lesion : thus, if at the cardiac end, the interval is short : if at the pylorus it may be hours after taking food. Sometimes the vomiting occurs every two or three days. An examination of the vomited matter often shows diminution or absence of hydrochloric acid and the presence of lactic acid. (v.) Some degree of anæmia is present in practically all cases by the time advice is sought. Sometimes this may be marked, and occasionally of megalocytic type as in pernicious anæmia. (vi.) Loss of general strength and energy, and an insidious loss of weight, are present at an early stage in most cases. Less frequent early symptoms include : (vii.) Brisk hæmatemesis or melæna occurs in less than 5 per cent. of cases. (viii.) Sudden perforation is unusual. (ix.) Persistent diarrhœa associated with " a leather-bottle stomach " is sometimes met. (x.) Acanthosis nigricans, unexplained phlebitis, poly-



FIG. 72.—CANCER OF STOMACH, CARDIAC END; note, filled stomach does not expand.

neuritis and even Korsakoff's syndrome may be the first expression of the disease. *Late Symptoms:* (xi.) Cachexia becomes marked, anæmia prominent, and the sallowness of the skin may suggest pernicious anæmia or even jaundice. (xii.) The appetite becomes non-existent, and the profound anorexia and wasting seriously disturbing. (xiii.) Vomiting occurs as a fairly constant sign: even the body of the stomach may be sufficiently obstructed to make the reception and passage of food difficult. (xiv.) A tumour is present sooner or later in two-thirds of the cases.

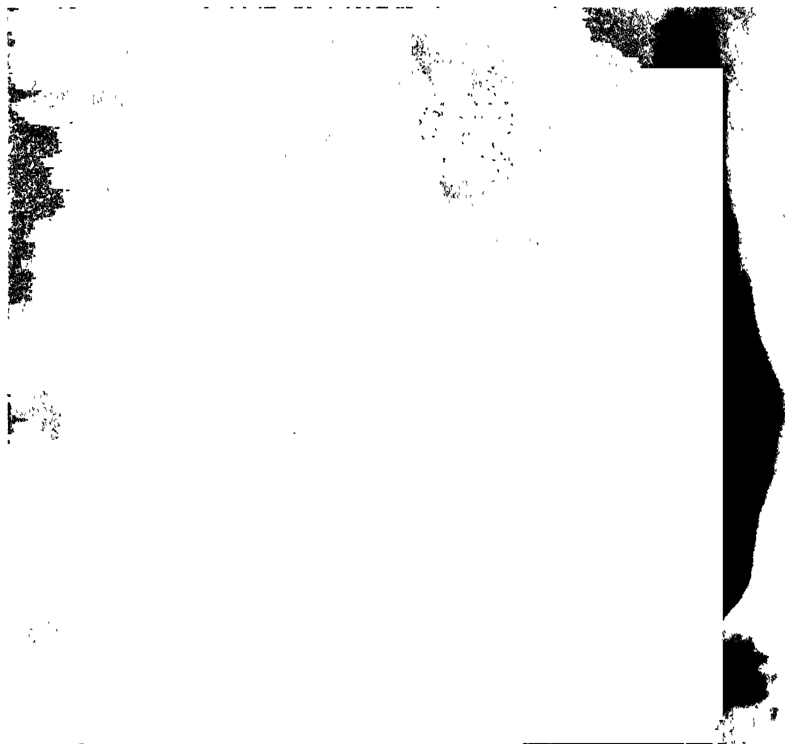


FIG. 73.—CANCER OF PYLORUS, filling defect (lower arrow).

Transmitted aortic pulsation, and slight fullness or rigidity of the upper end of the right rectus, may be present without a palpable tumour. When the cancer is deposited in the pylorus, it may cause adhesions which prevent the tumour from coming forward. The great majority of gastric tumours come forward to the left of the middle line. It is usually stated that whereas hepatic tumours move, gastric tumours usually do not move with respiration: but this feature has many exceptions. One of greater importance is the alternate appearance and disappearance of the tumour. At first it is extremely mobile, but later it becomes fixed by adhesions:

this is also the reason why perforation is rare. (xv.) Metastases occur in the pelvi-rectal pouch, in the ovary as a Krukenberg tumour, in the liver, or in the glands above the clavicles. In many cases, however, there are no symptoms referable to the stomach, and the diagnosis is only made at autopsy.

Etiology.—(1) Cancer of the stomach is more common in men. (2) It is rarely met with under forty, although cases occur under thirty. (3) Simple ulcer and chronic gastritis appear to predispose.

Diagnosis.—The disease should always be suspected when gastric symptoms first occur after the age of 40 years: and especially when there is only partial relief of the symptoms and the ulcer fails to heal on a strict ulcer régime. Radiology is essential if an early diagnosis is to be made. There is a characteristic irregular outline of the stomach wall, with rigidity, and defective or absent peristalsis. With cancer of the body, there is a tube-like stomach with food rushing through; in pyloric cancer there is obstruction and dilatation. Simple ulcers of the stomach are usually adjacent to the middle of the lesser curvature: many ulcers in the prepyloric area and all ulcers in the fundus and adjacent to the greater curvature are malignant. A fractional test meal may give a normal acid curve: often free HCl is absent, the total acidity is normal or low, and blood is present: organic acids (such as lactic acid) and also sarcinæ are present when there is gastric dilatation. The fæces show occult blood persisting even when the patient is on a strict ulcer régime. If emaciation be rapid, and gastric symptoms resist treatment, cancer should be strongly suspected. Gastroscopy is often helpful. *Dyspepsia* and *chronic gastritis* have pain directly related to food: for these, and *Simple ulcer* of the stomach, see Table XVII, p. 345. For Simple pyloric stricture, see Dilatation. *Tumour of the pylorus* or stomach has to be diagnosed from tumour in the neighbouring regions (§ 263). Thus a growth on the back of the stomach may resemble a kidney tumour. *Addison's disease* and other cachectic conditions must be excluded (Chapter XVI). *Pernicious anæmia* is sometimes strongly suggested by the colour of the patient, but in this disease there is not a corresponding amount of emaciation, and the blood-picture is different.

The *Prognosis* is very grave. The duration is rarely longer than six to eighteen months after the first definite symptoms appear. Death is the invariable result unless surgical measures are adopted early. The symptoms upon which one relies most in the diagnosis in these cases, anorexia and emaciation, appear to be those which also best measure the longevity of the patient. Death generally takes place by inanition, but almost as often it occurs suddenly by the involvement of important structures, and it would be unwise to assume that because the patient does not waste he will not die soon. Partial gastrectomy is successful if undertaken early, but of those diagnosed by X-ray and judged fit for surgery, only about half are capable of removal. A clinical diagnosis without X-rays and test-meal is rarely made early enough; the chance for successful treatment lies in the early investigation of cases of dyspepsia.

Treatment.—Early surgical treatment affords most hope of success. Apart from this the indications are to support the strength and relieve the symptoms. The former may be accomplished by easily digestible or predigested food (§ 297. XI), and by the use of pepsin and hydrochloric acid. For the latter consult § 295, Dilatation. For the flatulence and pain, give creosote and opium, or morphia hypodermically.

VIII. *The patient presents symptoms of* CHRONIC INDIGESTION, *and on physical examination there is* SPLASHING, *or the* AREA OF THE GASTRIC RESONANCE *is increased, or there are* FOOD RESIDUES *before breakfast.* The disease is probably GASTRIC DILATATION or ATONY.

§ **295. Gastric Dilatation** may be due to two main causes, (*a*) Motor Insufficiency or Hypotonia; (*b*) Pyloric Obstruction.

(*a*) GASTRIC HYPOTONIA occurs independently of pyloric obstruction. There is delay in emptying the stomach, often associated with gastroptosis. The condition is most common in women of poor physique, between the ages of 30 and 50.

Symptoms.—(1) The patient is of the asthenic type with general loss of muscle tone, and often backache. There is vague dyspepsia, or discomfort in the upper or mid-abdomen, which is usually worse after meals. (2) Flatulence and aerophagy often accompany this dyspepsia. (3) True pain is absent. (4) Prolonged lassitude follows a moderate-sized meal. (5) A disinclination for food is often associated with some loss of weight and constipation. (6) Depression and anxiety are often superadded.

Physical signs.—(1) The tone of the abdominal wall is poor. (2) Gastric splashing or succussion can be demonstrated several hours after a meal (§ 276). (3) Food residues can be detected six or more hours after a previous meal, and in extreme cases a fractional test meal will show food residues in the fasting stomach juice next morning. (4) A barium meal examination affords a ready method of detecting the degree of gastric atony, and will measure the amount of gastric delay. (5) A fractional test meal usually shows a low acid content in the gastric juice, and delayed emptying.

Etiology.—(1) Acute infectious diseases such as typhoid, influenza and pneumonia contribute by virtue of their toxins. (2) Chronic states of general debility such as are associated with anæmia, tuberculosis and neurasthenia predispose. (3) An ill-balanced diet, with irregular meals and especially lack of vitamin B leads to defective muscle tone. (4) Depression and chronic anxiety states are potent predisposing factors. (5) There is a definite correlation between the motor activity of the stomach and the athletic capacity of the individual—lack of regular walking exercise is undoubtedly detrimental.

Diagnosis.—It is important to exclude organic disease in the body as a whole and in the stomach by a thorough physical examination and by a barium meal X-ray of the stomach. This will set a firm foundation for treatment, and will be especially reassuring to the patient.

Prognosis.—The disease is always troublesome and liable to relapse.

If diagnosed and treated early and thoroughly, and if contributing factors such as anæmia can be remedied, a cure is possible.

Treatment.—The indications are: (1) To keep the stomach as empty as possible. This may be done by diets No. I or VI, § 297. (2) Give concentrated or predigested foods with very little fluid. Little carbohydrate, not at the same meal as animal food, is preferable. (§ 297. Salisbury diet VI.) Give vitamin B in the form of wheat germ, a teaspoonful twice or thrice daily. (3) Promote digestion: vide § 284. (4) Adequate rest and sleep are essential: lying on the right side for ½–1 hour after the bigger meals promotes emptying of the stomach. (5) A lower abdominal support may aid. (6) Regular exercise, and local abdominal exercises, massage and faradism help to improve the hypotonia.

(*b*) In PYLORIC OBSTRUCTION there is difficulty in emptying the stomach due to organic obstruction in the prepyloric canal or at the pyloric sphincter. Radiology has demonstrated two distinct types: in the first, due to temporary or minor pyloric obstruction, the vigorous peristalsis of the stomach keeps the stomach largely empty, and of small size. In the second type, with more marked obstruction, peristalsis is largely absent and the stomach remains as a dilated sac containing many pints of fluid and food residues.

Symptoms.—Early Cases. (1) Gastric pain is present, due to exaggerated gastric peristalsis. It is often of colicky type (vide pyloric spasm, § 246). (2) Vomiting of relatively small amounts produces a vomitus containing partially digested food, with an acid reaction, but no bile. (3) Loss of weight and constipation largely depend on the amount of vomiting. (4) There are symptoms of the cause of the obstruction, *e.g.*, duodenal ulcer, pyloric carcinoma. Physical examination may reveal (5) fullness in the left upper abdomen, (6) visible peristalsis (passing from left to right) in the epigastric region, which is more obvious after a meal and may be started by palpating or sharply flicking the abdominal wall. (7) A pyloric tumour may be felt. *Later cases.* (1) There is vomiting, particularly towards the end of the day, or at intervals of two or three days, of acid, sour-smelling, frothy material, on which a scum forms on standing. The quantity vomited may amount to several pints. (2) Decomposition and fermentation in the stomach give autotoxic symptoms and the symptoms of chronic gastritis: (3) loss of weight and constipation are marked: (4) dehydration may ensue. (5) In severe cases, tetany and alkalosis are sequelæ. Physical examination reveals, in addition to the wasting, (6) a dry tongue and poor volume pulse: (7) the distended atonic stomach may form a prominence in the left upper abdomen: (8) through a stomach tube, a large residue may be withdrawn.

Diagnosis.—To diagnose the cause and extent of the obstruction, X-ray examination is essential. If there is much gastric retention, the stomach may have to be emptied by a stomach tube, to prevent the barium emulsion being unduly diluted, and so obscuring the examination.

Etiology.—The causes are (1) spasm of the pyloric sphincter secondary

to a duodenal ulcer, and rarely as a reflex phenomenon due to other causes, *e.g.*, chronic appendicitis : (2) pyloric stenosis may occur from cicatrisation of a simple ulcer of the duodenal or prepyloric areas. (3) Obstruction due to a scirrhous cancer (§ 294). (4) Pressure from without, *e.g.*, enlarged glands in the portal fissure, or due to a band of adhesions, is rare. (5) For congenital hypertrophic pyloric stenosis, see § 271.

Treatment.—(1) The main indication is to treat the various causes of the condition. (2) Gastric lavage may be necessary as an adjunct to other treatment, or before operation : it may be carried out once or twice daily, and particularly in the evening. Normal saline, or water is best : add bicarbonate of soda (gr. 60–℥i) or hydrogen peroxide (♏ 30–℥i) to dissolve any mucus present. (3) To prevent fermentation, the symptoms of which are very troublesome, carbolic acid (1 to 3 minims), thymol (5 grains), or sodium sulphocarbolate (20 grains), are given preferably in a tumbler of water between meals. After lavage, calomel ($\frac{1}{8}$ grain t.i.d.) may be given with advantage. (4) Prevent dehydration by the administration of fluids rectally, subcutaneously or intravenously. (5) Inj. atropinæ gr. $\frac{1}{100}$, or tinct. belladonnæ, ♏ 10, 4–5 times a day, is most helpful in overcoming pyloric spasm. (6) Surgical treatment is essential in all cases of pyloric stenosis, of pyloric neoplasm producing obstruction, and in cases of pyloro-spasm not responding to medical measures.

§ **296. Gastroptosis** is a condition in which the stomach has dropped from its position. The symptoms and signs are apt to be confused with Gastric Dilatation, with which it may be associated. The condition is often part of a general visceroptosis (§ 251), and is most clearly demonstrated by a barium meal. Intestinal stasis is usually also present, and hence an aggravated state of neurasthenia is frequently associated with the condition. *Treatment* is on the lines of visceroptosis (q.v.).

Dietaries and Invalid Foods [1]

§ **297.** Less food is required in old age than in youth, and with a sedentary life than with an active or outdoor one. For a person in health three meals a day are usually sufficient ; but when a man is unable, from illness, to take more than a very small quantity at a time, he may require to take it more often. Dietetic errors are a fruitful source of dyspepsia and gastritis. Too frequent meals, habitual over-feeding, bolting the food, and irregularity of the meals will in time derange any stomach. A diet lacking or deficient in vitamins (especially vitamin B) leads to atony of the muscles of the digestive canal. Vitamin B is supplied by wheat germ, yeast (marmite) and National bread. Deficiency of food, and long restriction to the same kind of food, induce dyspepsia by affording no stimulus to excite the secretions ; and in this connection it is well to remember that a frequent cause of failure on the part of the physician to cure dyspepsia is his disregard of this latter fact. In anæmic cases, starchy foods, especially potatoes and new bread, do not afford sufficient stimulus for the gastric functions ; proteins such as tender and underdone meat are more readily

[1] These diets are not designed to conform with food rationing.

digested. It is often a good rule to begin treatment by cutting down the amount rather than by entirely prohibiting the use of certain articles of diet. The frequent use of condiments, spices, strong tea, and of alcohol especially, leads to chronic gastritis; while dyspepsia is induced by imperfect mastication, bolting of meals, too much fluid with meals, hard mental or physical work immediately after eating, too cold or too hot food, or food which is badly prepared. Excess of tobacco-smoking and constipation are certainly causes of dyspepsia. Greasy and fried foods are bad in dyspepsia, because the gastric juice cannot penetrate the coating of fat. "Well-made" pastry and other so-called rich carbohydrate foods are a source of dyspepsia, especially when eaten at the same meal as protein.

Without appropriate dietetic rules our best efforts may fail, especially in the treatment of gastro-intestinal disorders, and other diseases which depend on the proper elaboration and assimilation of food. A few specimen dietaries are therefore given.

I. The following table is given as a guide to aid in the drawing up of a diet for mild cases of **dyspepsia** or **chronic gastritis**: *Breakfast.*—Boiled sole, whiting, or flounder; or a slice of crisp fried bacon or a soft-boiled egg; a slice of dry toast or of bread (not new) and butter. *Beverage.*—One cup of cocoa or of milk and water, sipped after eating. *Luncheon.*—Chicken or game, with bread, and a little tender, well-boiled vegetable, such as spinach, vegetable marrow, or young French beans. For sweets and dessert, a plain biscuit or milk pudding. *Beverage.*—Half a tumbler of water sipped *after* eating. *Afternoon Tea.*—A cup of marmite, bouillon, or of weak tea with milk, and a slice of wholemeal bread and butter. *Dinner* (two courses only).—Vegetable soup, fish of the kinds allowed for breakfast, without potatoes. Or a slice of any tender meat, such as saddle or loin of mutton, or the thick part of an underdone chop with crumbled stale bread; custard, junket or jelly, or a little well-stewed fruit. *Beverage.*—Half a tumbler of water, with from one to two tablespoonfuls of spirit if desired.

Condiments and stimulants are good in some forms of dyspepsia, but must be avoided in chronic gastritis, as tending to irritate the mucous membrane. The patient should abstain from salted and cured meats, tinned foods, sweets, pastry, raw vegetables, cheese (except cream cheese), fried foods, strong tea and coffee.

II. In **Superacidity** fried foods, game, vinegar, jam, condiments and alcohol should be avoided. The food should consist of soft, well cooked, finely cut up or minced, fish, meat, or poultry, eggs and cream cheese; fats, olive oil if it can be taken, plain butter not made into sauces, ice cream; mashed potato, rice, macaroni; weak tea—not coffee—or plain water, preferably after meals. Weak meat soups may be allowed. Milk is often difficult. If there is delay in the stomach, meals should be taken dry. Sometimes it is necessary to give food more frequently than three times a day. The patient should not sit at an ordinary table with others eating appetising food. *Early Morning.*—Weak tea without sugar, or tumbler of hot water. *Breakfast.*—Eggs (boiled, poached, fried or scrambled), cold ham or bacon, crisp toast, plenty of unsalted butter and a cup of weak tea after the food is taken. *Lunch.*—Fish or well-cooked meat which may be minced, little mashed potato, rice or macaroni, suet or baked castle or stiff milk pudding, toast, butter and cream, tumbler of water after food. *Tea.*—Cup of weak tea with milk and no sugar, little bread and butter or rusk and butter. *Dinner* as lunch, with addition of fruit juice or jelly. Half to one ounce of olive oil half an hour before lunch and dinner, if it can be taken.

III. **Peptic ulcer.** SIPPY DIET (modified). *First week*, or until the patient has been free of symptoms for at least 3 days: 3 oz. milk, citrated milk, milk and cream, or with a raw egg stirred in, each 2 hours while awake and an extra feed may be given at night. *Second week.*—Breakfast: 1 egg, 1 oz. toast, ½ oz. butter, 4 oz. milk. Mid-

morning: glass of milk and rusk. Noon dinner: 2 oz. minced or pounded beef or chicken, 1 oz. toast, ½ oz. butter. 2 p.m.: 1 egg in 3 oz. milk. 4 p.m. tea: 1 oz. thin bread and butter, without crusts, 3 oz. milk. 7 p.m. supper: 1 oz. well-boiled rice or macaroni, ½ oz. butter, 1 oz. toast, 3 oz. milk. 9 p.m.: 6 oz. of Benger's, Allenbury's, etc. A tablespoonful of olive oil at 11.30 a.m. and 6.30 p.m. *Third to fifth weeks.*—Breakfast: egg (poached, boiled or scrambled), or steamed or boiled fish, toast, butter, milk. Midmorning: milk and biscuit. Noon: chicken or mince, mashed potato, dry toast, butter, boiled milk pudding. 2 p.m.: milk or egg and milk. Tea: toast or bread and butter, sponge cake, milk. 7 p.m.: steamed fish, toast and butter, macaroni or vermicelli, milk pudding, junket or custard. Bedtime: milk, Benger's, etc. Then a *post-ulcer diet.*—Breakfast: porridge, fish, cold fat ham, egg, toast, butter, cream, flavoured milk or weak tea. Dinner: chicken, tender mutton, lamb or beef, potato, sieved green or mashed root vegetables, milk or light steamed pudding, cream, cream cheese. Tea: bread and butter, sponge cake, milk and water or weak tea. Supper: fish, chicken or sweetbread, potato, sieved green vegetables, macaroni, milk pudding or milk shape, toast, butter, cream. In these diets with fresh milk, there is plenty of vitamin C; to avoid deficiency a little diluted orange juice may be given each day. On the whole, fruit is better avoided for several weeks, and should be as a purée, or as fruit juice alone, or in jelly.

HURST DIET. Mixture A. = Atropine Sulph. gr. 1/200: Aq. ad 60 min. Mist. Magnes. Hydrox. 60 min. may be added to milk feeds if needed as an aperient. *Strict diet.*—7.45 a.m.: Mixt. A. 60 min. 8 a.m.: 5 oz. milk, warm or cold, flavoured with tea and sugar to taste. Add Sod. citrate gr. 15. 9 a.m.: 5 oz. feed of arrowroot, cream of wheat, Benger's, junket, custard; to any of these can add red currant, apple or other fruit jelly; junket may be flavoured with chocolate. Salt may be added. Also 1 rusk and butter. 9.30 a.m.: olive oil ½ oz. 10 a.m.: as at 8 a.m. 11 a.m.: as at 9 a.m. + 1 oz. cream. 12 noon: as at 8 a.m. 1 p.m.: 5 oz. thick soup or semi-solid purée of potato, artichoke, cauliflower or parsnip + 1 oz. cream. Rusk and butter. 2 p.m.: as at 8 a.m. 2.45 p.m.: Mixt. A. 60 min. 3 p.m.: as at 9 a.m. 3.15 p.m.: Magnes. or calcium tribasic phosphate 60 gr. in little water. 4 p.m.: as at 8 a.m. 4.30 p.m.: olive oil ½ oz. 5 p.m.: as at 9 a.m. + 1 oz. cream. 6 p.m.: as at 8 a.m. 7 p.m.: as at 1 p.m. 7.15 p.m.: Magnes. or calcium tribasic phosphate 60 gr. in little water. 8 p.m.: as at 8 a.m. 9 p.m.: as at 9 a.m. 9.45 p.m.: Mixt. A. 120 min. 10 p.m.: as at 8 a.m. 10.15 p.m.: Magnes. or calcium tribasic phosphate 60 gr. in little water. Small quantities of water or sweetened orange juice and water may be drunk between meals. A feed of citrate and milk must be taken at night if in pain, and an extra dose of powder at any time if indigestion occurs. *Intermediate Ulcer diet* (for 2 or more weeks between strict diet and post-ulcer diet).—Every hour from 8 a.m. to 10 p.m. (except at 9 a.m. and 1, 5 and 7 p.m.): 4 oz. of a mixture of milk 2 pints, cream 5 oz., Sod. citrate gr. 120. At 9 a.m. and 5 p.m.: olive oil ½ oz. and Mixt. A. 60 min.; weak milky tea; 1–2 lightly boiled, poached or scrambled eggs, thin bread and butter or toast. 1 p.m. and 7 p.m.: olive oil ½ fl. oz. and Mixt. A. 60 min. Fish or chicken, mashed potato and vegetable purée, custard, junket, etc.

MEULENGRACHT DIET. Two-hourly feeds with four light meals a day. *Early morning*—milk or milky tea. *Breakfast*—strained oatmeal, porridge and milk, eggs, rusks, biscuits or bread (toasted), butter. *Mid-morning*—milk with rusk or biscuit, or milk soup with vegetable stock. *Mid-day*—minced chicken, rabbit, beef, mutton, pounded fish, or soufflé, brains, tripe or sweetbread; mashed potato, sieved spinach or greens or mashed carrot, parsnip or turnip; junket or milk pudding. *Tea*—milky tea, bread or biscuits, butter, jelly or honey. *Supper*—as at mid-day. *Bedtime*—milk with biscuit, ovaltine, Benger's or other invalid food. Milk in the night if awake.

IV. **Diet for Cholecystitis** and protective diet in convalescence from infective hepatitis. Plenty of protein with adequate carbohydrate is needed; fats should be restricted, only butter and milk fats being allowed. *Breakfast*—well-cooked porridge

4 oz., one egg with grilled bacon ½ oz., or fish 4 oz. (except herring or mackerel); bread or toast 1 oz., butter ½ oz., tea or coffee, skimmed milk [1] 6 oz.: 11 *a.m.*—skimmed milk 8 oz.: 1 *p.m.*—lean meat 4–6 oz., potato 1½ oz., green vegetables, salads or roots to suit taste, cheddar cheese 1 oz., fruit 4–6 oz.: 4 *p.m.*—bread and butter 1½ oz., plain cake 2 oz., tea, skimmed milk 6 oz.: 7 *p.m.*—clear vegetable soup, fish 4 oz., meat 6 oz., vegetables as at lunch, cheddar cheese 1 oz., fruit. *Bedtime*—skimmed milk 8 oz. Calories 2,200–2,500; protein 230–280 G; carbohydrate 200–250 G.; fat 80–90 G.

V. **Constipation.**—The first thing in the morning drink a tumbler of plain water, hot or cold, or eat an apple, pear, bunch of grapes, banana, orange, etc. *Breakfast.*—Coffee, not too strong, with a little milk; stone-ground flour or wholemeal bread with plenty of butter, honey or treacle; or well-cooked oatmeal, Kellogg's All-Bran or wheat germ, with cream or treacle. *Lunch.*—Sardines or olives in oil; fish, chicken or roast meat; vegetables, greens and salad; cream cheese, wholemeal bread and butter; fresh or stewed fruit with cream. *Tea.*—Coffee and milk, wholemeal bread, butter. *Dinner.*—Vegetable soup, fish or egg dish, vegetables and salads; suet pudding; fruit, wholemeal bread and butter. Lactose may be used instead of ordinary sugar. Cultures of *B. acidophilus* in milk can be taken once or twice a day. Fluids may be taken freely with meals, and half to one ounce of liquid paraffin or emulsion night and morning for a limited time. Some do better with bassorin or agar-agar preparations.

VI. The "**Salisbury**" **diet** consists of nitrogenous food only, the meals being taken almost without fluid, but a quantity of hot water being taken between meals. The solid food administered is in a highly concentrated form. Farinaceous and bulky substances are eliminated. By reason of the dryness and small bulk of the food, a dilated or atonic stomach is enabled to resume its normal dimensions. One pound (1 lb.) of lean butcher's meat, chopped or scraped very fine, so as to rid it of its white fibrous tissue, and lightly cooked, is taken per diem, divided into four or more meals. Occasionally a little well-toasted or twice baked bread is allowed also. For a change, ½ pound of fish may be substituted for an equal quantity of meat. The meals are taken quite dry, or 2 ounces of fluid only; but two hours later ½ pint of hot water is sipped.

VII. **Diet for Obesity** (§ 18).—The diet should be made up as far as possible from: Fish, eggs and meat (except as stated below); green vegetables, parsnips, carrots, turnips, celery, mushrooms, cauliflower, onions, tomatoes, beetroot or salad; stewed and fresh fruits. Bread, toast, rusks, water biscuits, butter, cream and cereals should be taken in small quantities only.

The following foods should be *avoided*: sugar, puddings, creams, cakes, sweet biscuits, shortbread, nuts and alcohol.

Take sparingly: salmon, herrings, pork, duck, bacon, savouries, rich sauces, potatoes, cheese, and made-up dishes such as pies, fricassées, rissoles, sausages and macaroni cheese, which are concentrated foods. So far as possible no fluid should be drunk during meals until all the solid food has been eaten.

The following is an example of a day's diet (weight in ounces). *Breakfast.*—Fish (2½ ounces), bacon (1) or cold ham (1), with 1 egg, or 2 eggs only; bread (1); butter (½); sugarless marmalade (½); milk (2); tea or coffee. *Lunch.*—Fish (4); or lean meat (2½); 2 vegetables (2½ of each), or salad (4); stewed fruit (3), or fresh raspberries, strawberries or gooseberries (6), plums, peaches, grapefruit or orange (5), cherries, apples, pears or grapes (4), banana or figs (2). *Tea.*—Bread or water biscuits (½); butter (½); sugarless jam (½); milk (1½); tea. *Dinner.*—Clear soup or soup made with vegetables only; fish (1½); meat, vegetables and fruit, as at lunch. Bovril or Marmite *at bedtime.* Caloric value = 1,250 (approx.).

VIII. **Diet in Nephritis.** (A) **Acute.**—A preliminary period of almost complete renal rest must be given, especially when anorexia or nausea is present. *Diet:*

[1] Can be made from Household Dried Milk.

Stage I. 1½ pints of fluid a day; water, barley water, Imperial drink, orangeade or lemonade with glucose added in the proportion of 4–6 oz. per pint, grape-juice or tomato-juice, and toffee if acceptable. This is continued until the hæmaturia has diminished, the blood pressure has fallen and the "critical diuresis" set in—usually a matter of three to four days; after this time the diet may be increased to *Stage II.* 2–2½ pints of fluid a day—milk ½ pint, weak tea, barley water, glucose-orangeade or lemonade, Imperial drink, 1 orange, 1 small tomato, grapes, stewed fruit, 2 oz. of wholemeal bread with *fresh* butter, 2 oz. of potatoes, porridge, milk puddings, honey, marmalade, jam, and ½ oz. of cream. (Fruit and vegetables to count as an equivalent weight of water.) After ten to fourteen days it is usually possible to increase further to *Stage III.* 3 pints of fluid a day—milk 1 pint, weak tea, barley water, orangeade or lemonade, Imperial drink, oranges, grapes, tomatoes, *ad lib.*, stewed fruit, milk puddings, cereal foods such as grapenuts, porridge, bread, cakes, fresh butter, cream, honey, jam, marmalade, steamed fish, potatoes, greens and salads. *Foods to be entirely forbidden:* Soups, bovril, beef-tea, liver, brains, sweetbread, alcohol, acid foods such as vinegar, and spices. *Foods to be avoided until œdema has disappeared:* Salt (in cooked food and at table). *To be avoided until blood pressure lowered:* Meat, bacon, ham and poultry.

(B) **Subacute Nephritis** *with Œdema.*—The total fluid intake, including fruit and vegetables, should not exceed the daily output of urine, but cannot be reduced below 35 oz. a day. Adexolin, 2 capsules, should be taken daily. Salt-free bread or same toasted 6 oz.; white fish, preferably fresh-water trout 6 oz.; mutton, veal, lamb, lean ham or chicken 10 oz.; skimmed milk flavoured with weak tea or coffee (1 pint a day); sugar, honey, jam, marmalade *ad lib.*, rice or other cereal 1 oz., made into milk pudding; flaked cereals *ad lib.*, grapes, tomatoes, apples, oranges, stewed fruit 10 oz.; salt-free butter ½ oz. a day. *Approximate Values*—Protein content, 150 G.; fat, 45 G.; carbohydrate, 300 G.; NaCl., 2·5–3·0 G.; calories, 2,100.

Karell Diet.—*Days* 1–7.—Milk, 7 oz., at 8 a.m., 12 noon, 4 p.m. and 8 p.m. Total salt is 1·3 G. *Day* 8.—Add at 10 a.m. a softly-cooked egg and one slice of toast. Total salt is 1·78 G. *Day* 9.—Add 2 oz. vegetables such as asparagus, celery, cauliflower or carrot, and two teaspoonfuls of cornflour to the milk taken at noon, to form milk soup. Add one slice of toast at 4.30 p.m. Total salt is 1·89 G. *Days* 10–12.—Add one egg, 1 oz. rice (weighed raw) and 2 oz. vegetables. Total salt is 2·41 G.

(C) **Chronic Nephritis** *with Blood Urea over* 100 *milligrammes per cent.*—Total fluid intake, 2½–3 pints per day: Weak tea, barley water, lemonade, orangeade, Imperial drink. Also glucose in the proportions of 4–6 oz. to the pint, with grape juice and tomato juice; cream, ½–1 oz. daily. After three or four days of such a régime, usually the blood urea has fallen to lower limits and then a higher protein diet should be given and a greater fluid intake should be possible.

Chronic Nephritis *with Blood Urea over* 80 *milligrammes per cent.*—Total fluid intake, 2½–3 pints of fluid daily, consisting of weak tea or coffee, milk (½ pint), lemonade, orangeade; bread or biscuits (5 oz.), butter (2 oz.), porridge or cereal foods such as flaked wheat or flaked rice (2 oz.), milk puddings, cream (1 oz.), cake; jam, honey or marmalade (½ oz.), sugar (2 oz.), green vegetables and salads *ad lib.*; potatoes (5 oz.), fish, meat or chicken (2 oz.), or one egg. Approximate content: Protein, 37 G.; fat, 69 G.; carbohydrate, 212 G.; calories, 1,600. Unrestricted Foods: Green vegetables, salads, fruits, sugar, honey, jam, arrowroot, butter.

Chronic Nephritis *with Blood Urea between* 40 *and* 80 *milligrammes per cent.*—Total fluid intake, 3–3½ pints a day (*i.e.*, average normal amount). Bread or cake or biscuits (10 oz.); milk (½ pint); white fish (4 oz.) or one egg; meat or chicken (3 oz.). Unrestricted Foods: Green vegetables, potatoes, salads, fruit, sugar, honey, jam, marmalade, oatmeal, cereal foods such as flaked wheat or flaked rice or other cereal preparations, milk puddings, butter, cream. Approximate protein value = 72 G.; approximate caloric value, 2,200. *Foods to be avoided:* Condiments, spices, meat extracts, brains, liver, sweetbreads.

IX. Diabetic Diets.[1]—

	Diet A.	Diet B.	Diet C.	Diet D.
Breakfast:				
Tea or coffee . .	*Ad lib.*	*Ad lib.*	*Ad lib.*	*Ad lib.*
Milk . . .	2½ oz.	2½ oz.	2½ oz.	2½ oz.
Herring. . .	1½ oz.	Nil	Nil	Nil
Bacon . . .	Nil	½ oz.	1½ oz.	1½ oz.
Eggs . . .	2	2	2	2
Tomatoes . .	7 oz.	7 oz.	7 oz.	7 oz.
Bread . . .	1½ oz.	1½ oz.	1½ oz.	2 oz.
Orange . . .	Nil	2½ oz.	2½ oz.	2½ oz.
Dinner:				
Soup—Hot water and Marmite or Bovril	*Ad lib.*	*Ad lib.*	*Ad lib.*	*Ad lib.*
Lean meat . .	2 oz.	2 oz.	2 oz.	2 oz.
Potato . . .	2½ oz.	3½ oz.	3 oz.	3½ oz.
Onions . . .	3 oz.	Nil	1½ oz.	Nil
Butter or fat of meat	½ oz.	½ oz.	1 oz.	1 oz.
Bread . . .	¼ oz.	¼ oz.	¾ oz.	Nil
Baked rice pudding	Nil	Nil	Nil	Milk 2½ oz., water 3 oz., rice ¾ oz., saccharin or vanilla to taste.
Orange . . .	2½ oz.	5 oz.	2½ oz.	2½ oz.
Tea:				
Tea . . .	*Ad lib.*	*Ad lib.*	*Ad lib.*	*Ad lib.*
Milk . . .	2½ oz.	2½ oz.	2½ oz.	2½ oz.
Vita-weat biscuits .	1½	2	2	3
Butter . . .	Nil	¼ oz.	Nil	½ oz.
Cheese . . .	1 oz.	1 oz.	1 oz.	1 oz.
Celery and Cress .	*Ad lib.*	*Ad lib.*	*Ad lib.*	*Ad lib.*
Apple . . .	2 oz.	2 oz.	2 oz.	Nil
Supper:				
Steamed fish . .	2 oz.	3 oz.	3 oz.	3½ oz.
Vita-weat biscuits .	3	2½	2½	4
Butter . . .	Nil	½ oz.	¼ oz.	1 oz.
Custard . .	1 egg, 5 oz. milk, saccharin or vanilla to taste	Milk 5 oz., banana 2 oz., 1 egg, saccharin or vanilla to taste	Nil	As for Diet B
Rice pudding . .	Nil	Nil	Milk 5 oz., rice (uncooked) ¾ oz., vanilla or saccharin to taste	Nil
Rhubarb stewed with saccharin . .	*Ad lib.*	Nil	Nil	Nil
Carbohydrate . .	100 grammes	119 grammes	120 grammes	148 grammes
Fat	58 "	83 "	114 "	133 "
Protein . . .	70 "	71 "	76 "	81 "
Calories . . .	1,200	1,500	1,800	2,100

Alternative articles of food.—Any amount of the following may be taken *in addition* to the prescribed diet, as they contain very little food material: lettuce, celery, radishes, asparagus, cress, rhubarb, cranberries, green artichokes, mushrooms, lemons, horse-radish, cabbage, greens, spinach, French beans, seakale, cauliflower, scarlet runner, stewed gooseberries, cucumber.

Carbohydrate: The food value of 7 oz. tomato, 3 oz. onion, 2½ oz. orange, 1 oz. banana, and ½ oz. bread are approximately similar and represent one carbohydrate portion of 5 grammes (two half portions of different foods may be taken). These amounts in the diet list may *be substituted* for one another, or by :—

7 oz. of marrow, black currants, water melon, endive (raw), red currants, stewed greengages, damsons, plums, apricots, brussel sprouts, raspberries (raw), loganberries (raw);

or 4 oz. of blackberries (stewed), turnips, leeks, Jerusalem artichokes, strawberries (ripe), apricots (ripe), stewed apples and pears;

or 3 oz. of grapefruit, carrots, ripe cherries, peaches, gooseberries;

or 2½ oz. of ripe greengages;

or 2 oz. of beetroot, raw apple or pear, dried apricots or peaches, ripe plums;

[1] As used in the Diabetic Dept. of Charing Cross Hospital by Dr. R. A. Hickling.

or 1½ oz. of parsnips, prunes (stewed), grapes;
or 1 oz. of potato, peas, broad beans;
or ½ oz. of oatmeal (weighed dry), Force or Cornflakes;
or ½ oz. of rice or tapioca (weighed dry).

Instead of Vita-weat biscuits, you may take the same number of Huntley & Palmer's Breakfast Biscuits, Oval, Bath Oliver, Fancy Lunch, Cream Crackers, Fine Water, Oval Water, Thin Captain, Cornish Water, or Milk biscuits.

Protein: The food value of 1 oz. of lean meat or of Cheddar or Dutch cheese is approximately equivalent to 3 oz. steamed fish, and these may *be substituted* for one another, or in the amounts stated, by:—

1 oz. of the lean of roast beef, mutton, lamb, or by chicken, turkey, goose, game, rabbit, Cheddar or Dutch cheese. (The meat should be weighed cooked and bone not included.);
or 1½ oz. of cod, skate, or dried haddock;
or 2 oz. of eels, sardines, well drained of oil;
or 3 oz. of sole, lemon sole, plaice, whiting, mackerel, hake, herring, haddock (fresh). (The fish should be weighed uncooked without skin or bone.)

Fat: Instead of butter, you may take:—

An equal amount of dripping, margarine, oil, fat of meat, clotted (Devonshire) cream;
or Twice the amount of whipped cream;
or Four times the amount of thin cream.

Saccharin or vanilla may be used for flavouring any articles of food.

X. **Milk Diet.**—The basis of the diet is 8–10 oz. milk every 3 hours from 7 a.m. to 10 p.m., with additional feeds if the patient is awake in the night. The milk may be hot or cold, citrated, junket, or mixed with 2 oz. barley water or lime water, one tablespoonful of cream, or flavoured with vegetable soup stock, tea, coffee, cocoa, ovaltine, ground rice, sago, or one of the invalid foods, if there is no special contra-indication. The addition of 1 oz. bread, three rusks or plain biscuits, and ½ oz. of butter for some meals is allowed.

XI. **Predigested Foods** are indicated in dilatation of the stomach, cancer, and advanced cases of chronic gastritis. Benger's *Liquor Pancreaticus* is the usual ferment employed, because the pancreas contains both a proteolytic and a diastatic ferment. *Taka-diastase* is a valuable aid in the digestion of farinaceous foods. The patient takes it with his food at the commencement of the meal.

1. *Peptonised Milk.*—One pint new milk. One tube Fairchild's Zymine peptonising powder. Five oz. cold water. Method: Mix peptonising powder with cold water, add to milk heated to 105° F.; keep at this temperature for the time ordered (5 to 30 minutes). Bring rapidly to the boil to stop peptonising process.

2. *Peptonised Beef-Tea.*—Half a pound of finely minced lean beef is mixed with a pint of water and 20 grains of sod. bicarb.. This is simmered for an hour. When it has cooled down to a lukewarm temperature, the peptonising powder is added. The mixture is then set aside for three hours, and occasionally stirred. At the end of this time the liquid portions are decanted and boiled for a few seconds.

3. *Other foods* can be similarly prepared.

4. *Nutrient Enemata.*—Glucose alone is of practical use.

XII.—**Beef-Tea.**—Cut up a pound of lean beef into pieces the size of dice; put it into a covered jar with 2 pints of cold water and a pinch of salt. Let it warm gradually, and simmer for a couple of hours, care being taken that *it does not boil.*

XIII. **Improved Beef-Tea.**—Three-quarters of a pound of steak, scraped or passed through a mincing machine, and pounded; ¾ pint of cold water; one piece of sugar, one pinch of salt, one teaspoonful of tapioca; simmered in a "Gourmet Boila" for three hours.

XIV. **Artificial Protein Foods.**—Beef-tea and other meat preparations do not contain the nutritive constituents of meat, except in small quantities, but may be useful as

stimulants of gastric secretion. *Peptonised albumin* (or peptonised meat) is more nourishing, but the taste of peptone is very bitter and nasty. The *albumoses* are intermediate between albumin and peptone. They are freely soluble, tasteless, and readily digested and absorbed. *Plasmon* is another artificial protein food. It is prepared from milk, and contains casein in a soluble form. It is a nutriment of some value. *Casein hydrolysate* can be given by mouth or intravenously.

XV. **Diet for Oxaluria** (after Poulton). Figures in brackets indicate oxalic acid content in mgm. per cent. Foods with high oxalic acid content must be *avoided.* Tea (1,380); cocoa powder (640); chocolate (90); sorrel (2,000); spinach (830); rhubarb (410); dry figs (100); beetroot and potatoes (40); parsnips (39); spring onions (55); red and black currants (70); bilberries and raspberries (42). Foods to be *taken sparingly* are: broad beans (39); French beans (20); lettuce (30); green peas (36); oranges (28); gooseberries (27). A smaller amount of oxalic acid is found in endive, tomatoes, strawberries, plums, carrots and beetroot. It is advisable also to avoid foods with high purin content.

XVI. **Diet for Gout.**—A low purin diet is essential: it is also advisable to exclude foods with a high oxalate content. Articles to be *avoided* are: chocolate, cocoa, strong coffee and tea, port, sherry, gin, burgundy, beer and stout, barley, oatmeal, shellfish, most meat—especially kidneys, liver, sausages, sweetbread, fish roe, caviare, rich sauces and pastry. Also broad beans, brussel sprouts, butter beans, haricot beans, lentils, peas, radishes, spinach, sorrel, raspberries and rhubarb. Foods *taken sparingly* include: simply cooked meat or fish, bacon rashers, mutton, poultry, ham, boiled ox tongue, oysters. Also cider, whisky and brandy. Fats can be taken in small amount: butter, cheese, cream, dripping, suet. Foods *allowed* include: milk, bread, eggs, milk puddings, fruit and vegetables (other than above), nuts, cereals, sugar, jam, honey, marmalade.

XVII. **Routine Treatment of Sprue Cases** (Hamilton Fairley).—

High Protein, Low Fat, Low Carbohydrate Diets.—Protein is largely supplied in the form of lean rump steak, which must be of the first quality. It is prepared by cutting away all skin, fat and gristle, mincing, and then lightly cooking in a dry pan for two to three minutes without the addition of any grease or water. It is continually stirred with a fork until the exterior is greyish in colour, and then rapidly removed and served hot. Much of the meat fibre is still raw, being cooked only sufficiently to render it palatable, in which form it is more readily digested. Rusks may be prepared from ordinary bread by baking thoroughly in an oven until crisp. Before doing so, all crust is removed. It is found that 2½ oz. of bread loses 1 oz. of moisture during the process. Heudebert unsweetened rusks—*biscottes de pain grillé*—are also excellent. When available in the Tropics, ripe papaya may be used either as a substitute for, or in addition to, orange juice in all these diets.

High Protein Diet, No. 1. (Calorie value = 770.) 8 *a.m.*—Underdone beef, 3 oz.; rusks, ¾ oz.; juice of ½ orange; and glucose, ¼ oz. 12 *a.m.*—Soup, 4 oz. + liver extract (= ⅝ lb.); underdone beef, 3 oz.; rusks, ¾ oz.; juice of ½ orange; and glucose, ⅛ oz. 6 *p.m.*—Ditto, 12 a.m. Protein : fat : carbohydrate = 1·0 : 0·3 : 1·2. *Note.*—Where patients are very ill, two hourly feeds of meat and beef juice can be substituted.

High Protein Diet, No. 2. (Calorie value = 1,280.) 8 *a.m.*—Underdone beef, 5 oz.; rusks, 1 oz.; calves-foot jelly, 2 oz.; juice of 1 orange + glucose, ¼ oz. 12 *noon*—Soup, 4 oz. + liver extract (= ⅝ lb.); underdone beef, 5 oz.; rusks, 1 oz.; juice of 1 orange + glucose, ¼ oz. 4 *p.m.*—Tea, 10 oz.; milk, 2 oz. 7 *p.m.*—Ditto, 12 *noon* + calves-foot jelly, 2 oz.. Protein : fat : carbohydrate = 1·0 : 0·3 : 1·0.

High Protein Diet, No. 3. (Calorie value = 1,820.) 6 *a.m.*—Tea, 10 oz.; milk, 2 oz. 8 *a.m.*—Underdone beef, 6 oz.; rusks, 1½ oz.; calves-foot jelly, 2 oz.; juice of 1 orange + glucose, ¼ oz. 10 *a.m.*—1 baked apple; custard, 1 oz. 12 *noon*—Soup, 4 oz. + liver extract (= ⅝ lb.); underdone beef, 6 oz.; calves-foot jelly, 2 oz.; rusks, 1½ oz.; juice of 1 orange + glucose, ¼ oz. 4 *p.m.*—Tea, 10 oz.; milk, 2 oz.;

baked apple, 1 oz.; custard, 1 oz. 7 *p.m.*—Ditto, 12 *noon*. Protein : fat : carbohydrate = 1·0 : 0·32 : 1·3.

High Protein Diet, No. 4. (Calorie value = 2,200.) 6 *a.m.*—Tea, 10 oz.; milk, 2 oz. 8 *a.m.*—Underdone beef, 7 oz.; rusk, 1½ oz.; calves-foot jelly, 2 oz.; juice of 1 orange + glucose, ½ oz. 10 *a.m.*—1 baked apple + custard, 2 oz. 12 *noon.*—Soup, 5 oz. + liver extract (= ⅛ lb.); underdone beef, 7 oz.; calves-foot jelly, 2 oz.; rusks, 3 oz.; juice of 1 orange + glucose, ½ oz. 4 *p.m.*—Tea, 10 oz.; and milk, 2 oz.; 1 baked apple; custard, 3 oz. 7 *p.m.*—Ditto, 12 *noon*, but only 1½ oz. of rusk allowed. Protein : fat : carbohydrate = 1·0 : 0·34 : 1·3.

Convalescent Sprue Diet.

Breakfast (8 *to* 9 *a.m.*).—Lightly boiled or poached eggs; underdone lean chop or steak; filleted, boiled or steamed fish—whiting, sole, plaice, haddock; thin toast, and butter in moderation; weak tea; stewed fruit: apples, rhubarb; honey or jam in small quantity; also butter. 11 *a.m.*—½ pint of milk, if desired, and if it agrees. *Lunch.*—Chicken or vegetable soup; underdone grilled steak; roast or boiled chicken; liver cooked in various ways; cold beef or mutton, but fat not to be eaten. Vegetables: spinach, marrow, cauliflower, French beans, celery, young peas, boiled onions; salad of lettuce and tomato; boiled potatoes in small quantity. Custard, junket, milk jellies, and jelly with fruit, such as bananas, etc. Baked apples. Small quantity of cream allowed. Fresh fruits such as oranges, Canary bananas, pears, peaches, grapes, raspberries, strawberries, melons, grapefruit. Rusks or toast allowed. *Tea,* 4 *p.m.*—Weak China tea, Madeira cake or sponge cake, dry toast and butter, Marie or water biscuits. *Dinner,* 7 *p.m.*—Chicken, rabbit, brains, sweetbread, tripe, cold lean meat; lettuce and tomato salad or other vegetables mentioned, but no potatoes; custard, junket, baked apple, stewed fruit or fruit in jelly, and a small quantity of fresh cream; rusks or toast and butter allowed.

Articles to be avoided.—Avoid overdone and twice-cooked meat and articles fried or cooked in fat. Condiments, like pepper, mustard, chillies, sauces, chutneys, curries and spiced food. Game, duck and fat fish, such as salmon, trout, mackerel and herrings. Fresh bread, grease, fat, salad oil dressings and sauces of all kinds, suet puddings, cakes with icing, raisins and pastry. Sweets and chocolates. Alcoholic drinks and mineral waters.

Note.—Smoking in moderation is allowed once convalescence has been established.

XVIII. **Diet in fevers** should consist mainly of nourishing fluids, water, barley-water, orangeade or lemonade with glucose (1–2 oz. per pint), lemon and barley-water, weak tea and Imperial drink allowed *ad lib.*, with milk (½–2 pints daily), egg and milk, oxo, bovril, meat or chicken broth, calves-foot jelly. Especially if there is high fever or much sweating, the total fluid intake should be 3–5 pints daily. Milk may be diluted with half to two-thirds of water, soda-water, or barley-water. If curds are passed, the milk may be peptonised, or sodium citrate may be added in the proportion of gr. 2 to the ounce of milk. Lime-water may be used instead if diarrhœa be present. If milk is not well tolerated, whey or cream may be given, or the yolks of eggs or egg-flip. Where intestinal infection is present, meat extracts and jellies are better avoided. Some invalid foods are given below. Iced water is agreeable, but generally increases the thirst.

XIX. **Milk, Egg, and Brandy.**—Scald some new milk, but do not let it boil. Put it into a jug, and the jug into a dish of boiling water. When the surface looks filmy, it is sufficiently done, and should be put away in a cool place in the same vessel. When quite cold, beat up a fresh egg with a fork in a tumbler, with a lump of sugar; beat quite to a froth, add a dessertspoonful of brandy and fill up the tumbler with scalded milk.

XX. **Imperial Drink.**—Acid potassium tartrate 60 grains, oil of lemon 3 drops, flavoured with sugar, or saccharine 1 grain, and dissolved in a pint of boiling water.

XXI. **Whey.**—Into a vessel of warm milk put sufficient quantity of rennet to cause curdling, and strain off the liquid, which is then ready for use.

XXII. **Sherry Whey** (especially good for infants with summer diarrhœa).—Half a pint of milk is boiled: as soon as it boils, add 2½ fluid oz. of good cooking sherry; allow the mixture to boil for a few minutes, then leave in a cool place in a basin. When the curd falls to the bottom, carefully pour off the whey, or strain through muslin. In grave conditions, with vomiting, give a teaspoonful every ten minutes; in inflammatory diarrhœa give a tablespoonful every hour.

CHAPTER XI

THE INTESTINAL CANAL

The physiological importance of the intestinal canal is evidenced by the fact that its length is between 25 and 30 feet, along the whole of which absorption may take place; yet the first feature of intestinal disorders which strikes the student is their inaccessibility to examination. Healthy individuals often show considerable variations in the size, length and position of the large intestine; on the other hand, dilatation and redundancy of the colon may be associated with disease. Micro-organisms or their toxins can make their way through the mucous membrane of the intestine into the lymph spaces beneath, and thence into the glands and the circulation, particularly when the mucous membrane is unhealthy, abraded, or ulcerated. The bacteriology and the chemistry of the intestinal contents are now assuming much importance and the examination of the stools is necessary in every complete investigation of a patient. X-ray examination after a barium meal has thrown much light on the nature of the intestinal movements. In the small intestine they are churning in character and result in a to-and-fro or pendulum action, with a peristaltic wave over a short length of intestine at intervals. The residue from the small intestine slowly fills the cæcum and ascending colon to the neighbourhood of the hepatic flexure. At intervals, usually after a meal (gastro-colic reflex), the mass of contents of the cæcum and ascending colon is rapidly passed through the remainder of the colon and evacuated, though usually a small residue remains in the sigmoid until the next mass movement takes place.

Another striking feature about diseases of the intestines is the disproportionate amount of prostration which accompanies them. When a patient is attacked by a slight but sudden diarrhœa or abdominal pain, the feeling of exhaustion, which in some cases may amount almost to collapse, is out of all proportion to the local mischief. This disproportionate degree of prostration or collapse is especially seen in early life, when "diarrhœa" is, mainly on this account, found to be one of the chief causes of death in children under two years of age. Again, among the acute specific fevers fatal collapse and prostration often occur in those in which the chief lesion is in the intestinal canal—in cholera, dysentery, and typhoid fever. This may be due in part to the large vascular bed in the abdominal cavity, and to the extensive surface through which toxins can be absorbed.

PART A. SYMPTOMATOLOGY

§ 301. The cardinal symptoms of intestinal disorder are Abdominal Pain, Diarrhœa, Constipation, and Intestinal Discomfort.

ABDOMINAL PAIN is frequently present, especially in the more acute conditions and may be due to many conditions within the abdominal cavity (see § 241). DIARRHŒA and CONSTIPATION are dealt with in Part C.

INTESTINAL DISCOMFORT may be due to colic, peritoneal pain or strangulation, or distension with wind, accompanied by borborygmi. It is a marked feature in COLOSPASM, which occurs in two conditions: (i.) reflex, as from adhesions after appendicitis or operation, with gallstones, and in the early stages of diverticulosis; (ii.) with worry, tense brain work and anxiety, associated with depression (visceral neurosis) (§ 252).

The GENERAL or REMOTE symptoms, such as loss of appetite from toxæmia and discomfort, are sometimes (especially in acute cases) of a very severe character, in view of the profound PROSTRATION which is associated with some intestinal disorders. PYREXIA is not usually a marked feature (see § 239). In the more chronic forms of intestinal disease EMACIATION is apt to ensue from malnutrition. The SALLOW SKIN of intestinal toxæmia is well known. Various NERVOUS DERANGEMENTS of a neurasthenic type are sometimes, as in gastric diseases, associated with disorders of the intestinal canal, consequent partly on mal-assimilation and intestinal toxæmia, and partly, no doubt, arising in a reflex manner. Less troublesome reflex symptoms—*e.g.*, vague pains, itching of the nose, or bad dreams—may be associated with intestinal parasites, constipation and other intestinal conditions.

PART B. PHYSICAL EXAMINATION

§ **302.** The physical investigation of the intestinal canal must be accomplished by an EXAMINATION OF THE ABDOMEN AND OF THE FÆCES. In all cases of abdominal disease a RECTAL EXAMINATION should be made. X-RAY and SIGMOIDOSCOPIC EXAMINATIONS are called for in some cases.

Examination of the Abdomen.—PALPATION and PERCUSSION will enable us to make out any general swelling or local tumour. The tenderness which often accompanies intestinal disorders may also be elicited. A loaded cæcum or descending colon, or the *scybala* present within the colon may be felt; these should not be mistaken for the nodules of cancer or other tumour. Their mobility is a very deceptive feature, and the occasional association of diarrhœa may delude us. Their disappearance after active purgation or a course of enemas, is the only certain method of diagnosis. The reader is referred to § 240 for further details as to examination of the abdomen.

§ **303.** An **Examination of the Stools** is always important, and sometimes absolutely necessary for the diagnosis of intestinal disorders. A great deal of information can also be thus obtained with regard to diseases of the other abdominal viscera. The fæces should be examined *first* as to their physical properties—size, consistence, colour, shape, odour, and reaction; *secondly*, for undigested food and other substances, such as

mucus, gall-stones, or parasites; *thirdly*, for the presence of blood or pus; *fourthly*, a microscopic examination. *Lastly*, culture of the stools is often of great value. One can rarely rely on a patient's statement, even as to the colour and appearance of the stools; they should be inspected by the physician. Early disease of the pancreas and intestinal canal can be detected by thorough investigation. For the technique of these examinations the student should consult pathological text-books.

It is preferable to see the fæces in bulk, the patient having used a night-stool. He should pass urine before going to stool. A large wide-mouthed glass jar, closed at the top by a stopper, is a convenient receptacle for their preservation. Nothing should be added to the motion until the doctor has examined it.

Physical Properties of the Stools.—(1) The *Consistency* is normally solid or semi-solid and the form roughly cylindrical. About four ounces are passed daily on an ordinary diet, but when fat is inadequately absorbed, as in sprue, the bulk is much increased. (*a*) When passed in hard, dry, roundish balls they are known as *scybala*, this condition being due to defective intake of fluid or its excessive absorption by a " greedy " colon. Scybala are generally coated with mucus and sometimes the irritation they cause sets up a spurious diarrhœa which may alternate with constipation. (*b*) *Pencil-like* stools may result from spasm of the anal sphincter, possibly associated with fissure, while *ribbon-like* stools may be produced by colospasm or stricture of the rectum resulting from cancer, syphilis, or gonorrhœa. (*c*) *Uniformly fluid* stools are common in lesions of the small intestine like typhoid, sprue and tuberculous or simple enteritis: in lesions of the large bowel the evacuations are generally more fæcal and slimy.

(2) The *Colour* of the fæces varies normally from light to dark brown, due to stercobilin, chlorophyll and other pigments; the depth of colour affords a fair index of the amount of bile passing into the intestinal canal. As diarrhœa progresses they become lighter in colour. *Pale-coloured stools* may be due to (*a*) obstruction to the entrance of bile into the intestine as in jaundice; (*b*) dilution of the stool as in cholera; (*c*) excess of unabsorbed fat; (*d*) a milk diet. Characteristic naked eye appearances are: (i.) *Clay-coloured* stools, found in obstructive jaundice, and pale, bulky, acid stools occur with steatorrhœa, due either to defective pancreatic secretion or defective fat absorption, as in tropical sprue, non-tropical sprue and cœliac disease; (ii.) *tarry stools*, dark or black coloured, due to blood entering the alimentary canal *high up*, as in duodenal ulcer. Black stools are also seen in patients taking iron, bismuth and charcoal; (iii.) " *red-currant jelly* " or " *strawberry ice* " stools are seen in intussusception. (iv.) *Streaks of blood* may be present with local lesions such as hæmorrhoids, or fresh blood in conditions such as ulcerative colitis, or cancer of the bowel and acute dysentery, when it is generally associated with mucus. *Muco-purulent* stools are also met with in the latter disease. Other characteristic stools are: (v.) the *green* stools of dyspeptic diarrhœa and enteritis of infancy and after calomel; (vi.) the odourless, colourless " rice-water "

stools of cholera, alkaline in reaction and containing flocculi of mucus and epithelium; (vii.) the frothy, acid, yellow stools resulting from excessive carbohydrate fermentation (the gaseous stool characteristic of sprue has a similar origin); (viii.) the soft, brown, offensive, alkaline stool of protein putrefaction; (ix.) the bilious "pea-soup" stools of typhoid.

(3) The *Odour* of the stools is due to skatol and indol, and is largely governed by the amount of meat ingested. A characteristic gangrenous smell is met with in severe ulceration, cancerous, dysenteric or syphilitic. An ammoniacal odour, if present, originates from contamination with urine.

(4) The *Reaction* of the fæces is normally amphoteric; with excess of protein it is alkaline, and of starchy foods and fats distinctly acid. The stool should be tested soon after being passed by moistening red or blue litmus paper with distilled water and rubbing a small portion of the stool on the paper; the colour reaction is seen on the other side. Steatorrhœa, due to pancreatic disease, sprue or cœliac disease, often yields acid-reacting fæces.

VARIOUS SUBSTANCES may be found:—

1. UNDIGESTED PARTICLES OF FOOD, if in excess, are indicative of imperfect digestion (gastric or intestinal), and, unless the food has been excessive, denote especially intestinal or pancreatic disease (see also p. 371 and § 256). In children this feature usually indicates over-feeding. Small, hard concretions, consisting of phosphates and other matter, are sometimes found. By noting those articles of diet (protein, vegetable, fruit, or carbohydrate) which pass for the most part undigested, the physician learns which the patient should reduce.

2. MUCUS in the fæces is often overlooked unless specially sought for. To discover it satisfactorily *water must be added* to the fæces, when any mucus present will be seen floating about like small pieces of jelly. The presence of mucus in small amount is of no consequence; it is usual in constipation. When in quantity, and intimately *mixed with the fæces*, it indicates catarrh of the *small* intestine. When in *isolated masses* it signifies the presence of catarrh of the large bowel. In membranous, or mucous, colitis, *long cylinders* of mucus are passed, sometimes without much fæces. These cylinders are generally swarming with organisms of both coliform and streptococcal types, which infest the colon.

3. BLOOD in the stools may appear either in streaks or in quantity, when from rectum or large bowel. If it comes from the stomach or small intestines, it will have undergone partial digestion and gives to the stools a tarry appearance (melæna). In either case it reddens the water in which the stool is placed, and gives the characteristic spectrum.[1] The *causes* are dealt with below (§ 314). *Occult* blood must be tested for in cases of suspected oozing from an ulcerated surface.

[1] Cases have been recorded where, after standing for some time, the fæces developed on the exposed surface a colour resembling blood, but no blood was detected by the spectroscope. It appears that in certain as yet unknown conditions some pigment is present in the fæces, which on exposure to the air becomes red like blood.

Occult blood test in fæces. The patient should be given a purge three days before the intended investigation, and take a hæmoglobin-free diet—milk and milk puddings, bread and butter, eggs, cheese, potatoes, fruit, tea, coffee or cocoa. Meat, meat extracts, liver or liver extracts, soups, poultry, game, fish, and green vegetables, must not be given. A charcoal biscuit given at the beginning of the diet will indicate when a fæcal specimen may be collected. During this period a soft tooth-brush should be used, lest bleeding from the gums occur. A series of tests over consecutive weeks is of importance where the result is positive. If each is positive the diagnosis is in favour of malignant ulceration; if the positive result is intermittent it is in favour of simple ulceration.

To a piece of fæces of the size of a walnut, add 5 c.c. each of glacial acetic acid and water, preferably in a large "boiling-tube." Break up the fæces thoroughly with a glass rod, add an equal quantity of ether, and stir well. The ether extracts all the blood pigments, and if it does not rise spontaneously to the surface add water till it does so. Decant the ethereal extract and divide it into two parts. To the first apply the guaiacum test (§ 382). To the other add a quarter of



Fig. 74.—Microscopic Elements of Normal Fæces: *a*, muscle fibres; *b*, connective tissue; *c*, epithelial cells; *d*, white blood corpuscles; *e*, spiral vessels of plants; *f–h*, vegetable cells; *i*, plant hairs; *k*, triple phosphate crystals; *l*, stone cells. Scattered among these elements are micro-organisms and debris.

its volume of conc. HCl, and shake. The acid hæmatin dissolves in the ether layer at the top, while the acid hæmatoporphyrin dissolves in the watery layer underneath. These pigments may be examined for by the spectroscope (Plate IV). If the bleeding is from the lower colon, the blood is not appreciably altered, and a positive guaiacum reaction and the presence of acid hæmatin is shown. If the blood is from a high level (*e.g.*, stomach or duodenum), only acid hæmatoporphyrin will be present; but when the bleeding is considerable, some undigested acid hæmatin will be present in addition.

4. Pus always indicates *ulceration* of the rectum or colon, which may be due to ulcerative colitis, dysentery, cancer, tuberculosis or of syphilitic origin (§ 310). Pus is difficult to detect when diarrhœa is present. When in large quantity, pus indicates an abscess bursting into the bowel, such as a pelvic or ischio-rectal abscess.

5. Gall-stones may be found by mixing the stools with water, and passing the mixture through muslin or a fine sieve. Gall-stones sink in water when recently passed, though they float when dried. They are

often friable, and any suspicious particles should be examined under the microscope for cholesterin; see Fig. 86.

6. WORMS, see Tables XVIII and XIX, § 304 and § 316.

7. Various FLY LARVÆ are common; generally they are deposited after defæcation, but sometimes man swallows the eggs and larvæ develop later in the gut, giving rise to intestinal myiasis; gastro-intestinal and toxic symptoms may result. A dose of castor oil is generally effective; if not, thymol, santonin or turpentine may be subsequently administered.

Microscopic Examination of the fæces (Fig. 74) is often necessary to diagnose pathogenic protozoa and helminthic ova. As a routine a loopful of mucus or fæces should be rubbed up on slides, with (1) warm physiological saline (0·9 per cent.); (2) Lugol's iodine solution, and cover slips applied. Smears stained by Gram's method may also be made. NORMALLY, under the microscope, a few undigested starch granules, fat cells and partially digested muscle fibres may be observed, also crystals of fatty acids, oxalate of lime and other calcium salts. Hæmatoidin, phosphates, cholesterol, and Charcot-Leyden crystals are rare. Various bacteria, cocci and yeasts are found, as well as occasional epithelial cells.

1. *Abnormal* constituents to look for first are: ova, segments of tape-worms, flukes and nematodes (§ 304).

2. Amongst the *undigested food products* note any excess of muscle fibres, starch granules and fat. STARCH GRANULES stain blue with iodine, and if their presence is pathological the stools are usually acid and show signs of fermentation (gas bubbles) and the presence of yeasts. With starch indigestion excessive gas is formed in fermentation tubes in the incubator. Where digestion of PROTEIN is defective there is an excess of undigested muscle fibres showing cross striations and frayed ends: the stools are generally brown, offensive and alkaline. Neutral FAT droplets are soluble in ether and stain with Sudan III; fatty acids show up as sheaves of colourless, acicular crystals, while soaps form greasy amorphous masses which dissolve on heating. Normally the total fæcal fat does not exceed 25 per cent. of the dried fæces and the ratio of split to unsplit fat = 3 : 1. Excess of fat amounting to 30–80 per cent. of the dried fæces indicates: (1) defective bile secretion; (2) disease of the pancreas (§ 256) with defective external secretion; (3) intestinal disease interfering with absorption. In (1), as in obstructive jaundice, there is lack of bile salts with resulting defective absorption; the fat, however, is mainly split. In (2), as in chronic pancreatitis, fat may fail to be adequately split owing to the diminution of lipase in the pancreatic juice. In (3) splitting is adequate, the excess of fat resulting from malabsorption; this may occur in tropical sprue (§ 311), idiopathic steatorrhœa, cœliac disease of infants, gastro-colic and gastro-jejuno-colic fistula, and in lymphadenoma, lymphosarcoma and tuberculosis involving the mesenteric lymph glands. To ensure reliable results for fat analysis the patient must be on a normally balanced diet for at least three days previously, and without liquid paraffin.

3. Various *micro-organisms*, such as those of the typhoid, dysentery, tubercle and cholera group, may be found in the fresh stool; and in Shiga, Flexner and Sonne dysentery it is important to make cultures from recently passed mucus at as early a stage of the disease as possible. The bacterial flora, including *B. coli*, streptococci, anærobes, etc., may be modified in certain conditions by diet and purgation as well as by the administration of *B. acidophilus* and antiseptic drugs.

4. *Intestinal sand* consists of fine granules of calcium salts and silica formed around an organic nucleus, or of granules from pears or other fruit. 5. *Charcot-Leyden* crystals are common in amœbiasis, but also occur in ankylostomiasis and mucous colitis.

§ 304. Certain Intestinal Parasites and their ova, described in Tables XVIII and XIX, may be found in the fæces (Figs. 75 to 81). In addition, the operculated eggs of certain intestinal and liver flukes are met with in tropical countries. Segments of tapeworms often appear in the stools: held between two glass slides and examined with a

Table XVIII.—The Principal Pathogenic Helminths of the Intestine.

For treatment, refer to § 316.

Specific Name.	Chief Characteristics of Worms and Habitat.	Clinical Features.	Ova or Embryo; Chief Characteristics, and Where Found.	Animal Hosts, etc.
Cestodes. **Tænia saginata.** (Tapeworm in man.) Fig. 75.	14 to 24 ft. long. Head, 4 suckers, no hooklets. Segments, over 1000, show central stem uterus with 15 to 30 lateral *dichotomous* branches. Fastens itself to mucosa of small intestine in man.	Reflex irritation, digestive or nervous disorders. Segments passed per rectum.	Recognised by segments containing ova discharged in fæces. Ova similar to those of *T. solium*. Cystic stage in beef (35μ × 25μ).	Cattle the intermediate hosts. Found in Great Britain. Wide-spread geographical distribution. Man infected by eating under-done beef.
Tænia solium. (Tapeworm in man.) Fig. 76. (Cysticercosis also in man.)	About 7 to 10 ft. long. Head, 4 suckers, and double row of 26 hooklets. Segments, about 850, show central stem uterus with 7 to 10 lateral *ramifying* branches. Fastens to mucous membrane of small intestine in man.	Ditto—from adult worm. Epilepsy from embryos (Cysticercosis).	Recognised by segments containing ova discharged per rectum. A six-hooked embryo inside ovum which, eaten by pig, bores its way into the flesh. Ova spherical; 35μ in diameter.	Pig the intermediate host. Adult worm takes 3 months to develop in man, who becomes infested by eating "measly pork." Man may also act as intermediate host, ingestion of eggs leading to Cysticercosis.
Diphyllobothrium latum or **Dibothriocephalus latus.** Fig. 77.	16 to 25 ft. long. Head club-shaped, with long lateral grooves. No hooklets or suckers. About 3000 segments; uterus, rosette-shaped. Found in intestinal canal of man.	Occasionally anæmia of pernicious type. Intestinal disorder in children.	Segments containing ova discharged per rectum. Sometimes ova discharged alone; brown shelled with a lid at one end, broadly oval (70μ × 45μ).	Ova hatch on reaching water. Swallowed by a *Cyclops* which is eaten by a fish (intermediary host). Chiefly found in Switzerland and other parts of Central Europe, also in U.S.A.
Echinococcus granulosus or **Tænia echinococcus.** (Hydatid cyst in man; tapeworm of dog). Fig. 85.	⅛ in. to ¼ in. long. Head pointed, with 4 suckers; double row of hooklets. Has 4 segments, the 4th longer than all others. Found in intestinal canal of dogs, wolves or jackals.	Hydatid cysts form in liver, or other organs in man, sheep, cattle and pigs.	Ova found in fæces of dog or wolf. Embryo becomes encysted in various organs, especially liver and lungs.	Man, sheep and cattle are intermediary hosts: Man becomes infected from contaminated food or water, or from contact with dogs to whose coats and mouths ova may be adherent. Found chiefly in Australia, New Zealand, Argentine and Iceland; occasional cases occur in Great Britain and elsewhere.

NEMATODES. **Enterobius vermicularis** or **Oxyuris vermicularis.** (Threadworm.) Fig. 78.	F. = 8–13 mm.; M. = 2–5 mm. in length. Found in large intestine, chiefly the rectum.	Reflex irritation. Worms tend to migrate at night, and cause itching of anus and genitals.	Worms emerge from anus at night to lay eggs in sticky masses on peri-anal skin. Fingers contaminated by scratching cause reinfection. Ova ($50\mu \times 20\mu$) thin-shelled, planoconvex and contain coiled embryo.	Often trouble children. Found in all countries.
Strongyloides stercoralis.	Only female worms found in intestine—1 in. long. Males develop outside body from rhabditiform larvæ.	Early—dermatitis and lung symptoms. Later—sometimes diarrhœa and urticaria.	Thin-shelled, oval eggs ($60\mu \times 30\mu$) which hatch out free rhabditiform larvæ passed in fæces.	Sexual cycle outside human body. The rhabditiform larvæ differ from ancylostome larvæ in being free and having a short presœsophageal mouth.
Ancylostoma duodenale. (Old world hookworm.) Fig. 81.	M. = 8–10 mm.; F. = 12–18 mm.: buccal cavity large and contains two pairs of ventral teeth; attached to mucosa of jejunum.	Asthenia: mental lethargy; hypochromic anæmia: eosinophilia: œdema: serous effusions.	Ova = $60\mu \times 40\mu$, thin, transparent shell showing 2–8 stages of segmentation. Ova found in fæces, rhabditiform larvæ rarely.	Man is infected by filariform larvæ in the soil penetrating human skin. Widespread geographical distribution.
Necator americanus.	M. = 7–9 mm.; F. = 9–12 mm. The buccal cavity is small with a less effective biting apparatus.	Ditto.	Ova = $70\mu \times 36\mu$ slightly narrower and longer than **A. duodenale.** Found in fæces.	Ditto.
Ascaris lumbricoides. (Roundworm.) Fig. 79.	M. = 6 in.; F. = 12 in. Inhabits the small intestine of man.	Early—urticaria and ascaris larval pneumonia. Later—produce symptoms by toxic, reflex and mechanical means.	Ova are yellow, elliptical with thick outer shell showing excrescences. ($60\mu \times 45\mu$.)	World-wide distribution; very common in children, also adults. May wander widely in human host.
Trichuris trichiura or **Tricocephalus dispar.** (Whipworm.) Fig. 80.	Length = 1¼ in. found in cæcum; anterior portion thread-like.	Generally nil. May be reflex disturbances, urticaria or verminous appendicitis.	Ova brown, barrel-shaped with terminal knobs. ($50\mu \times 23\mu$.)	Cosmopolitan distribution. Man is infected by swallowing embryonated eggs.

TABLE XIX.—SOMATIC HELMINTHIC INFESTATIONS OF MAN.

SPECIFIC NAME.	CHIEF CHARACTERISTICS OF WORM AND HABITAT.	CLINICAL FEATURES.	OVA OR EMBRYO; CHIEF CHARACTERISTICS, AND WHERE FOUND.	LIFE CYCLE AND GEOGRAPHICAL DISTRIBUTION.
TREMATODES. **Schistosoma hæmatobium** (Vesical Schistosomiasis.) Fig. 107. Syn., Vesical bilharziasis.	M. = 10–15 mm.; F. = 20 mm., female lives mainly in gynæcophoric canal of male in portal veins and pelvic venous plexuses.	Terminal hæmaturia, frequency, perineal, penile and loin pain. Eosinophilia.	Terminal spined ova (120–160μ × 40–60μ) containing a ciliated miracidium passed in urine.	Africa, Syria, Arabia and Mesopotamia. Intermediary host mainly *Bulinus* species of snail: man infected by cercariæ penetrating skin while bathing.
Schistosoma mansoni (Intestinal Schistosomiasis.) Syn., Intestinal bilharziasis.	M. = 12–20 mm.; F. = 12–16 mm. Worms inhabit portal and mesenteric veins.	Schistosomal dysentery, papillomatosis of colon common. Sometimes cirrhosis and splenomegaly. Eosinophilia, anæmia.	Lateral spined ova (140–165μ × 60–70μ) containing a ciliated miracidium passed in fæces.	Africa, South America. Intermediary host mainly *Planorbis* species of snail. Infection ditto.
Schistosoma japonicum (Japanese Schistosomiasis.)	M. = 12–20 mm.; F. = 18–26 mm. Worms inhabit portal and mesenteric veins.	Ditto.	Ova (70 × 100μ–50 × 65μ). Have a lateral knob; passed in fæces.	China and Japan. Intermediary host mainly Katayama and *Oncomelania* species of snail. Infection ditto.
Clonorchis sinensis.[1] (Liver fluke.)	Adult is a spatulate fluke (10–20 mm. × 2–5 mm.). Found in the bile ducts of man.	Anorexia, epigastric pain, diarrhœa, enlarged liver and ascites.	Oval, brownish ova (30μ × 15μ) with an operculum, found in fæces.	Common in Japan and China. Life cycle is through a snail and fish which has to be eaten by man.
Paragonimus westermani. (**Distoma ringeri**) (Lung fluke.)	Adult flukes (7·5 mm.–12·0 mm. × 4–6 mm.). Live in bronchi, where they produce cystic swellings and dilatation.	Cough, hæmoptysis; physical signs like bronchiectasis and broncho - pneumonia. Causes "endemic hæmoptysis" of Japan.	Broad, oval operculated ova (100μ × 60μ). Appear in brown, rusty sputum; sometimes in fæces also.	Ditto.

[1] **Fasciola hepatica**, the common liver fluke of sheep and other mammals, rarely affects man.

T. solium and *E. granulosus* also produce somatic infestations in man (vide Table XVIII p. 372).

NEMATODES. **Wuchereria bancrofti.**	Worms resemble fine cat-gut. M = 30–40 mm. F. = 75–100 mm. Inhabit lymphatics and discharge embryos, appearing in the blood stream at night.	Transient painful red swellings in arms, legs, scrotum, etc., with eosinophilia and occasionally mild fever; later lymphangitis, elephantiasis, chyluria, etc.	Embryos appear in blood at night: possess a loose sheath (230–320μ × 7·5–10μ). In Pacific filaria is non-periodic.	Mosquito (Anopheline, culicine or aëdine) is the intermediary host. In Pacific vector is *Aedes variegatus*; bites in day time.
Wuchereria malayi. (Syn., Filaria malayi.)	Adult worm resembles W. bancrofti. Embryos are nocturnal.	Similar but less severe.	Sheathed embryos (200–250μ × 5–6μ) in blood.	Mosquito (various species of *Mansonioides*) is the intermediary host.
Loa loa or Filaria loa.	Worms inhabit subcutaneous and retroperitoneal tissues. M= 30–34 mm. F. = 50–70 mm.	Urticaria, painless calabar swellings; worms cross conjunctiva producing conjunctivitis. Marked eosinophilia.	Sheathed embryos (250–300μ × 6–8·5μ) appear in blood from 9 a.m. to 9 p.m., *i.e.*, diurnal periodicity.	Transmitted by mangrove fly (*Chrysops* species) feeding in day time. Occurs in West Africa.
Onchocerca volvulus.[a] (Onchocerciasis.)	Adult male and female worms occur encapsulated in fibrous tissue nodules in subcutaneous tissues.	Subcutaneous nodules. Dermal lesions. Ocular lesions: blindness. Eosinophilia.	Sheathless embryos occur in local lesions and in skin; not found in peripheral blood.	Transmitted by black fly—(*Simulium damnosum*)—in West Africa and Congo Basin.
Dracunculus medinensis. (Guinea-worm.)	Adult female measures 40–80 cm. Inhabits the subcutaneous tissues producing a local ulcer through which embryos are discharged.	Urticaria; anaphylactoid features; local vesicle and ulcer: local abscess, cellulitis, etc.	Larvæ are reflexly ejaculated by female worm; they are filiform, actively motile measuring (500–750μ × 15–25μ).	Larvæ escape into water and undergo development in a water-flea, *Cyclops*; man becomes infected by drinking water containing it. Occurs in India, Africa, Arabia, etc.
Trichinella spiralis. (Syn., Trichina spiralis.)	Adults inhabit the intestine and liberate embryos which migrate to muscles. M. = 1·4–1·6 mm. F. = 3–4 mm.	Gastric and intestinal symptoms followed by fever, eosinophilia and myositis of affected muscles.	Embryos (100μ × 6μ) may be found in laked blood, or identified in piece of muscle removed at biopsy.	Rats act as reservoir hosts. Infection acquired by eating underdone pork in which larvæ are encysted.

[a] ***Onchocerca cæcutiens*** **which causes onchocerciasis in Guatemala and Mexico is transmitted by Simuliid coffee-flies and is probably the same parasite.**

hand lens they are identified by the number of lateral branches each side of the central uterine stem. *T. saginata* has fifteen or more, but *T. solium* never exceeds twelve. The cysticercus stage of the latter parasite may involve the muscles and brain: epilepsy may result (§ 723). The segments of *Diphyllobothrium latum*, the tapeworm of Central Europe, possess a rosette-shaped uterus. Eggs of the common round-worm (*Ascaris lumbricoides*) and the thread-worm (*Enterobius vermicularis, Oxyuris vermicularis*) are often found in Europe, while ancylostomes, including *Ancylostoma duodenale* and *Necator americanus*, the whip-worm, the thread-worm and another nematode, *Strongyloides stercoralis*, are frequent in patients from abroad. For symptoms and treatment, see § 316. The lateral-spined eggs of *Schistosoma mansoni* occur in the

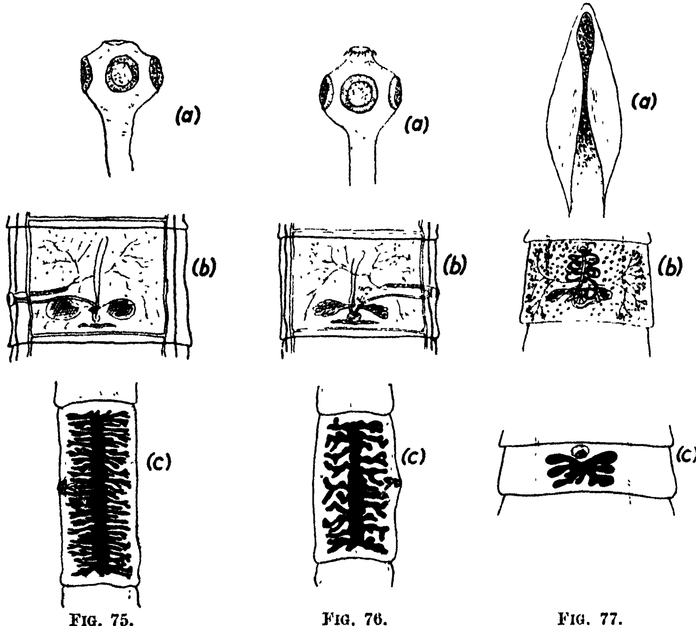


FIG. 75. FIG. 76. FIG. 77.

FIG. 75.—*Tænia saginata.* (*a*) Head × 10; (*b*) maturing segment showing reproductive system × 3; (*c*) segment, showing central stem uterus with 15 *to* 30 *lateral dichotomous* branches, × 3.

FIG. 76.—*Tænia solium.* (*a*) Head × 10; (*b*) maturing segment showing reproductive system × 3; (*c*) segment, showing central uterus with 7 *to* 10 *lateral ramifying* branches, × 3.

FIG. 77.—*Diphyllobothrium latum.* (*a*) Head × 10; (*b*) maturing segment showing reproductive system × 3; (*c*) segment, showing *rosette-shaped* uterus, × 3.

mucous coating of the stool, and the terminal-spined eggs of *S. hæmatobium* which produce hæmaturia are found in urine, but only occasionally in fæces.

Entamœba histolytica, the cause of amœbic dysentery and tropical liver abscess, must be distinguished from other amœbæ, e.g., *E. coli, Iodamœba bütschlii, Endolimax nana*, and *Dientamœba fragilis.* Mucus from a warm, freshly-passed stool is mixed with saline and examined microscopically; diagnosis of *E. histolytica* depends on the presence of an actively motile amœba containing ingested red blood corpuscles. Their cysts occur in the solid fæces and are best demonstrated by mounting in a weak solution of iodine; spherical and less than 14 μ in diameter, they characteristically have a diffuse glycogen mass, chromidial bodies, and one to four nuclei with central karyosomes.

Various flagellates, including *Giardia intestinalis* (Lamblia), are not uncommon, but unless in large numbers their pathogenicity is doubtful. A ciliate, *Balantidium coli*, gives rise to ulceration of the large bowel which may end fatally.

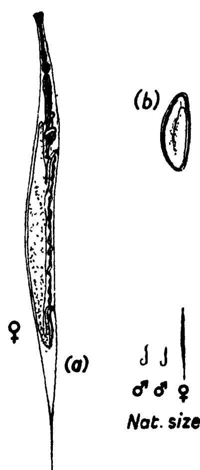


Fig. 78.

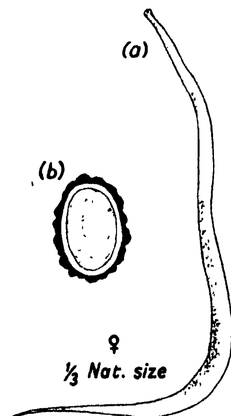


Fig. 79.

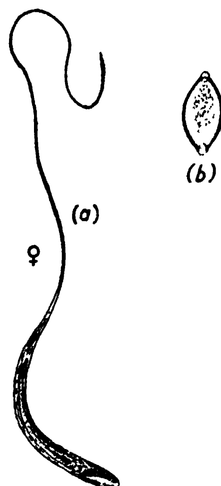


Fig. 80.

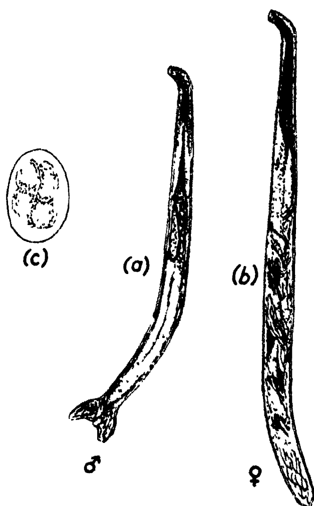


Fig. 81.

Fig. 78.—*Oxyuris vermicularis.*
(*a*) Female × 8, also two male worms and female, natural size;
(*b*) egg × 30.

Fig. 79.—*Ascaris lumbricoides* (Round Worm).
(*a*) Female, one-third natural size;
(*b*) egg × 200.

Fig. 80.—*Trichuris trichiura* (Tricocephalus dispar, "Whip-worm").
(*a*) Female × 30;
(*b*) egg × 170.

Fig. 81.—*Ancylostomum duodenale.*
(*a*) Male × 5;
(*b*) female × 5;
(*c*) egg × 170.

PART C. DISEASES OF THE INTESTINAL CANAL, THEIR DIAGNOSIS, PROGNOSIS, AND TREATMENT

§ **305. Routine Procedure, and Classification.**—Ascertain first that the patient's Leading Symptom is referable to the intestinal canal; and secondly, by inquiries into the History of the illness, whether it came on *acutely* and suddenly, or gradually in a *chronic* manner. In the History, the following points should be investigated: (i.) duration of the present symptoms, previous diseases and operations; (ii.) residence in

tropical climates; (iii.) fever; (iv.) pain and uncomfortable sensations in the intestine; (v.) tenesmus; (vi.) defæcation; frequency or incontinence of fæces, presence of blood or other abnormalities; (vii.) the appetite; any alteration in weight; (viii.) the presence of gastric symptoms may aid the diagnosis. Intestinal Colic is dealt with under abdominal pain without collapse, in § 246. Next proceed to the PHYSICAL EXAMINATION of the abdomen after the manner set forth in Chapter IX (§ 240). If, in the course of these inquiries, definite disease is suspected in any particular organ, reference should afterwards be made to the appropriate chapter.

A. **Diarrhœa** is the leading symptom:		
If *acute*, or attended by choleraic or dysenteric symptoms	turn to	§§ 307–309
If *chronic*	,,	§ 310
B. There is **Tenesmus** without diarrhœa ..	,,	§ 312
or Rectal Spasm	,,	§ 313
C. **Blood** or some other **alteration in the stools** is the leading feature	,,	§§ 314–316
D. **Constipation** is the leading symptom	,,	§ 317
E. **Intestinal Flatulence** is the principal feature ..	,,	§ 318
F. **Stoppage in the Bowels** is complete	,,	§ 319
G. **Pain in the Left Iliac Fossa** is associated with **Fever** and **Constipation**	,,	§ 321

TABLE XX.—CAUSES OF DIARRHŒA.

	Acute.		Chronic.
COMMON.	I. Unsuitable or infected food.	COMMON.	I. Acute causes becoming chronic.
	II. Water.		II. Local conditions about anus.
	III. Intestinal parasites.		III. Ulceration, other than ulcerative colitis.
	IV. Infantile diarrhœa.		IV. Chronic or mucous colitis.
RARE.	V. Typhoid and toxic blood conditions.		V. Ulcerative colitis.
	VI. "Chill."		VI. Portal obstruction or congestion.
	VII. Acute ulcerative colitis.		VII. Dysenteric diarrhœa.
	VIII. Some causes of chronic diarrhœa.		VIII. Nervous diarrhœa.
	IX. Dysentery.	RARE.	IX. Amyloid disease.
	X. Cholera.		X. Senile diarrhœa.
			XI. Mineral poisons (*e.g.*, arsenic).
			XII. Gastrogenous diarrhœa.
			XIII. Pancreatic disease.
			XIV. Gastro-colic fistula and gastro-enterostomy.
			XV. Rare constitutional causes.
			XVI. Sprue.

§ **306. Diarrhœa** is the frequent occurrence of loose or liquid motions; it is the *watery consistence* of the stools which is the chief characteristic. A frequent call to stool may arise from some local irritation (see Tenesmus), without any alteration in the consistency or form of the stool. This source of fallacy should be guarded against by careful inquiry.

Examination of the fæces (§ 303) may show the situation of the disease.

Thus, for instance, when the stools are coloured with bile, and contain undigested food, and *small pieces of mucus intimately mixed* with the fæces, catarrh of the small intestine may be suspected. When mucus or "slime" occurs in *larger masses*, in "strings" or "casts," it points to disease of the large intestine.

§ **307.** In **Acute Diarrhœa** there is usually a good deal of pain and tenesmus (straining at stool); the tongue is usually furred, there is thirst, and may be vomiting. Profuse vomiting and prostration indicate some violent irritant, or serious organic lesion of the bowel or peritoneum. In profuse diarrhœa the temperature is usually subnormal, and the urine diminished. Scybala retained in the intestines may give rise to attacks of diarrhœa alternating with constipation. The possibility of a "controlling appendix" is also to be considered.

Causes.—I. The **food** taken, and the vessels in which it has been contained and cooked, should be the first questions in all cases of acute diarrhœa coming on suddenly in a healthy person. Collapse and many of the symptoms of cholera can be produced by food cooked in a new copper vessel. One of the irritant poisons may have been introduced into the food accidentally or designedly. This should be borne in mind; and in cases of sudden and unexplained diarrhœa the physician should patiently consider every article taken at every meal during the preceding twenty-four hours, *e.g.*, unripe, over-ripe or decomposing fruit, too much raw vegetable food, meat which has been long in store and has undergone putrefaction. In this variety of acute diarrhœa there may be a considerable degree of intestinal colic (§ 246). The most common infections causing diarrhœa and colic include the bacillary dysenteries (Flexner, Shiga or Sonne); also the *B. enteritidis* (Gærtner) and *B. ærtrycke*, which in hot weather may occur in meat, fish, crabs, mussels, cheese and other milk products. *Staphylococcus aureus* and its toxins can cause acute outbreaks of gastro-enteritis even as an epidemic. The first or diarrhœic stage of *trichinosis* should be considered in pork-eating countries; in cases of acute diarrhœa in which trichinosis is suspected, the worm should be sought in the fæces. The diarrhœa which precedes the intestinal obstruction caused by *intussusception* in children frequently follows a heavy meal of indigestible articles; and diarrhœa is itself a cause of intussusception.

II. The quality of the **water** is often responsible for diarrhœa, acute or chronic. This is frequently the case in malarial districts in the summer and autumn, especially when the temperature is high. Soft water containing much peat from the mountains may be a cause.

III. Of the **intestinal parasites**, worms often cause diarrhœa, especially in children, who may have had uneasy abdominal sensations, night terrors, picking of the nose, itching of the anus, vulvo-vaginitis; but sometimes they are discovered in the stools when there have been no symptoms (§ 316).

Lamblia intestinalis (Giardia) is a frequent inhabitant of the small intestine: small numbers of organisms are not pathogenic, but very large numbers can cause recurrent attacks of acute diarrhœa, with mucus and some blood in the stools.

Treatment: mepacrine hydrochloride B.P. (atebrin) 0·1 G. t.d.s. by mouth for five days is a certain cure.

IV. **Infantile Diarrhœa** occurs in at least three well-recognised clinical forms: (i.) Acute Catarrhal or Dyspeptic Diarrhœa; (ii.) Inflammatory Diarrhœa or Entero-colitis; and (iii.) Epidemic Diarrhœa or "summer diarrhœa" (including Infantile Cholera)—mentioned in progressive order of severity.

(i.) In Acute Catarrhal (dyspeptic) Diarrhœa the stools are offensive, at first yellow, then greenish, slimy and mixed with curds of undigested food. Vomiting may or may not be present. It is usually transient if adequately treated.

(ii.) In Infantile Inflammatory Diarrhœa (Entero-colitis) the stools are green, slimy and often contain mucus and streaks of blood; there is some fever at the beginning, and abdominal distension. The stools vary with the predominant infection; they are acid and frothy in the fermentative type, alkaline and green in the putrefactive variety. The inflammation attacks chiefly the colon; consequently there is tenderness on pressure over the region of the colon. Prostration is great when much vomiting occurs. Adults also are sometimes affected. It lasts only one to three weeks if treated correctly.

(iii.) Epidemic Diarrhœa ("summer" or "autumnal" diarrhœa of children) is met with chiefly in childhood and infancy in the summer months of the year, and is attended by catarrh of the mucous membrane of the bowel. The *symptoms* of a severe attack are: Watery stools, foul-smelling, of altered colour, containing lumps of mucus; vomiting; acute abdominal pain and tenesmus; prostration, collapse, subnormal temperature with pinched aspect, rapid dehydration and wasting, and often (after a course of a week or so) death from exhaustion. Infantile Cholera forms about 2 per cent. of "summer diarrhœa" cases. The stools are serous, there is persistent vomiting; collapse rapidly supervenes, the temperature in the rectum is raised as in adult cholera, and death soon follows.

Etiology of Infantile Diarrhœa.—These diseases affect chiefly hand-fed and over-fed children, in warm weather, being probably in part due to dirty feeding-bottles, teats, sour milk, etc. Most of the cases occur in children under six months old. Dietetic errors account for some cases, but the cause of *Epidemic Diarrhœa* is usually an infection. Seasonal, epidemic, and microbic causes have long been suspected on account of its prevalence during the summer and autumn months. It occurs chiefly after hot, dry summers. Flies and dust have been blamed; flies undoubtedly act as carriers of infection. It occurs chiefly in towns, and certain localities have been notorious for recurrent lethal outbreaks in summer and autumn. Adults do not altogether escape; diarrhœa is widely prevalent in the hot, dry summer months in some years; but in children the death-rate is often high. In most cases a virus infection is probably causal: some cases are due to dysentery bacillary infection of

the Shiga, Flexner or Sonne types, or to Morgan's bacillus; in others the food-poisoning bacteria (*B. enteritidis* of Gærtner and *B. ærtrycke*).

In the *Treatment of Infantile Diarrhœa* (1) first free the gastro-intestinal tract of all irritant materials; small doses of calomel (gr. $\frac{1}{10}$), or grey powder (gr. $\frac{1}{4}$), or equal parts of lime water and castor oil (F. 64) or 2–5 grs. sodium sulphate in water every two or three hours, until the stools become healthy; (2) a period of starvation for the first 12–24 hours; and later, (3) easily digestible food such as barley-water, diluted fruit juice, apple purée, two-hourly feeds of whey, peptonised milk or diluted condensed milk. In the fermentative type, use only a small amount of sugar, or a non-fermentable sugar such as dextri-maltose or lactose; subsequent additions to the diet must be made very cautiously. When vomiting is troublesome, gastric lavage with saline solution (not bicarbonate) should be used; then give sherry-whey (§ 297. XXII). When diarrhœa is persistent, give phthalyl-sulphathiazole or sulphasuccidine, and combine with astringents. In mild chronic cases, give protein milk, such as sprulac, or milk protein such as soluble casein or casec. In more severe cases colonic lavage with warm normal saline removes irritating material. Albumen or barley-water can be tried, and brandy, arrowroot, and an astringent mixture are useful: (bismuth carbonate 2 gr., calcium carbonate 3 gr., tincture catechu 5 min., glycerin 10 min., water to 60 min.). If used cautiously, nepenthe (♏iii. t.d.s. at 6 months) is valuable. Dehydration is a serious complication and must be combated with water by mouth or saline by rectal, subcutaneous, intraperitoneal or continuous-drip intravenous methods. For collapse give a transfusion of human blood plasma; nikethamide (coramine) and a warm mustard bath are the best stimulants.

A rare form of severe recurrent diarrhœa may occur in children—**Cœliac disease.** The motions are pale and fatty, the abdomen is distended. Owing to the continued defective absorption of food, the child does not grow (infantilism) and is often rickety; there is anæmia—micro- or megalocytic—yet after years of protracted illness fatal cases show no recognisable cause at autopsy. Defective fat and carbohydrate absorption appear to be the chief errors; the fats, though split, are not absorbed in the small intestine. The slightest error in diet will bring about a relapse of severe diarrhœa which may last many months. The diagnosis from tuberculous peritonitis may be difficult.

Treatment.—The fats in the diet are largely eliminated; sugar and starches are reduced when there is distension, and a large amount of protein added, as in sprue. Vitamin deficiency must be corrected by suitable additions of vitamins A, C and D. Crude liver extract injections and iron are useful.

V. **Typhoid and Toxic Blood States.**—Diarrhœa is usual in typhoid fever, and may occur in measles and the other eruptive fevers (especially at their advent), some cases of Graves' disease, chronic renal disease, uræmia, and pyæmia. Sometimes it appears at the termination of acute illness, as in pneumonia. (And see chronic causes, § 310.)

VI. **A chill** to the surface in some individuals will determine an attack of acute diarrhœa.

VII. **Acute Ulcerative Colitis** is usually of sudden onset, with diarrhœa, and abdominal pain occurring in paroxysms. The motions are dark,

offensive, and contain mucus and blood. There is tenderness over the colon, and its ascending portion is usually distended. The tongue is furred at first, and the breath very offensive. Pyrexia may be present, about 101° to 102°. The commonest complications are exhaustion, anæmia, profuse hæmorrhage; less common are perforation and peritonitis. Sigmoidoscopy shows a uniformly inflamed mucosa with exudate, necrosis of membrane, and often ulceration. Its etiology is unknown, although it resembles the epidemics of so-called *ulcerative colitis in asylums*, in which the organisms of bacillary and sometimes of amœbic dysentery may be found. (And see § 310. V.)

VIII. In cases of acute diarrhœa in which the cause is obscure, reference should be made to the other **Causes of Chronic Diarrhœa**, any of which may from time to time give rise to an acute attack. **Dysentery** (§ 308) and **Cholera** (§ 309) are the commonest causes of diarrhœa in tropical climates, and are occasionally met with in this country.

Prognosis of Acute Diarrhœa.—The causes of acute diarrhœa are for the most part removable; and though weakened by the attack, the patient generally makes a good recovery. Acute Epidemic Diarrhœa in children, however, is a more fatal affection, and it leads to a high death-rate in infancy. The prognosis in any given case depends upon (i.) the cause; (ii.) the severity of the symptoms and the evidences of dehydration; (iii.) the state of the hygienic surroundings; and (iv.) the response to treatment. Infantile cholera is usually fatal. Dyspeptic diarrhœa may be cured in a few weeks, but if untreated, is apt to go on to catarrhal or mucous colitis. Without treatment all forms of epidemic diarrhœa, even in adults, are serious. Should symptoms of prostration or collapse ensue, the outlook is bad; but it is only at the two extremes of life that this disease is so grave. Ulcerative colitis is serious; death may occur from complications, exhaustion, anæmia, or relapses.

Treatment of Acute Diarrhœa.—The indications are (*a*) to remove any irritating matters left in the intestinal canal; (*b*) to provide absolute rest, and warmth to the abdomen; and (*c*) to check excessive diarrhœa. (*a*) Thus, simple acute diarrhœa following the eating of bad food is readily arrested by giving castor oil, ½ oz., with tr. opii, ♏x., followed by a simple bismuth salicylate mixture. No food is allowed for a day, but as much water as desired is drunk: half-normal saline by mouth is essential when dehydration is severe. Then arrowroot made with water is given, and a gradual return to ordinary diet, beginning with milk and milk puddings. For some time all irritating skins, seeds, vegetable cellulose and raw fruits may not be taken. Simple dyspeptic diarrhœa in children is cured with grey powder every night and alkaline carbonates by day. After the acute stage is over, if the condition threatens to become chronic, other drugs are used. Opium checks diarrhœa; it can be given in the form of Tr. chloroformi et morphinæ, B.P./'85, 5–10 minims. Catechu, kino, chalk and tannin are excellent astringents. Bismuth carbonate or kaolin (up to 3–4 G. daily) soothe the congested mucous membrane. For

offensive stools give salol, calomel or charcoal. A course of intestinal antiseptics is often useful—e.g. sulphasuccidine or phthalyl-sulphathiazole. For putrefactive diarrhœa, protein foods should be avoided ; *B. acidophilus* helps to implant a healthier intestinal flora. In fermentative diarrhœa carbohydrates must be restricted ; sugar, bread and flour are usually digested ; but rice, tapioca, bananas, root vegetables, especially potatoes, should be avoided for a time, and a diastase preparation taken. In more obstinate cases of diarrhœa colonic irrigations may be required ; saline or permanganate douches, introduced slowly and without pressure, give excellent results in some cases. Where there is much loss of fluid, saline infusions may have to be given to prevent collapse. The treatment of acute is similar to that of chronic ulcerative colitis and is therefore dealt with in § 310. V.

In the tropics **Diarrhœa** is a common complaint ; it may merely indicate some simple intestinal derangement due to bad food, indiscretion of diet or chill such as may follow sleeping under a fan or punkah during the hot weather. On the other hand, many serious intestinal maladies such as cholera, typhoid and sprue may begin with diarrhœa ; the appearance of mucus, pus and blood in the stools, however, indicates that the trouble, which may be due to a number of different causes, probably arises in the large bowel.

The patient, who is living or has lived abroad, complains of severe DIARRHŒA, WITH BLOOD, MUCUS, *and perhaps* PUS *in the stools.* The disease is probably DYSENTERY.

§ **308.** IX. **Dysentery** is a colonic inflammation, often leading to necrosis and ulceration of the mucosa, due to certain specific bacilli, protozoa or helminths, and characterised by the frequent passage of stools containing mucus, blood and pus. Three main types occur : (1) ACUTE and CHRONIC BACILLARY DYSENTERY, due to a number of different micro-organisms ; (2) PROTOZOAL DYSENTERY, due to (*a*) *Entamœba histolytica*, (*b*) *Balantidium coli*, (*c*) malignant tertian malaria and (*d*) kala-azar ; (3) HELMINTHIC DYSENTERY, associated with (*a*) blood flukes : *Schistosoma mansoni*, *S. japonicum*, and (*b*) the intestinal nematode *Œsophagostomum apiostomum*.

(1) **Acute Bacillary Dysentery** has an incubation period of 1 to 7 days and sudden onset with fairly high fever, nausea, vomiting and headache, followed by colicky abdominal pain, tenesmus and the frequent passage of small stools, 5 to 50 times daily. As toxæmia increases the cheeks become flushed, the pulse rapid, the tongue coated. Dehydration produces restlessness, mental confusion, thirst, dry brown tongue, pinched features, sunken eyes, collapsed veins, and in infants a depressed fontanelle. The stools soon lose their fæcal character and consist of odourless, gelatinous mucus mixed with bright red blood, later becoming muco-purulent ; with recovery bile-stained fæcal matter reappears. Localised abdominal pain and tenderness are infrequent in the absence of peritoneal involvement ; and though some rigidity of the abdominal muscles may at first be present, the contracted sigmoid can later be palpated. Arthritis and iritis may occur. Fulminating and severe Shiga cases die of toxæmia or dehydration with subnormal temperature, but with appropriate treatment the average case becomes apyrexial in 7 to 14 days ; renal failure sometimes occurs associated with glomerulo-nephritis from dehydration and toxæmia. Most cases of Flexner and Sonne dysentery recover.

Chronic Bacillary Dysentery may (1) follow an acute attack or (2) be subacute from the onset. In (1) there is a history of acute dysentery from which there has never been complete recovery. Generally there is frequent defæcation, rectal discomfort or tenesmus, muco-pus and blood in the fæces. During exacerbations fever

may recur. Emaciation, asthenia, secondary anæmia and often œdema of the limbs follow. The thickened, spastic descending colon is palpable. During exacerbations dysentery bacilli may be isolated in about 25 per cent. of cases. In (2) the onset is more insidious, and the original attack mild; bouts of diarrhœa with mucoid, bloody stools follow; remissions are common. The disease closely resembles chronic ulcerative colitis.

Etiology.—The specific organisms are Shiga's bacillus, the Flexner Y group (V, W, X, Y and Z strains) and Sonne's bacillus. Shiga dysentery is mainly a tropical malady; the others may occur in Europe, especially in military barracks, prisons, asylums and in certain outbreaks of summer diarrhœa in children. The disease is spread by water and food contaminated by carriers, or by infected flies.

Treatment of Acute and Chronic Bacillary Dysentery. The *sulphonamide compounds* are of the greatest value. Give by mouth sulphaguanidine, 6–8 G., initially followed by 3–4 G. four-hourly until the stools are less than five per day; then eight-hourly until the stools are normal. This drug is effective and safe in patients initially dehydrated; crystals may be seen in the urine but they appear soft and do not produce renal blockage: rarely headache, nausea, mild erythematous or maculopapular rashes, with or without slight fever, may result from sulphaguanidine, but the risks of agranulocytosis or of exfoliative dermatitis seem negligible. Succinyl sulphathiazole 20 G. daily, or phthalyl sulphathiazole 20 G. daily, in divided doses at three-hourly intervals, have been advocated, as also have the absorbable sulphonamides, *e.g.*, sulphadiazine 1 G. four-hourly. All these drugs act by bacteriostasis, and consequently in severe Shiga infections, where there is already much toxæmia, Shiga antitoxin in addition is advisable, *e.g.*, 200,000 I.U. of refined antitoxin intravenously and repeated in 12 hours if indicated. *Dehydration* is treated by liberal fluids by mouth and if necessary by intravenous injections of saline or 5 per cent. dextrose solution. Routine treatment with saline purges is no longer favoured as it appears to be of little real value and leads to further dehydration; gentle colonic lavage with warm physiological saline may help to remove toxic material. In severe cases with circulatory failure serum, plasma or blood *transfusions* may be of value. The *diet* should be fluid with glucose and albumin for the first day or two, then gradually increased but maintaining a low residue; in the later stages a high calorie, high vitamin, low residue diet is required. Symptomatic relief of griping or tenderness may be obtained with warmth to the abdomen, or gentle saline bowel washouts, or injections of morphine. In chronic conditions, as a last resort, cæcostomy or ileostomy may be considered, to rest the bowel.

(2) Protozoal dysentery. (*a*) **Amœbic Dysentery** is characterised by afebrile diarrhœa with several voluminous fœtid stools daily, containing brownish mucus and dark red blood; tenesmus occurs if the rectum be involved. In 10 per cent. of patients the condition is more acute, fever is present and the bowels may act a dozen times in the 24 hours. Palpation reveals a thickened, tender colon; sigmoidoscopy may show typical painless, yellow, amœbic ulcers surrounded by a zone of hyperæmia, and healthy intervening mucosa. *Entamœba histolytica* can be found by examining the fresh material obtained by swabbing or lightly curetting the base of the ulcer. The disease is acquired by swallowing the cysts in contaminated water and food, especially vegetables, infected from convalescent or contact carriers.

Diagnosis.—The actively motile amœba containing red blood corpuscles is found in the mucoid exudate in acute cases, and the cysts in the solid fæces of chronic cases. Ulceration is almost always confined to the colon and often involves the muscular coats. Complications include intestinal hæmorrhage, perforation with peritonitis, retro-colic abscess and post-dysenteric adhesions, and amœboma (a chronic amœbic granuloma) often closely simulating neoplasm. Amœbic hepatitis is associated with fever, enlarged, tender liver and slight leucocytosis, while the formation of liver abscess is suggested by rigors, sweating, shoulder pain, and involvement of the base of the right lung (§ 336). Amœbiasis of the lung is occasionally found; involvement of the brain, spleen and abdominal wall is rare.

Treatment.—Patients require a low-residue diet, and should be kept in bed during treatment with emetine. For intestinal infection, each night emetine bismuth iodide gr. iii, preceded half an hour before by phenobarbitone gr. i, is given orally for ten to twelve days: while each morning (after a preliminary bowel washout with 1 per cent. sodium bicarbonate in water) a retention enema of 250 c.c. of 2½ per cent. chiniofon (B.P.) in water is given slowly and retained for 4–5 hours. Following this combined treatment carbarsone (B.P.) or stovarsol (B.P.) 0·25 gm. is given orally morning and evening for a further ten days. When there is severe ulceration, specific treatment may be preceded by a course of penicillin (30,000 units three-hourly subcut.) and sulphaguanidine (3 G., three-hourly by mouth) for five to seven days to eliminate secondary infection. Amœbic hepatitis or abscess requires emetine hydrochloride gr. i. intramusc. daily for seven to ten days, while pus, if present in any quantity, may require aspiration: open operation is only performed if secondary infection necessitates it. Emetine is a myocardial poison, producing tachycardia and a lowered blood pressure; its prolonged use may lead to profound asthenia, neuromuscular weakness and heart failure: an increase in diarrhœa, after an initial improvement, may be also a sign of over-dosage. Diodoquin 1·5–2·0 G. daily by mouth for three weeks may be useful in ambulatory treatment of intestinal amœbiasis; it is not absorbed and therefore is useless for hepatitis.

(*b*) **Balantidial Dysentery** occurs in people who handle pigs. There are frequent muco-sanguineous stools and anæmia; many cases remain latent. Ulcers form in the colon and may perforate. Clinically, this condition is indistinguishable from amœbic ulceration, the diagnosis being made by microscopical examination of the stools or of material curetted from the ulcers during sigmoidoscopy. *Balantidium coli*, in vegetative forms or cysts, may occur in the excreta.

Treatment.—Stovarsol, gr. iv. t.i.d. for 1 week, and enemata of methylene blue (1 in 3,000) are advocated.

(*c*) **Malarial Dysentery.**—Malignant tertian malaria (*Plasmodium falciparum*) may manifest itself by severe diarrhœa, with occasionally blood and mucus, producing a syndrome indistinguishable clinically from bacillary dysentery; the condition originates from obstruction in the capillaries with clumps of agglutinated corpuscles infected with parasites. Sigmoidoscopy in the milder cases shows a diffuse or patchy hyperæmia; in severe cases scattered hæmorrhagic areas are seen in the hyperæmic bowel wall, and these may go on to actual necrosis. The *diagnosis* is made by finding the parasites in blood smears and by the associated clinical features, such as splenomegaly, anæmia and fever.

(*d*) **Kala-azar Dysentery.**—In kala-azar, diarrhœa is a not uncommon complication; *Leishmania* may be found in the intestinal villi or polypoid tissue formed in the gut wall. Occasionally a dysenteric-like syndrome with the passage of blood and mucus supervenes, which may prove to be due either to kala-azar itself or to a superadded bacillary dysentery infection.

(3) Helminthic Dysentery.—(*a*) **Schistosomal dysentery,** especially at first, is characterised by diarrhœa or loose motions containing mucus and blood; later there are often solid stools coated with mucus containing the lateral spined ova of *S. mansoni* or the lateral knobbed ova of *S. japonicum.* Occasionally the terminal spined ova of *S. hæmatobium* may be found (Fig. 107), though this species rarely gives rise to dysenteric features. Tenesmus, loss of weight and secondary anæmia may follow: many cases remain latent. Subacute or chronic schistosomal appendicitis, colonic and rectal papillomata, fistulæ, and a periportal cirrhosis of the liver may develop; the latter may be associated with an enlarged spleen (Egyptian splenomegaly). Eosinophilia is common, and the schistosomal complement fixation reaction is of value when it is difficult to find ova. Sigmoidoscopy aids: early minute tubercles are seen and eggs are found on scraping these; later, the pathognomonic schistosomal papillomata, which on sloughing leave punched-out circular ulcers.

Treatment.—Trivalent antimonial compounds are specific. Sodium antimony tartrate intravenously on alternate days for one month is frequently used; give an

initial dose of gr. ½ and increase in successive doses by gr. ½, to a maximum of gr. 2½. Recently a short intensive course of a total dosage of gr. i per 12 lb. body weight (12 mgm./kgm.) divided into 6 doses has been advocated, 3 doses being given at three-hourly intervals on two successive days: each dose is dissolved in 10 c.c. of 5 per cent. dextrose solution and injected not faster than 2 c.c. per minute. Cough, vomiting or rheumatic-like pains may follow administration of this drug. Stibophen, in 6·3 per cent. solution, is also used, doses of 1·5 c.c., 3·5 c.c., and then 5 c.c., being given intravenously on alternate days to a total of 40–60 c.c. Anthiomaline, in 6 per cent. solution intravenously or intramuscularly, is preferred by some in doses of 4 c.c. on alternate days to a total of 40–60 c.c. Miracil in doses of 10 mgm./kgm. twelve-hourly by mouth for 10 doses appears of great value.

(b) *Dysentery due to* **Œsophagostomiasis.**—The nematode parasite, *Œsophagostomum apiostomum*, commonly affects man in Northern Nigeria. Its embryos embed themselves in the walls of the colon and become enclosed in fibrous tissue nodules; as they approach maturity they escape into the lumen, often leaving behind an ulcerated area, and finally attach themselves to the mucosal lining of the gut. Diarrhœa with blood and mucus may result, and peritonitis is an occasional complication. The eggs, which resemble ancylostome ova, are passed in the stool. Tetrachlorethylene or carbon tetrachloride cures the disease. (See Ancylostomiasis § 547.)

The patient complains of ACUTE DIARRHŒA, *coming on very suddenly, and attended with severe* COLLAPSE, *abdominal* CRAMPS, *and "rice-water" stools.* The disease is probably CHOLERA.

§ **309.** X. **Cholera** (synonym: Asiatic Cholera) is a disease caused by *Vibrio choleræ*; it begins with urgent vomiting, purging, and colourless evacuations, cramps and a tendency to collapse, and which, if not fatal in the first stage, is attended by secondary fever. The period of incubation is usually three to six days, but it may vary between one and ten. There are three well-marked stages:

(a) *Stage of evacuation*, which lasts from two to twelve hours, or longer. The patient is *suddenly* seized with violent vomiting, profuse diarrhœa and later cramp. The stools, after the first few, are colourless and opaque, resembling rice-water, and containing flakes of columnar epithelium and casts of villi, and the *comma-shaped bacillus*. There are severe cramps in the fingers, toes and abdominal muscles, great exhaustion, small and weak pulse, and coldness of the body. (b) The *algid stage*, cold stage, or stage of collapse, lasts a few hours to a few days according to the severity of the case. The patient looks like a corpse; the surface temperature falls, and the skin becomes a deadly livid hue; the pulse cannot be felt at the wrist. The temperature is most remarkable, for in the rectum it may be as high as 105° F., while in the axilla it is only 90° F. During this stage the purging ceases, but the vomiting and cramps persist. The mind remains clear. There is suppression of urine and bile. (c) *Stage of reaction.*—The pulse returns, the temperature rises, the bile reappears, the urine is scanty and deficient in urea. The temperature goes up, and may be attended by typhoid symptoms. The bowels are confined. There may be erythematous, urticarial and other eruptions upon the skin. This stage is followed by great weakness. Fluid and salt loss is important in this disease. Diarrhœa and vomiting also produce salt depletion, decreased blood volume with increased viscosity of blood and dehydration of the tissues. Polycythæmia and leucocytosis also result from concentration. The blood chemistry shows reduced blood chloride, diminished plasma alkalinity, phosphate retention and increased blood urea, with decreased urinary output. Finally the weakened heart fails to pump the viscous blood through the kidneys and anuria with acidosis results.

The *Diagnosis* is easy in severe cases on account of the extreme suddenness and severity of the symptoms. The copious colourless evacuations are characteristic of cholera. Conditions which resemble it are acute poisoning by arsenic, croton oil, and other irritants, ptomaine poisoning, and certain cases of malignant malaria. The identification of the bacillus renders the diagnosis certain.

Etiology.—The disease occurs in great epidemics, but has not visited this country, except sporadically, since 1865–6–7. In India it is endemic. As regards age, none are exempt. All epidemics in this country have occurred in the autumn and the end of the summer. The exciting cause is the specific organism, which must be introduced into the alimentary canal. As with typhoid, the disease is usually communicated by drinking water contaminated by the evacuations from the bowels and stomach, and requires the same preventive measures (§ 522 *et seq.*). It may be conveyed in other ways, as by flies, through *want of cleanliness.* One attack does not confer immunity.

Prognosis—The earlier cases of an epidemic are the most fatal. The mortality rate varies from 30–70 per cent. in different epidemics. Aged and debilitated people, young children and alcoholics do badly. New methods of treatment have reduced the mortality by half. In the reaction stage uræmic coma, hyperpyrexia, or the typhoid state may cause death. *Untoward Symptoms* are blood in the evacuations, long stage of collapse, restlessness, extreme cyanosis, and absence of the pulse at wrist. Favourable signs are a perceptible pulse in the algid stage, the early occurrence of reaction, cessation of cramp, secretion of urine, and the occurrence of sleep. *Complications* include pneumonia, occurring in the reaction stage, bronchitis, pleurisy, parotitis, bed-sores, inflammation of the pharynx, genitals, or bladder, corneal ulcers and gangrene of the fingers, toes, scrotum or penis.

There are two rare *varieties:* (1) Choleraic diarrhœa, or "cholerine"—resembling autumnal diarrhœa occurring during an epidemic of cholera. (2) Dry cholera or cholera sicca, where there has been no vomiting or diarrhœa, the patient dying of collapse before these have had time to develop. At autopsy the intestines contain much fluid.

Treatment.—Prophylactic vaccine gives immunity for several months. Tinct. opii (♏ 20.) may be given at the onset of the preliminary diarrhœa, but never after the characteristic colourless evacuations have set in. Rest in bed, warmth, and fluid farinaceous diet are essential; animal protein in soups and jellies is harmful. Kaolin may be given in massive doses. Sulphaguanidine by mouth, 6–8 G. initially followed by 3–4 G. four-hourly, has recently been advocated. Injections of hypertonic or isotonic saline have been used with excellent results. When collapse appears, saline injections by rectum are useful so long as the systolic blood-pressure is above 70 mms.; below that point they are not absorbed, and an intravenous injection should be administered of sufficient amount (3 to 5 pints) to raise the blood pressure, and ensure excretion by the kidneys. The hypertonic solution contains sod. chlor. gr. 120, calc. chlor. gr. 4, potass. chlor. gr. 6, to a pint of sterile water, and is given at the rate of 4 ounces per minute. Nikethamide, sips of tea and coffee, act as stimulants. Potassium permanganate gr. 2 is given by the mouth every half-hour as an oxidising agent to destroy the toxins of the cholera bacillus.

In milder cases treatment with essential oils is good, the mixture consisting of ol. anisi, ol. cajuputi, ol. juniperi-āā ♏ 5, spt. æther. ♏ 30, ac. sulph. arom. ♏ 15: Dose ♏ 30 in water every ¼ hour for 16–28 doses.

§ 310. Chronic Diarrhœa.—The term chronic diarrhœa signifies the occurrence of frequent *loose* evacuations, say three or more in the twenty-four hours, extending over a period of weeks, months, or even years (as in Sprue). It is usually, though not necessarily, attended by tenesmus. The stools should be examined (§ 303) and the anus and rectum carefully inspected. Tenesmus points to disease of the rectum.

I. Chronic Diarrhœa may be due to some of the same causes as **Acute Diarrhœa** (*q.v.*). In children think of worms or bad feeding; and in adults, errors in diet, carbohydrate dyspepsia, ulceration, and chronic irritant poisoning.

II. **Fissure of the Anus,** slight ulcers or abrasions, or even an inflamed

pile, may cause a chronic diarrhœa. Actually there is underlying retention of stool (constipation), and the diarrhœa is "false diarrhœa."

III. **Ulceration of some part of the Intestinal Canal** is a not infrequent cause of diarrhœa in England; it is well to mention here the ulcerating lesions which may affect the intestine. (1) Ulcer of the lower part of the ileum may be due to tuberculosis, typhoid fever or Crohn's disease. (2) Ulcer of the cæcum may arise from the pressure of inspissated fæces or some foreign body—*e.g.*, a tooth-brush bristle—which has been swallowed. (3) Ulcer of the appendix may similarly arise from foreign bodies or as part of appendicitis (§ 247). (4) Ulcer of the rectum is generally of malignant, gonococcal or syphilitic origin; it is attended by the passage of blood and pus, and stricture may result. Mainly in tropical areas, lymphogranuloma inguinale can cause chronic ulceration and stricture formation. (5) Ulcers of the large intestine and rectum occur in dysentery. These may contract on healing and produce stricture. (6) Cancer of the bowel may produce ulcer, the most frequent situation being the sigmoid. (7) Simple ulcerative colitis (§ 310. V). (8) Ulceration may follow prolonged constipation with atony of the colon. (9) A submucous streptococcal infection may cause chronic diarrhœa with precipitate stools, as may chronic nephritis, severe anæmia, and other wasting diseases.

The commonest causes of ulceration in this country are ULCERATIVE COLITIS, CANCER, TUBERCLE, REGIONAL ILEITIS, SYPHILIS, and in tropical climates DYSENTERY (§ 308).

1. ULCERATIVE COLITIS, (§ 307. VII and § 310. V) causes one of the most intractable forms of chronic diarrhœa.

2. INTESTINAL CANCER presents the following features: (i.) The patient is usually over fifty; (ii.) diarrhœa and anæmia due to hæmorrhage are common if the disease is in the cæcum or ascending colon: obstructive symptoms and diarrhœa alternating with constipation if in the descending or sigmoid colon; (iii.) paroxysmal abdominal pain is frequent; (iv.) tenesmus indicates a lesion in the rectum; (v.) a tumour may be palpable through the abdominal wall, or by rectal examination. It is most difficult of access when in the lower sigmoid colon; and may then (vi.) be within reach of the sigmoidoscope. (vii.) Cancerous cachexia often accompanies. (viii.) Pyrexia and leucocytosis may be due to ulceration. (ix.) The stools vary; they may contain blood in considerable quantity, but invariably occult blood. (x.) X-ray with a barium enema may show a characteristic filling defect. (Fig. 82.) And see § 320.

3. TUBERCULOSIS of the lungs may be attended by diarrhœa, even without ulceration of the bowel. Multiple ulcers due to tuberculosis may be found in the lower ileum, and less commonly in the rectum, where the symptoms mimic ulcerative colitis (see ulcerative proctitis, § 314. VIII.) Tuberculous ulceration of the bowel is recognised by (i.) evidences of tuberculosis in the lungs or other parts of the body; (ii.) the presence of night sweats and intermittent pyrexia; (iii.) the stools are watery, and

there is rarely any pain ; (iv.) tubercle bacilli may be demonstrated in the stools by appropriate staining methods. Relief is generally effected by quinine and opium internally, combined with appropriate dietary ; if these fail, recourse may be had to iron, opium, and lead.

4. Regional Ileitis (Crohn's disease), usually of the terminal ileum, due to chronic non-specific inflammation, may cause symptoms simulating appendicitis, or ulcerative colitis with much diarrhœa. Secondary abscesses may form. Young people are usually affected, and the symptoms are progressive and recurring. With X-ray there is filling defect of the ileum or cæcum. Excision is the best treatment ; sometimes in two stages, with a short-circuit of the affected part of the bowel first.

5. In Syphilitic Ulceration of the rectum (i.) the motions often consist largely of pus and blood ; (ii.) great pain and tenesmus are usual, combined with (iii.) other evidences and a history of syphilis. Stricture occurs in the later stages. (iv.) Opium and antisyphilitic treatment are here of great value to check the diarrhœa.

IV. **Chronic** or **Mucous Colitis** (Syn. : Intestinal Dyspepsia) is much more common in women. It can cause many years of ill-health, and needs much patience in treatment. The *Symptoms* vary, but in the main are : (i.) Irregular attacks of diarrhœa alternating with obstinate constipation, brought on by slight dietetic errors, by nervous causes, or by the misuse of aperients. (ii.) During the attacks of diarrhœa, mucus may be passed in masses, shreds or casts several inches long : the stools often contain intestinal sand but not blood. (iii.) The stools may show carbohydrate fermentation. (iv.) There is abdominal discomfort, and a good deal of flatus passed per rectum ; the flatulence may cause insomnia. (v.) B. coli may infect the urinary tract. (vi.) Nervous prostration, ready fatigue and loss of weight are almost always present and may be the presenting symptoms. (vii.) Examination may reveal a distended cæcum while spasmodic contraction of the descending colon may be felt. The sigmoidoscope aids diagnosis in obscure cases.

Diagnosis.—The disease is distinguished from ulcerative colitis by the presence of blood in this latter condition, and from carcinoma coli by the length of history and the type of stool. Sometimes neurasthenia is accompanied by marked colonic dyspepsia, in which case treatment of the colon may aid recovery.

Treatment.—Sometimes many of the symptoms of early colitis are due to excess of purgatives and enemas to which the patient has resorted in the mistaken belief that she suffers from constipation. In such cases, no strong purgatives should be used ; relief is obtained by prescribing liquid paraffin or petroleum-agar, belladonna or eumydrin, codeine or kaolin. A non-irritating diet is essential ; all seeds, skins and stringy foods must be forbidden ; forbid or curtail foods which are not digested —in some, fruit and vegetables, owing to their cellulose ; in others, starchy or protein foods. The patient may benefit from diet consisting entirely of milk for a time. A diet deficient in vitamins can lead to atrophy of the intestinal mucous membrane ; vitamins (especially Vit. B) must be restored to the diet in such cases. Fruit juices and purées are given to provide the vitamins which are lacking. B. acidophilus with

lactose aids the development of a healthy intestinal flora. Taka-diastase is useful when undigested starch appears in excessive quantities in the stool. Adequate warm clothing covering the abdomen is essential, and when visceroptosis is also present, a suitable abdominal belt aids. Occasionally lavage of the colon with saline or weak potassium permanganate is of help, when carried out by an expert. In other cases an intestinal autogenous vaccine and abdominal diathermy are useful. The psychological aspect must receive attention: often an unhappy environment at home or at work needs correction: fatigue and overwork should be avoided, and at all times, the patient must be discouraged from examining his stools.

V. **Chronic Ulcerative Colitis** may follow the acute variety (§ 307. VII), but much more often its onset is insidious, with (1) apyrexial diarrhœa, which at first is not regarded seriously, and lower abdominal pain and flatulence: (2) then follow blood and muco-pus, mixed with fæces, (3) rectal discomfort and tenesmus, (4) loss of weight, (5) secondary anæmia and fever may develop. (6) Hypochlorhydria is common. The condition often begins as a granular proctitis which tends to ascend to the pelvic and descending colon and in extreme cases the cæcum. The *Diagnosis* depends on: (1) Sigmoidoscopy in a well-established case shows a uniformly inflamed, granular mucosa bleeding readily on pressure, with miliary or larger-sized ulcers; the lumen of the rectum and large bowel is narrowed, its walls rigid and thickened so that ballooning with air becomes painful and difficult. (2) X-ray reveals a shortened, tubular bowel with complete loss of haustration; if deep ulcers exist the outline of the colon is feathery and moth-eaten in appearance. *Prognosis.*—Intermissions are common, apparent recovery being followed by relapses extending over many years; many of the gravest cases recover eventually. Complications include hæmorrhage, perforation, stricture, polyposis, malignancy and arthritis.

The specific *cause* is unknown. *Treatment.*—Keep the patient strictly in bed until the condition has healed. First the abdominal discomfort, fever and diarrhœa disappear, later the pus, blood and mucus are absent from the stools, but healing is not complete until confirmed by sigmoidoscopic examination. The diet must be ample and varied, but restrict foods with vegetable residues: supplementary vitamins, especially vitamins B and C, should be given. A mixture containing tinct. opii ♏ 10, tinct. belladonnæ ♏ 7½ with bismuth carbonate gr. 10 is often most helpful in controlling diarrhœa. Courses of colonic lavage on alternate days, for 10–14 days, are given—first, sodium bicarbonate solution (60 gr. to 1 pint) or normal saline is used to remove as much fæces and mucus as possible: this is followed by chiniofon B.P. (yatren 1 per cent.), tannic acid (1/500), protargol or albargin (1/1,000) or potassium permanganate (1/5,000) for their astringent and antiseptic effects. Hurst obtained good results with polyvalent dysenteric serum given intravenously in doses of 50–100 c.c., diluted with 150 c.c. saline daily for 7–10 days. Long courses of sulphasuccidine or phthalylsulphathiazole (G. 1–2 q.i.d. for many weeks or months) until the diarrhœa ceases, are often of great value. Septic foci must be suitably dealt with, but there is little evidence that other sera or vaccines are of any help. When anæmia is present, one or more blood transfusions are often of remarkable value. In long-standing or obstinate cases, especially with polyposis, ileostomy may be performed, as this allows the fæces to be drained and permits lavage of the whole colon through the artificial opening. If this fails, colectomy may have to be resorted to. To prevent relapses, the stools must be kept soft with liquid paraffin, warm clothing used to prevent chill, and foods with irritating residues avoided over a period of many years.

VI. **Obstruction in the Portal Circulation** produces diarrhœa, due to the congestion of the intestinal wall. It is recognised by: (i.) A previous history of heart disease, or of intemperance and alcoholic dyspepsia; (ii.) other signs of liver or cardiac disease;

(iii.) other evidences of portal obstruction, such as ascites, piles, and a large spleen (§ 260); (iv.) there is little or no pain, and the stools are liquid and dark, occasionally bloody. The *Treatment* requires caution, because the diarrhœa and hæmorrhage of themselves relieve the condition by diminishing the venous engorgement. (i.) If the diarrhœa has not lasted long, a dose of calomel will relieve the portal congestion, and so cure the diarrhœa. (ii.) Magnesium sulphate, gr. 20, with alum and dilute sulphuric acid, are recommended. Bismuth and opium, with caution, are the most useful for checking the diarrhœa.

VII. **Dysenteric Diarrhœa** is a sequel of dysentery, which may perhaps have been contracted abroad many years previously (see § 308).

VIII. **Nervous Diarrhœa** may continue for years; it has the following characteristics: (i.) The motions are often quite healthy. There is usually no pain or tenesmus. The diarrhœa is recurring or intermittent, occurring in the early morning, or when the patient is "nervous." Sometimes it follows each meal (*lienteric* diarrhœa). (ii.) Diet has little or no influence; the *attacks are determined* by mental emotion or bodily fatigue. The administration of nux vomica, belladonna, and bromides is more efficacious than astringents. Arsenic (♏ ii. Fowler's solution), with meals, is said to be a specific for lienteric diarrhœa.

The crises of LOCOMOTOR ATAXY sometimes take the form of acute diarrhœa, with or without pain. In HYSTERIA acute attacks of diarrhœa, with noisy borborygmi, may occur, determined in the same way as other hysterical attacks.

The *rarer* cases of chronic diarrhœa are:

IX. **Amyloid Disease** of the intestines gives rise to a most intractable form of chronic diarrhœa. Indeed, this is the common mode of death in amyloid disease of the viscera. The characteristics here are: (i.) A history of long-standing purulent discharge, or of syphilis; (ii.) great pallor of the skin, accompanied by evidences of lardaceous disease in the spleen, liver, and kidney; (iii.) the stools are generally liquid and extremely offensive, sometimes attended by hæmorrhage. The *Treatment* is very unsatisfactory. Opium does no harm, even when there is amyloid disease of the kidney, as there is no tendency to uræmia.

X. **Senile Diarrhœa** occurs in persons over sixty or seventy, and is very chronic in its course, but the patient suffers very little. Careful examination for organic disease should be made before concluding that the condition is simple senile diarrhœa. Most remedies fail to relieve it; it may exist for years without emaciation or danger to life.

XI. **Mineral Poisons,** and especially arsenic and antimony, in small and continued doses, may cause persistent diarrhœa.

XII. **Gastrogenous diarrhœa** may occur in cases of achylia gastrica, even when it is not associated with pernicious anæmia. It ceases when 30 to 60 drops of dilute hydrochloric acid are taken in a tumblerful of water with meals.

XIII. **Pancreatic Disease** has been associated with diarrhœa which resists treatment until pancreatic extracts are administered.

XIV. **Gastro-colic Fistula** and **Gastro-enterostomy** occasionally produce persistent diarrhœa and, especially in the case of the former, gross emaciation. If symptomatic treatment, together with that of the associated anæmia, does not relieve, operation must be considered.

XV. Other *causes* are hyperthyroidism, Addison's disease, excessive tobacco smoking in susceptible persons, chronic renal disease, anaphylaxis, and exhausting or wasting diseases of any kind.

§ 311. XVI. Sprue.—Psilosis or sprue is a tropical disease of unknown etiology associated with derangement of the gastro-intestinal tract, characterised by deficient absorption of fat, glucose, certain vitamins and calcium; the secretion of the intrinsic hæmatopoietic factor is often also defective.

Signs and Symptoms.—(i.) Apyrexial morning diarrhœa with bulky, acid, pale, frothy, fatty stools. (ii.) Inflammatory lesions of the mouth; the tongue is tender, shows patches of inflammation, and later becomes pale and atrophic with disappear-

ance of the papillæ. Aphthous ulcers may involve the lingual or buccal mucosa. (iii.) Anæmia, which may be severe; this is almost invariably megalocytic in type with an increase in the average diameter of the corpuscles; it closely resembles pernicious anæmia and responds in the same manner to liver extract therapy and to folic acid. (iv.) Asthenia with low blood pressure. (v.) Emaciation and wasting; the skin becomes dry and wrinkled and sometimes brown pigmentation occurs over the forehead and malar eminences. (vi.) Intestinal flatulence; occasionally vomiting and dyspepsia. (vii.) In advanced cases neuritis, œdema of the feet, cramp and tetany may occur occasionally. (viii.) Physical examination shows a distended abdomen with thin abdominal parietes.

Diagnosis.—Sprue has to be diagnosed from pernicious anæmia, chronic pancreatitis, carcinoma of the pancreas and stomach, gastro-colic fistula, tuberculous enteritis and lymphadenoma involving the mesenteric lymph glands. Biochemical and radiological investigations often assist. Sprue shows a high fæcal fat which is adequately split, and low gastric acidity; with the aid of histamine, however, 78 per cent. secrete acid. Blood analysis often shows lowered calcium, slight increase in plasma bilirubin and delayed or low glucose tolerance curve, due to malabsorption of glucose; blood sugar curves following the intravenous injection of glucose show sluggish utilisation of glucose.

Prognosis.—Sprue is a very chronic disease, with spontaneous remissions and exacerbations. It particularly affects Europeans or those of mixed European blood, and until recently often terminated fatally if the patient remained in the tropics.

Treatment.—Patients are put to bed for one or two months and given a high protein, low fat, low carbohydrate diet (see § 297. XVII), in which the ratios of these different food constituents are 1 : 0·3 : 1·3 ; red meat is the main source of protein, or alternatively a defatted, dried milk known as "sprulac" may be substituted: the diet is graded from 600 to 3,000 calories. Crude liver extract, equivalent to 1½ lb. of whole liver daily should be given orally or intramuscularly; in severe cases, where large doses of liver are so beneficial, oral and intramuscular liver extract therapy may be combined. Recently remarkable initial results have been obtained with folic acid 20 mgm. daily for a week, followed by a daily maintenance dose of 5 mgm., but liver may be required to complete the response. Suitable diet with liver extract has made it possible to restore the majority of sprue cases to normal health in two months and to permit of their return to the tropics. It is, however, difficult to be certain that a permanent cure has been obtained in sprue; relapses may follow indiscretions in diet such as taking spiced and sugary foods or excess of carbohydrate and fat. Chill is especially to be avoided after return to England. In most cases a maintenance dose of liver extract is not required, but where repeated relapses have occurred it is advisable to give the patient liver extract by mouth in dosage equivalent to ½ lb. of whole liver daily for a period of 6 months. Nicotinic acid, 50 mgm. t.i.d., is indicated when oral symptoms are severe, riboflavin 3 mgm. daily for angular stomatitis, acid hydrochlor. dil. ♏ 30–60 t.i.d. with meals for achlorhydria, and calcium lactate gr. 30 t.i.d. for calcium deficiency.

§ 312. **Tenesmus** literally means straining at stool (τείνω, to strain or stretch); but in its widest sense it may be taken to mean any local rectal sensation of "bearing down" which results either in constant desire to go to stool, or straining when at stool. The latter may lead to prolapse of the rectum, especially in children. Diarrhœa is always attended by more or less tenesmus, but tenesmus is not always attended by diarrhœa. (1) Ascertain if the tenesmus is accompanied by diarrhœa —*i.e.*, are the motions frequent and liquid? If so, refer to the section on Diarrhœa, § 306. (2) Examine the motions; note their consistence and any abnormal constituents such as mucus and blood. (3) Search for

any local anal or rectal condition such as fissures, piles, polypi, or ulcers. All the pelvic organs should also be thoroughly investigated, especially in women, in whom the symptom is commoner than in men.

Causes.—Tenesmus (not necessarily accompanied by diarrhœa) may arise from four groups of causes:

1. Various conditions of the ANUS—pruritus, eczema, or fissure—may be overlooked for a long time. Piles also, if internal, may be difficult to detect, even by the examining finger, but streaks of bright blood will appear in the motions from time to time.

2. Various RECTAL CONDITIONS, especially carcinoma, simple ulceration (proctitis) or (rarely) stricture. The former are attended by pus or blood, or both. The latter (usually of syphilitic origin) is attended by tape-like stools. In the aged, always suspect cancer of the rectum. Prolonged use of purgatives, or constant use of enemas may result in straining at stool and prolapse of the rectum. An impacted fish-bone is a rare cause.

3. PRESSURE on, or irritation of, THE RECTUM FROM WITHOUT, such as may be caused by chronic congestion, retroversion or other disease of the uterus. Ischio-rectal abscess, pelvic hæmatocœle, and various ovarian and Fallopian tube lesions in women, and congestion or new growth of the prostate in men, are common causes. Any bladder disease, such as stone—a frequent cause of tenesmus in children, and apt to result in prolapse of the rectum—or new growths or chronic cystitis may cause this distressing condition. Menstruation and the later stages of pregnancy may be attended by a certain amount of tenesmus.

4. In HYSTERICAL AND NERVOUS SUBJECTS any fright or other emotion may at once determine tenesmus, which the patient calls "diarrhœa." In tabes dorsalis the "rectal crises" may take the form of tenesmus.

Treatment.—The indications are (1) the removal of the cause, the treatment of piles and other causal conditions being described elsewhere; (2) the relief of local congestion or irritation of the rectum. In any case, morphia, bismuth, belladonna, or cocaine or allied drugs in the form either of suppositories or ointments inserted by an applicator, will relieve the distress from which the patient suffers.

§ 313. In **Proctalgia Fugax** the patient has recurrent severe cramp-like pain in the rectum and perineum, which usually wakens him up at night; it may last three to ten minutes or even longer. Rectal examination is negative. The pain is regarded as due to rectal or anal spasm, but some medical sufferers regard it as an allergic symptom or attribute it to venous engorgement. In women it occurs near the menstrual period.

Treatment.—The pain is usually relieved by a small meal which excites a gastro-colic reflex: otherwise insert suppos. bism. subgall. (B.P.C.) or inject the rectum with air or warm enemata. Sometimes attacks cease when the patient gives up smoking.

§ **314. Blood in the Stools** is met with, as we have seen, in dysentery and some cases of simple diarrhœa; it may occur in other conditions. The presence of blood in the stools may be recognised by the reddening of the water in which the stool is placed, or by the spectroscope (§ 303). Clinically, blood in the stools may present two widely different characters:

(*a*) When the blood is of *bright crimson colour* and is on the surface of the stool, it indicates either that the bleeding comes from the rectum or the lower part of the large bowel; or, if it comes from the upper part of the intestinal canal, that it is too large in amount to be acted upon by the intestinal secretion. (*b*) *Melæna* (*tar-coloured stools*) is met when hæmorrhage in moderate quantity has taken place in the stomach or the upper part of the alimentary tract, in which case the digestive fluids of the stomach and intestine acting on the blood give it a tarry colour. The causes of these two conditions are to some extent interchangeable, for what will produce a large hæmorrhage at one time may at another produce only a little. Bleeding, even if small in quantity, should never be neglected; often slight intermittent bleeding is the first sign of a malignant growth somewhere in the gastro-intestinal tract.

(*a*) **Bright Red Blood** may be due to lesions of the lower part of the alimentary canal. Of these several causes are referable to the anus or rectum, and are discovered on local examination or by proctoscopy.

I. HÆMORRHOIDS, or PILES, are undoubtedly the commonest cause of blood in the stools. The blood is generally met in streaks only, but the quantity may at other times be very large (§ 315).

II. PROLAPSE OF THE RECTAL MUCOUS MEMBRANE, either acute or chronic, with contraction of the anal sphincter, may cause the appearance of bright blood, usually after a motion.

III. FISSURE OF THE ANUS may also produce streaks of blood. It is not infrequent, and is recognised by the excruciating pain during and after defæcation. The irritation it causes may give rise to a variety of false diarrhœa. The fissure can always be seen by *careful* examination. There may be a history of trauma.

IV. In CARCINOMA of the RECTUM or COLON the blood is usually mixed with the stool, and may be intermittent. A sudden and very severe hæmorrhage from the rectum may be the first sign of carcinoma. An innocent adenoma may cause hæmorrhage; it should be looked on as a precancerous condition and removed. Careful digital and sigmoidoscopic examination should be made.

V. A discharge of blood-stained mucus, coming on somewhat suddenly in an infant, is highly suggestive of INTESTINAL INTUSSUSCEPTION, which is one of the causes of acute obstruction (§ 319).

VI. RECTAL POLYPI are met chiefly in children.

VII. TYPHOID and TUBERCULOUS ULCERATION sometimes produce profuse discharges of bright red blood, coming from the lower end of the small intestine. Other evidences of these affections are present.

VIII. **Proctitis** may be simple, traumatic or infective (gonorrhœal, syphilitic, tuberculous or pyogenic). *Simple proctitis* occurs in association with chronic constipation or the repeated use of soapy enemata. *Ulcerative proctitis* is usually present in the upper part of the rectum, and may spread to the sigmoid and cause ulcerative colitis. It can only be diagnosed with certainty by proctoscopic examination. Lympho-granuloma inguinale is a cause of ulcerative proctitis and rectal stricture (90 per cent. of cases are women): and see § 310. III.

The leading *symptoms* are tenesmus and painful defæcation, sometimes discharge of blood and mucus *after* the passage of normally formed stools. *Treatment* is by avoiding hard vegetable residues in the diet, keeping the stools soft and the use of mild astringent retention enemas.

IX. ULCERATIVE COLITIS (§ 307. VII) occasionally causes severe hæmorrhage, more usually small repeated hæmorrhages.

X. SCHISTOSOMA MANSONI and HAMATOBIUM infestations cause a spurious dysentery with polypoid masses in the rectum. They are described in § 308 (3). *Schistosoma japonicum*, a third species, gives rise to Katayama disease or Schistosomiasis of the Far East.

XI. Various GENERAL BLOOD CONDITIONS may give rise to hæmorrhage coming from the rectum or elsewhere in the alimentary canal in varying amount. This occurs in purpura, primary thrombocytopenia, scurvy, agranulocytosis, hæmorrhagic forms of the specific fevers, acute yellow atrophy of the liver, and leukæmia.

(*b*) **Melæna** (*tarry stools*) is met when bleeding takes place in moderate quantity from the stomach, or high up in the alimentary tract. 60 c.c. of blood can cause a tarry stool. Its causes are:

1. When coming FROM THE STOMACH, it may be associated with profuse *hæmatemesis* (§ 272); the commonest causes of hæmatemesis are peptic ulcer and hepatic cirrhosis. Melæna occurs often without hæmatemesis in duodenal ulcer.

2. PORTAL OBSTRUCTION (§ 260) is one of the most frequent causes of melæna, especially that form due to peri-portal cirrhosis of the liver. It may also occur with advanced cardiac disease. In either case the hæmorrhage is a natural safety-valve, and relieves the engorged state of the portal circulation.

3. CANCEROUS, TUBERCULOUS, and other ULCERATIONS of the small intestine (see §§ 307 and 310), Crohn's disease, lardaceous disease of the bowel, mesenteric thrombosis or embolism, may produce melæna.

4. The GENERAL BLOOD CONDITIONS above named, when the hæmorrhage is small in amount, are attended by tarry instead of bright red stools. *Melæna neonatorum* is a rare condition in which there is a passage of blood in new-born children (see § 551. VII).

5. ANCYLOSTOMIASIS is a possible cause of melæna in Egypt and other foreign countries (§ 547), but generally it is only revealed by tests for occult blood.

The *Treatment* of melæna should be directed to the cause, but the general principles are those laid down for hæmatemesis (§ 272). Turpentine (10 minims capsule), lead acetate, and opium are recommended. Worms are dealt with in § 316. For melæna neonatorum, see § 551. VII.

§ 315. Hæmorrhoids, or Piles are varicose rectal veins. This varicosity forms a swelling of variable size, which may be altogether within the anus (internal piles), or partly internal and partly external. Internal piles may in some cases be seen, when the patient "bears down," as small purple swellings protruding through the sphincter.

Symptoms.—(1) Streaks of *bright red* blood occur in the stools, usually dripping after the bowel has acted; sometimes as much as ¼ pint of blood may be passed at a time. (2) There is pain on defæcation,

the pain continuing for some time after the passage of a stool. When a pile becomes inflamed, or strangulated by the sphincter, severe pain and discomfort are experienced, and the patient may have to remain in bed. Pain may be referred to other parts of the body—*e.g.*, to the testicles or bladder. (3) Constipation nearly always accompanies piles, due partly to mechanical obstruction, and partly to the pain caused by defæcation. (4) Pruritus is often troublesome. (5) In severe cases constitutional symptoms develop due to severe anæmia.

Etiology.—(1) Portal obstruction is itself a cause of piles, and in all cases we should seek for the other symptoms of this lesion (§ 260). (2) Habitual constipation is undoubtedly the most common cause of hæmorrhoids, particularly in women, who in early life are so apt to contract this habit. (3) Alcohol causes portal congestion, and thus becomes a cause of piles. (4) Sedentary occupations and deficient exercise. (5) Various local conditions, such as sitting on a cold seat or soft cushions which constrict the inferior hæmorrhoidal veins, uterine displacements, pregnancy, carcinoma of the rectum, pelvic and other tumours.

Prognosis.—Hæmorrhoids are not serious, but may be extremely troublesome, by the constant loss of blood, by their liability to repeated attacks of inflammation and thrombosis, and by the pain they cause.

Treatment.—Much may be done by three simple means: (1) The avoidance of alcohol (especially malt liquors) and sugar; (2) keeping the piles scrupulously clean, and (3) the bowels regularly and loosely open. Prolapsed piles must be replaced at once. Rich food, wines and other causes of hepatic congestion must be forbidden. Confect. sulph. or sennæ, with an occasional cholagogue at night is good; paraffin is apt to cause the piles to descend. Local applications should be simple. The old-fashioned gall and opium ointment is now replaced by hamamelis, with bismuth, morphia, or cocaine for the pain, if necessary, or calamine powder on a pad of lint: Suppos. bismuth subgallate co. B.P.C. is very useful. Liquid hazeline is excellent, and is best applied on a strip of lint inserted within the anus, and left there; or a suppository may be employed, containing gr. 1 to 3 of hamamelis, and morphia gr. $\frac{1}{8}$, if requisite. Inflamed piles are very painful, and are best treated by warm hip-baths, frequent bathing, sitting over hot water in a bidet, warm fomentations with opium, belladonna or cocaine. Surgical removal is sometimes called for, but a cure may be obtained by a perivenous injection of the subcutaneous tissues around the pile. Use 5 c.c. of a solution containing phenol 20 gr., menthol 1 gr., almond oil to 1 fluid oz. Thrombosis is caused, and healing by scar. A strangulated pile may be incised radially under local anæsthesia, and the clot evacuated: this effects a cure.

A PERI-ANAL HÆMATOMA may be mistaken for a pile. It causes a local swelling and pruritus and is best treated by simple incision and evacuation of the clot. Gas-gangrene infection is rare, but is invariably fatal.

§ 316. Intestinal Worms often cause no symptoms. They are common in children, in inmates of mental hospitals and in adults who come from the tropics. The mor-

phology, symptoms and habitat of various parasitic helminths are described in Tables XVIII and XIX, p. 371 *et seq.* Thread worms (Fig. 78) and round worms (Fig. 79) are the most common in Britain.

Symptoms may result from reflex disturbances, mechanical action, helminthic toxins or anaphylaxis. They include (1) abdominal pain, sometimes paroxysmal in character, (2) capricious or ravenous appetite associated with (3) loss of weight, (4) irregularity of the bowels or diarrhœa, (5) such reflex disturbances as grinding of the teeth at night, enuresis, strabismus and even convulsions. (6) Erythema, urticaria and eosinophilia result from helminthic toxins or anaphylaxis. Skin hypersensitiveness to helminthic protein is manifest by rapid wheal formation following intradermal injection of saline extracts of the different parasites. The round worm, *Ascaris lumbricoides*, may produce helminthic pneumonia during the first week of infection owing to embryos traversing the lungs. Later, after the worms reach the small intestine, the above-mentioned symptoms may appear and occasionally severe manifestations such as: perforation of the bowel with generalised peritonitis or localised abscess with a fistula from which the worm is discharged, intestinal obstruction from masses of worms impacted near the ileo-cæcal valve, obstructive jaundice due to worms obstructing the common bile duct, or cholecystitis, liver abscess or œdema of the glottis associated with worms in these regions. Thread worms (*Enterobius vermicularis* or *Oxyuris vermicularis*) inhabit the colon and migrate through the anus at night, producing pruritus and eczema ani, bladder irritability, sometimes vulvitis and vaginal discharge, and even catarrhal appendicitis. Whip worms (*Trichuris trichiura* or *Trichocephalus dispar*) inhabit the colon, appendix and terminal ileum: the eggs have characteristic knobs. Reflex symptoms may appear and rarely appendicitis and peritonitis. *Strongyloides stercoralis* is a common tropical parasite, the females living in the jejunum and duodenum, occasionally invading the bile ducts and stomach: the eggs hatch out rhabiditiform larvæ which are found in the fæces. Dermatitis and lung symptoms may appear a few days after exposure; later, in severe cases, there is epigastric discomfort and diarrhœa. Urticaria, œdema and occult blood may be present. Ancylostomes are dealt with in § 547. *Heterophyes heterophyes* is a minute intestinal fluke infesting man in Egypt; by means of a sucker it clings to the mucosa and causes indigestion and diarrhœa. *Diagnosis* depends on finding the eggs in the stools, or the parasites themselves after straining the fæces through muslin.

Three different tapeworms may inhabit the intestine of man—*Tænia saginata*, the beef tapeworm; *Tænia solium*, the pork tapeworm; and *Diphyllobothrium latum* (*Dibothriocephalus latus*), the broad-fish tapeworm which undergoes development first in the water flea, *Cyclops*, and later in fish, man becoming infected by eating underdone fish. *Symptoms* may be absent or there may be (1) gastro-intestinal symptoms; (2) reflex disturbances, especially in children and occasionally (3) megalocytic anæmia occurs with *D. latum.* The cysticercus stage of *T. solium* is occasionally found in man, especially in India, producing encapsuled nodules in the muscles, subcutaneous tissues and organs, including the brain; in the latter case epilepsy may result several years after infection, symptoms being associated with the death of the cysts (§ 723).

Diagnosis of the cysticercus stage of *T. solium* is made by biopsy of a cyst, or X-ray examination may show calcified cysts in muscle tissue. Eosinophilia, skin hypersensitiveness to *Tænia* antigen and positive complement fixation reactions may be obtained, but are frequently negative. The prognosis is bad if the brain be involved. The cyst stage of *Echinococcus granulosus*, the small tapeworm of the dog, may affect man, especially involving the liver (see § 347) and lungs (see § 140).

Treatment.—Round worm: santonin 2-grain doses to a child of 3 years and upwards; for an adult, gr. 5. with calomel gr. 2 on three consecutive nights followed by magnesium sulphate ½ oz. in the morning. Oil of chenopodium (1½ c.c.), tetrachlorethylene (4 c.c.) or carbon tetrachloride (3 c.c.) are also specific remedies and are particularly effective where ancylostomiasis co-exists. Children infected with *threadworms* should wear drawers and gloves at night to prevent scratching; the nails should be cut

short; a mercurial ointment may be applied to the anus. The hands should be scrubbed and the buttocks cleaned with antiseptic soap on rising and whenever there is any chance of their being soiled by contact with contaminated clothing, skin or excreta. Gentian violet gr. ½ t.i.d., a.c., may be given in enteric-coated tablets for 3 weeks. The migrating gravid females are best dealt with by enemata, given at night as soon as anal itching appears; 4 oz. of hypertonic saline (1 oz. to 1 pint) or cold quassia are effective; the injection should be given with a baby's Higginson's syringe, and courses are given until the disease is eradicated. In intractable cases a retention enema of hexylresorcinol (1 in 2,000) should be used on alternate days for 10 doses. *Whip-worm* infestations are treated with thymol and oil of chenopodium, by no means always successfully. *Strongyloides stercoralis* is treated with gentian violet given as an enteric coated pill (½ grain) thrice daily after food for 10 days. With the intestinal fluke, *Heterophyes heterophyes* tetrachlorethylene, beta-napthol or thymol is effective. The treatment of the *tapeworm* infestations has three stages: (1) starvation for 12 hours —overnight; (2) the administration of an anthelmintic; (3) purgation. (1) Give no food or drink for 12 hours: this empties the small intestine. (2) Next morning at 7 a.m. give extractum filicis liquidum 30 minims in gelatine capsules or emulsion; this is repeated three or four times during the next hour; in severe cases 30 minims of oil of turpentine may be given with the last dose. (3) Sodium or magnesium sulphate (½ oz.) is given at 10 a.m. and all the motions sieved against a black background to identify the head; if this is not removed recurrence is inevitable. Castor oil must never be given with filix mas, as the active principle, filicic acid, is soluble in it and dangerous toxic effects result. Other anthelmintics include tetrachlorethylene, 4 c.c. for adults, followed by a saline purge in 3 hours, also kousso or pelletierine. Surgical treatment may be necessary in *hydatid* of the liver and lungs, and rarely for intestinal perforation or obstruction in ascaris infection.

§ 317. **Constipation** is insufficient action of the bowels, delay in the passage of the contents of the intestine, causing hard, dry fæces (scybala), independent of organic disease within or outside the intestinal canal. This source of fallacy must be carefully excluded before diagnosing a case as one of simple constipation. *Causes.*—(1) The usual cause is insufficient or incomplete movement of the mass of contents collected in the proximal colon. (2) In about a fourth of cases the delay is in the sigmoid colon and rectum (dyschezia). (3) A third form occurs when spasm of the colon prevents the mass movement from forwarding the contents through the region of the spasm (spastic constipation). (4) In elderly patients and those with feeble musculature there may be delay or absence of initiation of the mass movement. (5) A rarer type is due to lack of residue from too complete absorption of water and ingested food. A simple test for delay consists in giving a tablespoonful of powdered charcoal at night; it should normally have completely disappeared from the stools before seventy-two hours.

The *Symptoms* which accompany or result from constipation are sufficiently familiar—at first headache, languor, and depression, followed by a furred, coated tongue, dyspepsia, sallow or pigmented skin, anæmia, sleeplessness, and eruptions, usually of an urticarial or erythematous nature. The temperature may rise a degree or so in certain conditions from temporary constipation, and even up to 102° F. The retention of hard fæcal masses may give rise to an alternating diarrhœa, which leads to error in diagnosis. Habitual constipation may give rise to hæmorrhoids,

and even to a distended ulcerated colon or atony of the colon. In women, in whom the condition is more common than in men, chronic constipation aggravates any pelvic disease. In both sexes varicose veins, œdema of the legs, sciatica, especially on the left side, and numbness of the legs may occur; these are more usually associated with diverticulitis (§ 321). In some cases there may ensue ptosis of part of the intestine, leading to delay of the bowel contents; then follows prolonged ill-health due to the toxæmia of INTESTINAL STASIS.

For purposes of treatment we may consider the Causes of simple or uncomplicated cases of constipation under three headings:

(*a*) **Errors of Diet.**
- (i.) Too bland food—*e.g.*, no vegetables, no food with coarse residue.
- (ii.) Too rough and irritating food, in certain cases of spasm.
- (iii.) Too dry food—*e.g.*, deficient fluid ingesta.
- (iv.) Too little or poor food, deficiency of vitamins.

(*b*) **Causes of Defective Peristalsis,** other than errors of diet.
- (i.) Sedentary habits.
- (ii.) Depressing emotions, anxiety, worry, etc., cause sympathetic inhibition, hence spasm, as in "spastic colon."
- (iii.) Old age and other conditions with poor general tone, such as anæmia.
- (iv.) Prolonged disregard of the calls of nature, with dilatation of rectum and pelvic colon consequent on blunted sensation.
- (v.) Weak abdominal muscles.
- (vi.) Atony of the colon, with or without colitis.
- (vii.) Some febrile states.
- (viii.) Endocrine disorder, especially deficient activity of thyroid and pituitary.
- (ix.) Reflex spasm, as in catarrh of the colon or uterine disease.
- (x.) Disease of brain or cord, such as tabes and cerebral tumour.
- (xi.) Drugs, such as opium, iron, lead.

(*c*) **Deficiency of Bile,** or **Intestinal Secretions.**
- (i.) Functional inactivity of the liver.
- (ii.) Profuse vomiting.
- (iii.) Excessive loss of fluid by skin or kidneys.
- (iv.) Astringents, such as chalk or catechu. Hard waters also act in this way.

Diagnosis.—As chronic constipation may lead to the troublesome consequences mentioned above, we must first *find the cause.* With the patient lying down and the muscles well relaxed, examine the abdomen to see if the colon be distended or loaded; place one hand at the back, and press it forwards between the iliac crest and the last rib to meet the other hand, which is placed flat on the anterior abdominal wall. Make a rectal examination. An X-ray examination assists in deciding the presence or absence of mechanical obstruction, and the position of chief delay in the passage of the intestinal contents. Having excluded local causes by a thorough examination, we should consider the various causes above mentioned.

The *Treatment* of constipation comes under the following headings.

(1) *Dietetic.*—Increase the amount of fluid taken—*e.g.*, by sipping a tumbler of cold water slowly whilst dressing in the morning and undressing

at night. Avoid large quantities of milk or hard water. To provide bulk and stimulus, where there is no spasm, but chiefly dyschezia, foods to be eaten include oatmeal, wholemeal, National or brown bread, green and raw vegetables, onions, figs, prunes, and ripe fruits (see § 297. V). A teaspoonful or tablespoonful of salad-oil at mealtimes aids this diet. Where there is colonic spasm, give smooth food with little residue. (2) *Lubricants* may be used for short periods; paraffin, plain or in emulsion, bassorin, psyllium seeds and agar-agar preparations, provide bulk without irritating material, an important point in cases of spasm. (3) Inculcate *regular habits.* As stated above, in about one-fourth of the cases of simple constipation the delay is in the sigmoid and the upper rectum; hence it is important, even when there is no inclination to go to stool, that an attempt should be made at a regular hour daily, for 10 minutes by the watch, trying at regular intervals but being careful not to strain so hard as to cause pain in the abdomen or in the head. The squatting position aids, and lessens strain. If there is no result, a glycerine suppository should be inserted, and after waiting 10–15 minutes another effort made. That failing, a soap and water enema is given on the second day. Psychotherapy is useful in some cases. (4) *Active exercise* is advisable except when uterine or ovarian disease or colonic spasm is present; many systematic exercises are now taught which strengthen the abdominal and pelvic muscles. (5) *Electricity* is used in various ways; some forms relax spasm, others stimulate to healthier muscular action. Abdominal *massage* is useful; gently "rolling" the abdominal wall, or rolling a 7-pound shot-ball over the abdomen in the direction of the hands of the clock. (6) With signs of *endocrine deficiency,* as of the thyroid or pituitary, extracts of these glands greatly aid constipation. Bile extracts are efficacious in other cases.

(7) *Drugs.*—To avoid prolonged use of drugs give methodical trial of the measures above mentioned. For occasional constipation, senna with the evening meal and a seidlitz-powder in the morning are the most harmless. Calomel or other mercurial preparations should not be given habitually, but may be taken once a week for a few weeks. Phenolphthalein is an excellent preparation for temporary use. Cascara, aloes and senna may be used frequently. A useful vegetable pill is pil. colocynth co., pil. rhei co., āā gr. I., ext. hyoscyami, gr. ½; one or two at bedtime. Another good formula is tr. nuc. vom., tr. belladonna, āā ♏ 5; tr. hyoscyam. ♏ 10, ext. casc. sag. liq. ad ♏ 60. Nux vomica in small doses promotes peristalsis; belladonna is especially useful to relax spasm of the colon, and in simple dyschezia. Jalap, elaterium, scammony and gamboge are useful for drastic purgation. Salines given daily for some weeks will often re-establish the functions of a torpid intestine (F. 46). These may be given in the form of the mineral waters, such as Carlsbad, or their equivalents, which contain 20–60 grs. of sulphate of soda, sometimes with alkalies. They are best given on an empty stomach (F. 51 and 90 are also useful). An excellent aperient for children is cascara and malt mixed together in the proportion of 10 to 20 ♏ of the

ext. casc. sagrad. liq. to the teaspoonful of malt. (8) *Enemata* are used in conditions of atony of the descending and pelvic colon, and dilated rectum—1 or 2 pints of plain water may be given, at gradually longer intervals. Half an ounce of glycerine is an effective enema, but it should not be used longer than a few weeks, for it tends to irritate the rectum. In cases of very prolonged constipation, ⅛ to ¼ pint of olive oil may be given every night. If this be injected very slowly, it is retained, and after a course of one or two weeks the bowel often resumes its functions. (9) *Lumbar sympathectomy*, including the removal of the presacral plexus, may succeed in very obstinate cases, where these measures fail, and where evacuations occur at long intervals or only with enemata.

Colon irrigation with normal saline is often necessary where hard masses can be felt in the cæcum. One or two pints at a time, at body temperature, are introduced slowly under a pressure of not more than two feet, and are immediately evacuated. This is repeated until the washing is returned clear. It is best preceded by injection of 3 fl. oz. of warm olive oil to be retained for a few hours. Carried out daily for a week, then on alternate days, and later once a week, this is very effective in clearing the colon of accumulated fæces. Gradually the bowel resumes its normal functioning. The only type of case in which this is not very satisfactory is that in which there is considerable ptosis and as a result the whole of the saline is not evacuated at once. The repeated calls to stool are annoying, and frequently this type of patient complains of depression and increase of toxic symptoms.

Hirschsprung's Disease (megacolon) is a condition of atony and dilatation of the colon of congenital origin: it is ten times more frequent in boys than girls. The cause is developmental or an abnormality of the neuro-muscular mechanism of the bowel due to overaction of the sympathetic nervous system. There are two types—(*a*) pelvi-rectal and (*b*) anal.

Symptoms.—There is obstinate constipation from the first weeks of life, and subsequently gross abdominal distension, tympanites with visible peristalsis, auto-intoxication and emaciation. If early childhood is survived, complications such as peritonitis and intestinal obstruction may ensue. The disease is often fatal in the absence of treatment.

The *Diagnosis* can be made only by the history and obvious signs of a distended colon. A barium enema shows enormously dilated and redundant bowel. A similar condition may be acquired by prolonged bad habits.

Treatment.—Attend to the diet and try the effect of liquid paraffin and large enemata combined with prostigmin 5–10 mgm. t.i.d.: otherwise regular dilatation of the anus with the fingers or an obturator. Spinal anæsthesia—which may have to be repeated—has given promising results. Lumbar sympathectomy, with removal on both sides of the greater and lesser splanchnic nerves, the first four lumbar sympathetic ganglia, and the pre-aortic plexus has also helped, but must be performed before permanent damage has been done.

§ 318. **Intestinal Flatulence** may be due to fermentation of carbohydrates, especially in the colon, but more often to interference with the absorption of air which has been swallowed with food or drink. Anything which causes congestion of the intestinal veins will delay the absorption of the intestinal gases, and give rise to distension and flatulence, such as heart failure and pulmonary disease, portal congestion and local venous block, as in volvulus and intestinal obstruction.

Symptoms.—There is a sense of fullness with discomfort which may be painful, relief being obtained by loosening the clothing, eructation

or passing flatus. Breathing may become embarrassed and palpitation or irregularity of the heart occur.

Treatment consists in reducing the vegetables and cellulose of the diet, and giving charcoal biscuits and carminatives. When constipation is present, Gregory's powder is most useful. Pituitary extract or prostigmin may be used, by injection: to relieve spasm, give trasentin, tablets of phenobarbitone, or atropine.

The patient complains of SUDDEN STOPPAGE OF THE BOWELS *with inability to pass even flatus,* ABDOMINAL PAIN, *and* VOMITING *which gradually becomes stercoraceous; there is increasing abdominal distension, and a tendency to* SHOCK. The case is one of ACUTE INTESTINAL OBSTRUCTION.

§ 319. **Acute Intestinal Obstruction** is one of the most serious medical or surgical emergencies.

The *Symptoms* common to all forms of acute obstruction are—(1) complete constipation, not even flatus being passed. Absolute constipation can be assumed only when flatus cannot be passed even after repeated enemata. (2) Pain may be acute at first, and referred to the umbilicus, though later it may be superseded by colicky pains, owing to the peristalsis of the bowel trying to overcome the obstruction. There is not usually much tenderness. (3) Vomiting is a prominent symptom from the onset. It is copious and projectile, first of food, then bile, and later material which is alkaline to litmus and finally fæcal. It comes on earlier, is more urgent, and becomes more rapidly stercoraceous in proportion as the obstruction has taken place high up in the intestines. (4) Abdominal distension is generally present; it is more in the flanks with obstruction to the colon, and more central with obstruction to the small intestine. (5) Peristalsis may be visible. (6) Constitutional symptoms gradually supervene, with prostration and a thready, *rapid pulse.* These also are more urgent when the small intestine is involved. The urine is diminished in proportion as the obstruction is near the stomach, for then the vomiting is more urgent. (7) Tetany can occur in high obstruction of the small intestine.

Diagnosis of Acute Intestinal Obstruction.—When summoned to a case presenting these three symptoms—stoppage of the bowels, vomiting, and acute abdominal pain—the first step is to identify the case as one of acute obstruction. In *colic* (renal, hepatic, or intestinal) all of these three symptoms may be present, but the patient's general condition is not so serious. Moreover, the position of the pain in renal and hepatic colic is characteristic (see § 246). In *acute peritonitis* there is great tenderness over the abdomen, thoracic respiration, and some fever (see also § 244). But when there is *perforation* into the *peritoneum* shock is present, at first without fever, and perforation is diagnosed with difficulty only by (i.) the pain is constant and there is local tenderness; (ii.) the passage of wind by the bowel; (iii.) the collapse being much greater even than that in acute obstruction; and (iv.) a possible history of the condition which has resulted in perforation or rupture (consult also § 243). It is

sometimes impossible to diagnose between these two conditions, and an exploratory operation should be undertaken without delay.

Causes of Intestinal Obstruction.—It is of some importance to ascertain the cause, for the prognosis and treatment differ somewhat in each case. (*a*) In *acute* intestinal obstruction, in which the symptoms come on *suddenly* in a person previously healthy, there are four *common* causes: (I) External hernia; (II) intussusception; (III) internal strangulation; (IV) paralytic ileus. (*b*) Sometimes, however, acute will supervene on chronic obstruction, and the *common causes of chronic obstruction* (§ 320) are four in number: (I) Malignant stricture of the bowel; (II) simple stricture; (III) pressure of a tumour; and (IV) diverticulitis.

Features special to the several causes of acute intestinal obstruction.

I. External Hernia is known by the presence of a tumour in the femoral, inguinal, or umbilical region. No impulse on coughing is present. Obturator hernia is very rare, and is usually only discovered at operation.

II. Acute Intussusception, or invagination of the bowel, is the commonest cause in childhood. True intussusception is always from the bowel above into the part below, and in more than half of the cases the lower part of the ileum becomes invaginated into the cæcum. In a third of the cases some other part of the ileum, and in about one-eighth some part of the colon, is implicated. The invaginated portion slowly sloughs, the two edges may be welded together, the slough may pass about the eighth or tenth day; thus spontaneous recovery may occur, though this is rare. Death from perforation and collapse is more usual unless the case is dealt with surgically. Intussusception is known by (i.) severe tenesmus; (ii.) a rectal discharge of *blood and mucus* with a red jelly appearance; (iii.) a sausage-shaped tumour may be felt, altering in position, on palpating the abdomen, and in extreme cases the invaginated portion of bowel is felt *per rectum*; and (iv.) the patient is usually a previously healthy boy under two years of age.

III. Internal Hernia or Strangulation—*e.g.*, by bands of adhesion—is known by (i.) the urgency of the symptoms; (ii.) the patient is an adult, with (iii.) a history of old peritonitis or previous operation. Volvulus (or twisting of the bowel) may be indistinguishable from the preceding—indeed, it practically results in strangulation—but (i.) it occurs in men over forty, usually with a history of chronic constipation; (ii.) abdominal distension may be great; (iii.) sometimes a tumour is felt over the sigmoid flexure, the usual site of volvulus.

Internal strangulation may also arise from (1) adhesion of the end of the appendix vermiformis through which a knuckle of the bowel gets nipped. (2) Adhesions of the bowel. (3) Congenital deficiencies in the mesentery or bowel, or the foramen of Winslow.

IV. **Paralytic ileus** is one of the most dreaded complications following a surgical operation. It may complicate pneumonia. It may be defined as a condition of "intestinal inertia," in which the intestine is incapable of muscular action, and becomes distended with gas. Its pathology is

not settled; it may be caused by injury to the wall of the gut, by interference with its blood supply or the nervous visceral connections.

Symptoms.—A mild form is met with after any major abdominal operation, and is manifested by constipation, gas and windy spasms; there may be some contraction of the sphincters. This occurs after the first 24 hours, with moderate distension, usually relieved at the end of the 3rd day by an enema. True paralytic ileus sets in about the 2nd or 3rd day, with pain, vomiting, distension and absolute constipation. The pain is a dull ache, not colicky, and there is complete cessation of peristalsis. Vomiting is persistent and copious. It may last 3 to 4 days and then clear up, but if unrelieved death takes place about the 3rd, 4th or 5th day.

It must be *diagnosed* from mechanical adhesive obstruction. The latter occurs later, from the 3rd to the 7th day. It has the same insidious onset; vomiting becomes progressive, and the material is alkaline to litmus, but there is usually colicky pain and peristalsis may be detected.

Treatment.—At the end of 24 hours ox-bile or turpentine enemata should be given and a flatus tube left in. Nothing should be given by mouth; the stomach should be kept empty by continuous aspiration, and the fluid intake kept up by a continuous glucose-saline infusion intravenously. Heat to the abdomen and morphia (gr. $\frac{1}{12}$ – $\frac{1}{8}$ four-hourly for five or more doses) are beneficial. If these are unsuccessful, carbachol subcutaneously (0·25–0·50 mgm.) or prostigmin 1 c.c., repeated two or three times, and followed by a glycerine enema often helps. Should these methods fail, a spinal anæsthetic should be given and if no result follows, operation is necessary. Only in desperate cases should ileostomy be performed.

The **rarer causes** of acute obstruction are three in number:

V. IMPACTION IN THE BOWEL of a large GALL-STONE. A large gall-stone escapes from the gall-bladder by ulceration into the bowel. The obstruction is high up in the small intestine, and consequently (1) the pain and constitutional symptoms are of extreme severity, and of very sudden onset. (2) The patient is usually a woman at or beyond middle age. (3) There may be a history of biliary colic, and in all cases there is a history of localised peritonitis some weeks or months before the seizure. (4) The symptoms may intermit, from the stone shifting its position.

VI. Obstruction of the bowel may sometimes be due to an EXTRAVASATION OF BLOOD into the coats of the intestine. It occurs only in purpura, hæmophilia, and other blood disorders. Such cases are recognised by evidences of hæmorrhage in other positions—melæna, epistaxis, purpura, or a history of urticaria or angioneurotic œdema.

VII. Among the still rarer causes of obstruction may be mentioned masses of round worms (Trousseau), impaction of too much cellulose, orange-peel, etc., hair-balls, concretions of ammonio-phosphate of magnesium (a frequent cause in horses, though rare in man), and other foreign bodies in the intestine.

Clinical Investigation and Diagnosis of the Cause of Obstruction.—If the case occurs in a child, and there is a history of sudden onset, it is almost certainly intussusception; in an old person suspect growth, impacted fæces, diverticulitis, or volvulus; in a young adult suspect strangulation or hernia. If the vomiting comes on early and is urgent,

it points to a tight constriction *high up* in the intestinal tract. So also after the onset of obstruction high up there may be a movement of the bowels. If the distension is chiefly in the centre of the abdomen, the obstruction is probably above the ileo-cæcal valve; if it is chiefly in the flanks, the obstruction is below the valve; if more in the right than in the left flank, the obstruction is probably in the splenic flexure.

When called to such a case, first examine for swelling in the positions of external herniæ. If the abdomen be distended, and presents visible waves of peristalsis, inquire as to the causes of chronic obstruction (*infra*), as the case is probably an acute supervening upon a chronic obstruction. Always *examine per rectum*, for in acute intussusception the invaginated part of the bowel may be felt *per rectum*, and there may be a discharge of blood and mucus; or a stricture or other cause of chronic obstruction may thus be discovered. Next inquire into the past history—*e.g.*, for peritonitis (as this is a cause of internal strangulation), or for appendicitis or hepatic colic. Then examine the abdomen by palpation and percussion for tumour or tenderness. If the abdomen is distended only on one side, the site of the obstruction may be localised.

Prognosis.—The prognosis of obstruction of the bowels is always very serious. Death occurs in the natural course either from (1) gangrene and rupture of the bowel, or (2) exhaustion and collapse. At the present day the prognosis almost entirely depends upon the *stage at which the case comes under notice*, and the treatment adopted. All the acute cases require early surgical interference, and a surgeon should be summoned at once. As regards the *Causes*, obstruction from a gall-stone is perhaps the most serious, then intussusception, then internal strangulation. Among the gradual causes, carcinoma of the bowel gives the gravest prognosis, and paralytic ileus the most favourable. Cases in which the obstruction is high up are less favourable than those in the large bowel.

Treatment.—Acute intestinal obstruction is one of those serious conditions that demand the resources of both a physician and a surgeon, who should jointly undertake the management of a case. The indications are (1) to ascertain the cause; (2) to endeavour to remove the obstruction; and (3) in the meantime to support the strength and relieve the pain by controlling the peristalsis upon which it depends. Enemata of soapy water to which olive oil, glycerine or oxgall is added may be given in all cases; purgatives by mouth should be avoided. Warmth is applied to the abdomen in the form of hot fomentations, turpentine, belladonna, or opium stupes. The question of the administration of opium is debated (see Appendicitis), but, generally speaking, for the relief of the pain opium may be given as soon as the diagnosis is certain. The diet should consist of fluids, such as iced milk, beef-tea, and stimulants, given in small quantities, and frequently.

In *external hernia*, after a warm bath, it is best to proceed at once to operation. In *intussusception* some mild cases have a tendency to

spontaneous recovery. Some surgeons recommend that an attempt should be made to reduce it by injections of warm saline or olive oil, but it is better to proceed at once to laparotomy. In *internal strangulation* or twisting it is best to operate without delay if an injection does not relieve and we are certain of the diagnosis. In cases of recovery without operation there has probably been a simple volvulus. But death almost always occurs in cases of internal strangulation if unrelieved. Manipulation, and inflating the bowel by means of bellows are dangerous procedures. In *impacted gall-stone*, the progress is so rapid towards a fatal issue that operation, if undertaken, must be immediate. The same remark applies to other foreign substances in the intestine.

The patient complains of CONSTIPATION *progressively increasing,* ABDOMINAL PAIN, *and from time to time* VOMITING; *there is general ill-health.* The case is one of CHRONIC INTESTINAL OBSTRUCTION.

§ 320. In **Chronic Intestinal Obstruction** (1) the abdominal pain is generalised, intermittent, and of increasing severity. (2) There is constipation, or a history of alternate constipation and diarrhœa culminating in complete stoppage; and (3) abdominal distension in most cases, and peristalsis in some, may be visible. The chief causes are four in number:

I. MALIGNANT STRICTURE by new growth in the wall of the bowel—*e.g.*, cancer. Its most common situations are the colon, especially the sigmoid flexure, and the rectum. This cause of obstruction may be recognised by (1) the presence of a tumour or stricture which may be felt on examination *per rectum*, and the distension of the abdomen being mostly in the flanks. When the tumour is situated higher up than the sigmoid flexure, it may generally be felt through the abdominal wall; and when situated in the sigmoid flexure, it may be inspected by a sigmoidoscope. (2) When the sigmoid flexure or rectum is affected, the illness is often preceded by sciatica on the left side. (3) There are cancerous cachexia, the age of the patient, and perhaps hæmorrhage, fœtid discharge and often ascites to aid in the diagnosis. (4) X-ray with barium meal and barium enema combined with air insufflation will show a filling defect (Fig. 82).

II. SIMPLE—*i.e.*, NON-MALIGNANT STRICTURE of the intestine may arise in consequence of amœbic dysentery, syphilitic, or other ulceration, either in the colon or in the rectum. An ulcer alone is capable of producing symptoms of obstruction. This cause is recognised by a previous history of dysentery (perhaps only a mild attack), and residence in a tropical climate; or a history of syphilis, with a rectal discharge. Syphilitic stricture is rare, except between the sigmoid flexure and the anus.

III. PRESSURE ON THE BOWEL by a tumour, tuberculous or other adhesions, or an enlargement of some viscus such as the uterus. This cause is recognised by the physical signs of tumour or enlargement respectively.



FIG. 82.—BARIUM ENEMA.—A. Carcinoma of transverse colon; could possibly be confused with a filling defect caused by pressure from spine. B. The atrial picture. Compression brings the filling defect clear of the spine. C. Oblique view after air inflation, showing the absence of relaxation of the indurated part.

Rarer Causes of chronic intestinal obstruction are:

IV. CHRONIC PERITONITIS (§ 250) causes a matting together of the intestines, and intestinal obstruction may result. Cancerous peritonitis is attended by much pain and the effusion of much fluid; but in tuberculous peritonitis (§ 557) there are mostly adhesions, less pain, and less fluid. Localised peritonitis occurs as a result of diverticulitis, usually in the left lower abdomen.

V. CHRONIC INTUSSUSCEPTION is thus known: (1) It occurs usually in children; (2) tenesmus is present; (3) a tumour may be felt similar to that met with in acute intussusception; and (4) there is usually no great distension (see also Acute Intussusception, above).

VI. HIRSCHSPRUNG'S DISEASE (§ 317).

Prognosis of Chronic Intestinal Obstruction.—In all forms the symptoms of acute obstruction are apt at any time to supervene, from impaction of fæces above the narrowing lumen of the gut, but apart from this the prospect differs considerably in the various causes. A cancerous stricture is the most, diverticulitis the least, serious. Syphilitic stricture may be relieved by arsenic and iodides; dysenteric stricture is irremediable. The course of a tumour varies with its nature. Chronic intussusception *may* spontaneously resolve, the invaginated part sloughing off and being passed by the rectum, but the outlook is always grave.

Treatment of Chronic Intestinal Obstruction.—In most cases surgery is ultimately necessary, but at first the treatment consists in watching the patient until a diagnosis can be formed with as much accuracy as possible, giving digestible food, preferably such as leaves but little residue, and relieving pain by opium and external applications (hot fomentations with turpentine or opium). For simple *stricture of the rectum* gradual dilatation by bougies may be tried. In *chronic intussusception* operation is advisable. In *cancerous stricture* where radical removal is impossible life may be prolonged by colostomy; the longer the operation is delayed, the worse is the prognosis. It should never be delayed until vomiting has begun. Deep X-ray therapy sometimes aids.

A man of middle age or over who has suffered from FLATULENCE *and* IRREGULARITY OF THE BOWELS—*usually constipation—for some time, has an attack of* PAIN IN THE LEFT ILIAC FOSSA *with some fever; the disease may be* DIVERTICULITIS.

§ **321. Diverticulitis** of the colon is not an uncommon complication of diverticulosis. Patients are usually above middle age and often obese. For some years there may have been irregularity of the bowels, with a tendency to constipation, and at intervals attacks of diarrhœa which are ascribed to indigestible foods. Sometimes there is mucus and occasionally even blood. There is (i.) some degree of fever and often leucocytosis; (ii.) tenderness and rigidity in the left iliac fossa; (iii.) sometimes a local mass is felt, due to simple inflammation or abscess formation; (iv.) irritation of the bladder with frequency of micturition; (v.) there may be occasional rigors; and (vi.) cystitis may accompany.

For *Diagnosis* of these cases, see also § 319. Diverticulitis has to be diagnosed from *cancer* of the colon and *appendicitis*. X-ray after a barium enema shows a contracted and irregular lumen of the bowel in the sigmoid region and diverticula scattered throughout the rest of the colon. Complications are: local peritonitis from peri-diverticulitis, perforation with general peritonitis, or obstruction.

Treatment: (*a*) during the acute attack, consists of bed, fluid and semi-solid diet, poultices or stupes to the abdomen, liquid paraffin by



FIG. 83.—DIVERTICULITIS OF SIGMOID.

mouth and by injection into the bowel. (*b*) Between the attacks, the diet should be fuller, but must contain nothing hard or indigestible; liquid paraffin should be taken by mouth two or three times a day. A course of intestinal douches with normal saline, given at low pressure, may be necessary, to clear away the accumulation of fæces and soothe the bowel. The rectal injection of 3 fl. oz. of warm olive oil two or three times a week, the patient lying on the left side and retaining it as long as possible, is of considerable value.

CHAPTER XII

THE LIVER

THE liver is the largest gland in the body and the fact that it can contain a fourth of the blood in the body shows its importance in the control of the circulation. All the blood passing from the stomach and intestines circulates through the liver, after which it joins the general circulation considerably altered in its composition. The pancreas and the liver work in close co-operation, the pancreatic internal secretion passing direct to the liver. The liver aids in preparing proteins, carbohydrates and fats for the tissues. Other important functions of the liver are: the manufacture and the storage of glycogen; the secretion of bile, containing bile salts and pigments; a detoxicating action by arresting poisons and bacteria absorbed from the intestinal tract, and converting certain noxious chemical substances—indol and skatol—into innocuous compounds; the elaboration of the products of protein metabolism into urea and uric acid, the storage of the anti-anæmic factor against pernicious anæmia, and the production of prothrombin and fibrinogen, both necessary for blood clotting. The Küpffer cells form part of the reticulo-endothelial system. The liver has considerable reserve and much power of regeneration.

PART A. SYMPTOMATOLOGY.

The symptoms due to disorders of the liver are not so clearly defined as those of cardiac or pulmonary diseases. The cardinal symptoms of *structural* disease of the liver are PAIN IN THE HEPATIC REGION, JAUNDICE, and a group of symptoms due to PORTAL OBSTRUCTION, which includes Ascites. When the liver cells become gradually destroyed, as in hepatitis, serious disturbance of the general health ensues, and in the later stages of that and of some other hepatic disorders LETHARGY passing into COMA supervenes. Functional derangement of the liver is attended by DEPRESSION and vague DIGESTIVE DISTURBANCES.

§ 325. **Pain and Tenderness over the Liver** occur in PERIHEPATITIS and any other condition in which the capsule is involved or stretched, as in heart failure. The pain may radiate upwards towards the right scapula. The onset of pain in the course of a liver complaint may therefore be of considerable importance; for example, in hydatid of the liver the natural course of which is painless, it would point to a danger of rupture of the cyst. When the upper surface of the liver is involved, the pain is very often *referred to the right shoulder*; it is, indeed, a symptom of phrenic (diaphragmatic) irritation. The most severe form of pain, however, is that which occurs with the passage of GALL-STONES (*biliary colic*). Pain may be completely absent in hepatic disorder. There is,

however, in many cases of disease or enlargement of the liver a feeling of weight or fullness in the right hypochondrium, accompanied by an inability to lie on the left side.

Hepatic pain may be *simulated* by Pleurodynia (rheumatism of the intercostal muscles), Intercostal Neuralgia, Pleurisy, Dyspepsia and various gastric conditions, and by Intestinal or Renal Colic.

§ **326. Jaundice** is the term applied to the yellow pigmentation of the skin and other tissues due to the non-elimination of bile. It appears first in the blood then in the urine, in which increased urobilin, bile pigments and salts may be detected (§ 383), next in the conjunctivæ, then in the skin uniformly.

FALLACIES.—The yellow coloration of the conjunctivæ differentiates jaundice from all similar pigmentations of the skin. (1) Excess of *subconjunctival fat* may simulate jaundice, but this is readily distinguished by its unequal distribution. (2) The *sallowness* of the skin in anæmia is distinguished by the absence of bile in the urine and of yellowness of the conjunctivæ. (3) The *cachexia* of carcinoma, malaria, tuberculosis, and certain other forms of visceral disease, is differentiated in the same way. (4) The *bronzing* of the skin in Addison's disease is hardly likely to be mistaken for jaundice. (5) Long-continued mepacrine administration colours the skin yellow. (6) *Santonin* and *rhubarb*, administered internally, colour the urine, but do not give the reactions for bile in that fluid. (7) Carotinæmia (§ 653) may be mistaken.

Jaundice is classified as follows: (*a*) Obstructive; (*b*) Toxic or infective; and (*c*) Hæmolytic. Pure instances of these varieties are rare; even with obstructive jaundice, damage to liver cells follows.

(*a*) Clinically, **Obstructive Jaundice** is distinguished by the colour of the stools, which are pale, slate or clay-coloured, due to the absence of bile in the intestinal canal. There is increased intestinal putrefaction and steatorrhœa, due to the increase of soaps and fatty acids in the stools. The urine is high-coloured and contains bile. The blood gives an immediate direct van den Bergh reaction (§ 331), the icteric index (§ 331) is raised to 10–15; the bile salts and cholesterol in the blood are also increased and the coagulation time is prolonged. Pruritus is severe. Leucocytosis is present in advanced obstructive jaundice, but not in mild cases unless accompanied by inflammation or suppuration.

Obstructive jaundice may be produced in four ways:

I. FOREIGN BODIES within the bile-duct, such as (1) gall-stones and inspissated bile; (2) hydatids, round worms, Fasciola and other parasites; (3) foreign bodies from the bowel.

II. INFLAMMATION of the bile-ducts, usually spreading from the duodenum.

III. STRICTURE, or obliteration of the duct owing to (1) congenital absence; (2) perihepatitis; (3) cicatrisation after ulcer of the duodenum; (4) ulceration of the bile-duct, which may produce obstruction by the swelling around, or lead to stricture; and (5) chronic pancreatitis.

IV. TUMOURS pressing on the duct, such as (1) cancer and other tumours of the liver; (2) enlargement of the glands in the transverse

fissure of the liver; (3) tumours of the stomach, pancreas, kidney, great omentum; (4) fæcal masses in the intestines; and occasionally (5) tumours growing from the walls of the ducts.

(*b*) In **Toxic or Infective Jaundice** some bile usually reaches the intestine, so that the stools are not always clay-coloured, and severe damage to the liver may occur without marked jaundice. The blood in toxic jaundice gives an indirect or a biphasic van den Bergh reaction (§ 331). The urine contains excess urobilin, increased amino-acids and ammonium salts. Pruritus is not severe. This form of jaundice follows damage to the liver cells, which become inefficient, thus favouring retention of bile pigment and salts in the blood. The damage may be mild and recovery be complete, or severe, as in acute yellow atrophy of the liver, or partial recovery may follow, leaving some degree of cirrhosis. The causes of toxic jaundice are: (1) bacterial or protozoal poisons such as occur in infective hepatitis, pneumonia, syphilis, septicæmia, typhus, typhoid, relapsing fever, malaria, spirochætosis ictero-hæmorrhagica, yellow and other tropical fevers; (2) chemical poisons such as trinitrotoluol, tetrachlorethane (poisons affecting munition workers), phosphorus, arsenobenzol derivatives, nitrobenzene, cinchophen B.P. (atophan), dinitrophenol, ether and chloroform; (3) toxæmias as in pregnancy; (4) chronic heart disease with congestion.

(*c*) In **Hæmolytic Jaundice** (1) the fæces are of normal colour; usually there is no bilirubin or bile salts in the urine, but much urobilin or urobilinogen (§ 383). The spleen is usually enlarged and there may be perisplenitis. (2) There is a delayed direct and an indirect van den Bergh reaction. (3) This form of jaundice is caused by increased blood destruction and is of extrahepatic origin. Its causes are: (A) increased fragility of the red blood corpuscles, as in congenital or acquired acholuric jaundice; (B) increased destructive agents: (1) animal poisons, such as snake-venom; (2) streptococcal infections; (3) pernicious anæmia; (4) specific hæmolysis as in transfusion with incompatible donors. The icteric index is much increased (§ 331). Physiologically, this form occurs in the jaundice of the newly-born.

Of all these causes *gall-stones* and *infective hepatitis are the most common.*

To *diagnose* the type of jaundice (see Table XXI): 1. EXAMINE THE FÆCES, which are slate or clay-coloured in complete obstruction, and of normal colour in hæmolytic jaundice. The presence of fat or parasites may assist in diagnosing the cause. But it must be remembered, as possible fallacies, that the fæces may become stained if mixed with urine; and that the bile-duct may be only partially obstructed, and enough bile may thus escape to colour the fæces.

2. EXAMINE THE URINE for bile pigments and salts (§ 383).

3. Inquire as to the HISTORY OF THE ATTACK. Jaundice coming on suddenly, especially in a middle-aged woman previously in good health, almost invariably indicates obstruction by gall-stones. The intensity of the jaundice varies from week to week as the stones pass. Jaundice coming on slowly, and ultimately becoming intense, is generally due to a

tumour pressing on the common bile duct, and is most often seen in association with cancer. Severe jaundice persisting some weeks is almost certainly obstructive. Occupation in a munition factory or previous intravenous treatment with arsenobenzol derivatives, renders easy a diagnosis of the cause. A history of previous temporary attacks points in adult life to gall-stones; in youth, to infective hepatitis.

4. EXAMINE THE HEPATIC REGION CAREFULLY. If the liver is enlarged, cancer is the most probable cause; interstitial hepatitis is less common. If the gall-bladder is enlarged, cancer is more probable than stone. If ascites be present, the diagnosis rests between cancer and cirrhosis.

5. Inquire as to PAIN AND CONSTITUTIONAL SYMPTOMS. Pain of a spasmodic and severe character accompanies jaundice due to gall-stones and cancer. It is more constant and gnawing in character in congestion of the liver and catarrh of the bile-ducts. The temperature is not often

TABLE XXI.—DIFFERENTIATION OF TYPES OF JAUNDICE.

	Obstructive	*Toxic or Infective*	*Hæmolytic*
Onset	Stormy.	Quiet.	Chronic.
Colour of skin	Yellow, orange or greenish.	Yellow, orange or greenish.	Light yellow—"lemon yellow."
Distribution of pigment	Seen in conjunctivæ before skin.	Seen in skin before conjunctivæ.	Conjunctivæ rarely affected.
Irritation of skin	May be severe.	May be present.	Not present.
Colour of stools	Pale.	Normal or pale.	Normal.
Urine	Bile pigments present.	Often no bile pigments: urobilin present.	No bile pigments: urobilin present.
Liver	Large or very large.	Large, normal or small.	A little large or normal
Gall bladder	May be palpable.	Not palpable.	Not palpable.
Spleen	Rarely palpable.	Rarely palpable.	Often palpable.
Anæmia	May or may not be present.	Usually not marked.	Severe.
Van den Bergh Reaction	Biphasic.	Biphasic or indirect.	Delayed direct or indirect.
Sedimentation rate	Usually normal.	Normal or prolonged.	Much increased.

elevated, but it may be so in infective hepatitis, jaundice due to poisons in the blood, pyæmic hepatitis, tuberculous affections, and local pus formation, such as liver abscess. Cerebral symptoms are rarely present, except when a fatal termination is at hand, unless the jaundice occurs in the course of pneumonia, fevers, or in acute yellow atrophy of the liver.

6. EXAMINE THE BLOOD with the van den Bergh test and estimate the icteric index (§ 331).

The *Prognosis* and *Treatment* of jaundice depend on its causal diseases (*q.v.*). The disappearance of bile pigment from the urine indicates that the attack is coming to an end, though it may be some weeks before the skin clears. Plenty of milk, preferably skimmed of excess cream and diluted or citrated, is the staple diet (§ 297, IV); extra amino-acids such as in casein digest or as methionine are advocated but are often nauseous. Fluids must be taken freely, and glucose and insulin given when the liver cells are damaged. The flatulent dyspepsia and many of the concurrent symptoms may be relieved by the administration of extract of

ox-gall (gr. 5 to 15) with meals, together with alkalies and carminatives after meals (formula 66). Calomel is a suitable purgative, followed by salts in the morning. Ammonium chloride, gr. 5 to 15 well-diluted, three times daily is used for the associated ascites, or, better still, injections of mersalyl, 1–2 c.c. The itching of jaundice is often a most troublesome symptom, but it can generally be relieved by pilocarpine or atropine, by calcium salts, or better, by ergotamine tartrate 1 mgm. t.i.d., or by sodium thiosulphate in doses of 10 gr. in saline intravenously; local treatment with alkaline lotions or bran baths, or bathing in potassium permanganate 40–60 grs. to 30 gallons of water, is beneficial. Phenobarbitone helps. Vitamin K and blood transfusion are given for bleeding. Great care is needed to secure efficient sterilisation of needles and syringes after use with jaundiced patients (§ 918).

By the time jaundice appears in a MUNITION WORKER the condition is serious. Symptoms of acute toxæmia may develop, with delirium, coma and death. Prophylaxis consists in strict cleanliness of hands and food, abundance of milk and glucose, and intermission of work in the trinitrotoluol department.

§ 327. **Icterus Neonatorum** is a mild transitory form of jaundice which affects a very large number (estimated by various observers at from 70 to 90 per cent.) of new-born infants. It appears usually on the second or third day of life, is not generally very intense, and rarely lasts longer than one or two weeks. The fæces are normal in colour, and apart from the jaundice the infant presents no other symptoms. The condition is almost certainly due to increased hæmolysis of the red cells. No *treatment* is required.

Some cases of jaundice in the new-born have a much graver prognosis. (1) Icterus Gravis Neonatorum is a severe form which affects several members of a family, and if untreated is fatal in 50–75 per cent. of cases: usually the first and second members of a family are exempt. It is recognised by (i.) being present at birth or within the first 24 hours after birth: (ii.) the accompanying severe hypochromic anæmia, in which nucleated red cells are excessive and persist for 3–4 weeks (erythroblastæmia): (iii.) purpura may develop: (iv.) there is enlargement of the liver and spleen. In cases which recover, damage to the brain may later cause spasticity, athetosis or mental defects (Kernicterus). Recent work shows this condition to be due to the newly-born child having an Rh factor present in its blood, the mother being Rh negative, but forming hæmolysins which destroy the infant's red cells (see §§ 537, 551, V). *Treatment* consists in giving repeated blood transfusions and injections of vitamin K. (2) Congenital syphilis, acting by stricture of the bile-duct or otherwise; or (3) congenital absence of the ducts. Both are usually fatal in a few months: in a small number of cases of congenital atresia, anastomosis of the common bile duct to the stomach or duodenum has been successfully performed. It should be remembered that jaundice in the new-born may be due to (4) sepsis. In stenosis of the ducts the stools are colourless; with sepsis there will be other symptoms.

§ 328. **Acholuric Jaundice.**—There may be no *symptoms*; it is a notable point in connection with the disease that the patients are often able to go about their work as if they were not the subjects of any abnormality. Symptoms when present are jaundice, weakness, a degree of anæmia, and splenomegaly, which may be extreme. These are liable to exacerbations in which the jaundice grows deeper, the anæmia and weakness more profound, and the general malaise may be associated with fever and perhaps vomiting. These attacks seem to be especially determined by an acute infection or by cold. Hæmorrhages from the gums or stomach or into the retina, gall-stones and intractable ulceration of the legs are rarer symptoms. The blood changes are characteristic: the red cells, usually 3–4 millions, are small in diameter

but more globular (spherocytes) and are abnormally fragile—a point which clinches the diagnosis (§ 533). A constant high reticulocyte count (10–50%) is also characteristic. The blood also contains an excess of bilirubin, whereas the urine contains only urobilin. The van den Bergh test (§ 331) shows a strong indirect reaction. The colour of the fæces is normal.

The *Etiology* is not known. The disease may be congenital or acquired. The former occurs in families, and may be transmitted by affected members of either sex. The acquired form is more severe, and spherocytosis and increased fragility are less constant.

The *Prognosis* of the congenital form is good as regards life, though cure is not to be expected. Death may occur during the hæmolytic crises or from biliary tract complications. In the acquired form the prognosis is much worse.

Treatment.—It is important to avoid cold and exposure. In the familial form splenectomy has now been proved successful and alone holds out a prospect of permanent cure. The chief indications for it in the case of a patient hitherto at work are (*a*) increasing and disabling anæmia or debility, or (*b*) frequent or excessive pain. Increased fragility and spherocytosis may persist after the operation.

PART B. PHYSICAL EXAMINATION

The liver lies chiefly in the right hypochondrium; the left lobe extends across the epigastrium above the stomach (Figs. 67, 84).

The gall-bladder is dealt with on p. 435. See Figs. 87 and 88.

The routine methods of examination of the liver consist of INSPECTION, PALPATION, and PERCUSSION. Examination of the urine and fæces and hepatic EFFICIENCY TESTS are necessary in many cases. X-ray examination may assist in the diagnosis of obscure cases—*e.g.*, hydatid and abscess.

§ 329. **Inspection** locally teaches us little, as a rule, unless the symmetry of the abdomen, as observed from the foot of the bed, be altered. However, the presence or the absence of *jaundice* should always be noted. If slight, it may be seen only in the conjunctivæ and urine and on observing by daylight. Deficient expansion of the lower chest is noticed with inflammatory disease of the liver. The lower edge of an enlarged liver may be seen moving with respiration. Note also if there are multiple small *telangiectases* on the skin generally, or dilated venules and capillaries on the face or enlargement of the veins of the abdominal wall, such as occur with cirrhosis and portal obstruction.

Palpation.—All the directions given in § 240 for the palpation of the abdomen must be followed when palpating the liver. The knees should be drawn up and the shoulders supported. Standing on the right side of the patient, place the palmar surface of the hands, previously warmed, on the right side of the abdomen, immediately above the iliac crest, pressing firmly yet gently inwards. The pads of the fingers should be inclined slightly upwards and inwards towards the median line, and should be pressed firmly down, working little by little upwards towards the costal margin. In this way the pads of the fingers, always held perfectly flat, will come in contact with the margin of the organ if it be enlarged. But if it is not enlarged, the liver can only be felt below the xiphisternum, for

laterally it lies altogether beneath the costal margin in the adult. In young children the liver is proportionately larger, and the lower edge normally protrudes beneath the costal margin. If the liver is enlarged, try to feel its surface and consistency by gently dipping the fingers down. Notice if its surface is irregular, smooth (as in fatty liver), or simply rough ("hobnail"), and if it is tender. Umbilicated nodules may be felt in cancer of the liver. When there is fluid in the peritoneal cavity, the method of "dipping" the fingers (suddenly) is also useful; anything but gentle use of the finger tips only excites contraction of the abdominal muscles, and so frustrates our object. Expansile pulsation of the whole liver is felt in cardiac disease with tricuspid regurgitation. The rectum should be examined in all cases of suspected liver disease. For examination of the *gall-bladder* see p. 435.

§ **330. Percussion** should be light so as to elicit only the superficial or absolute dulness of the organ. In percussing the upper margin, begin where there is a good lung note above, and percuss down from rib to rib in the nipple, mid-axillary, and scapular lines. Then repeat the process from space to space. In defining the lower edge, still lighter percussion

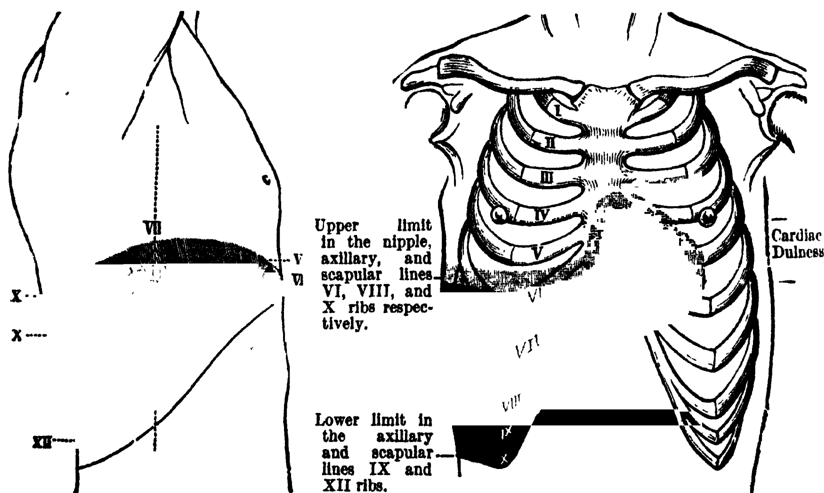


FIG. 84.—AREA OF LIVER AND CARDIAC DULNESS.—The *superficial* (absolute) dulness corresponds to the deep shading; the area of *deep* (or relative) dulness is larger and includes the lighter shading.

should be used, and the examination should proceed from the tympanitic note of the intestine upwards towards the hepatic region. But the more certain method of detecting the lower edge is by palpation.

The normal boundaries of the liver are given in Fig. 84. The *absolute* dulness measures on an average about 2 *inches in the mid-sternal line* and 4 *inches in the right nipple line.*

These landmarks do not indicate the deep dulness of the liver, which is more difficult, and in most cases less useful, to determine. But in some cases, such as

abscess or hydatid, it is desirable to make out the deep (or *relative*) dulness of the liver by heavy percussion. X-ray for diagnosis is not always available.

FALLACIES.—The physician should never feel satisfied with mapping out the liver once only, because the organ may be temporarily affected by many varying conditions, and the *percussion* boundaries by no means always give us a true index. Thus the lower edge may be masked by the dulness of the stomach after a full meal, by an accumulation of fæces in the colon, by a thickened omentum, by great rigidity of the muscles or œdema of the abdominal walls.

Apparent *diminution* of the liver may arise from (i.) distension of the stomach or intestines with gas; (ii.) by contractions of Glisson's capsule, especially on the under surface, giving rise to puckering or distortion of shape anteriorly; or (iii.) emphysema of the lungs, which obscures the *upper* border very much. Great diminution or absolute loss of the liver dulness, owing to gas in the peritoneal cavity, is a diagnostic feature of perforation of the stomach or intestine.

Apparent *enlargement*, when attention is paid solely to the lower edge of the organ, may be due to a *displacement* of the liver downwards by (i.) pleural effusion, empyema, or pneumothorax; (ii.) intrathoracic tumours; or (iii.) enlargement of the heart or hydro-pericardium. These and other fallacies may arise from paying attention solely to the *lower edge* of the organ; and, finally, the liver may in rare cases be dropped or "*floating*." "Riedel's lobe" is mentioned under Abdominal Tumours. Tumour or enlargement of the *gall-bladder* may be percussed as a dulness extending down from the liver towards the umbilicus.

Fluid in the Peritoneum (Ascites) is a frequent accompaniment of some hepatic disorders, and its presence or its absence must always be carefully noted. The methods of investigating Ascites have already been given (§ 259 and § 260), in which PORTAL OBSTRUCTION is dealt with in full.

§ 331. Liver Function Tests. As stated in the introduction to this chapter, the liver has numerous functions and these may vary independently. There is a wide margin of reserve, so that 70% of the liver may be put out of action before signs of insufficiency occur. Some of the functions may be controlled quantitatively and thus the so-called liver function tests are made.

(1) Sugar Tests: According to the patient's weight, 30–50 G. of lævulose are given in water, and the blood sugar examined before the test, and at half, one and two hours after it. With a normal liver the blood sugar curve should show no rise above the fasting level greater than 30 mg. per 100 c.c.; any higher rise in blood sugar indicates liver insufficiency. The appearance of sugar in the urine is not so useful; lævulosuria and galactosuria, after taking these sugars, occur in 70–80 per cent. of cases of diffuse liver disease, but also in 10 per cent. of the normal. Diabetes mellitus invalidates the test.

(2) By means of the *van den Bergh test* small amounts of bile pigment (bilirubin) in the blood can be detected, even before any clinical sign of jaundice is evident.

For the VAN DEN BERGH TEST two solutions are required. (1) Concentrated HCl 15 c.c., sulphanilic acid 1 c.c., distilled water 1000 c.c. (2) Sod. nitrite 0·5 G., distilled water 100 c.c. Mix 25 c.c. of (1) with 0·75 c.c. of (2). For the *direct reaction*, 1 c.c. of blood serum is mixed with 1 c.c. reagent. A blue violet colour begins at once and attains its maximum in 10 to 30 seconds. The colour change may be delayed for 1–15 minutes or even longer. It may be reddish at first, slowly or rapidly changing to blue (*biphasic*).

The *indirect reaction* is also tested with serum. Add 2½ c.c. of 96 per cent. alcohol and 1 c.c. saturated ammonium sulphate and centrifugalise. A violet red hue results, rapidly attaining its maximum.

For the *quantitative estimation* of bilirubin, the colour reaction is compared in a colorimeter with a standard solution of cobalt sulphate in water or of methyl red in acetic acid: in normal persons the value is not above 0·5 mgms. (1 unit). It is of value in detecting latent jaundice, and in assessing the progress of a case of manifest jaundice. In the interpretation of the results, it is now recognised that neither the direct van den Bergh reaction nor its modifications enables the distinction to be made between hepatogenous jaundice and that due to obstruction. Even in hæmolytic jaundice the test may be unreliable owing to the presence of intercurrent damage to the liver cells.

(3) the *icteric index* is a measure of the yellow colour in the serum, which is mainly due to bilirubin. A solution of potassium chromate is used for comparison. The normal is between 4 and 6. Clinical jaundice gives values above 16, latent jaundice between 6 and 16. This test is not so reliable as the van den Bergh reaction.

(4) The excretory function of the liver may be tested by giving intravenous bilirubin, or rose Bengal, and noting the time of disappearance from the blood. Bromsulphthalein in doses of 2–5 mgm. per kilo of body weight is a reliable test.

(5) *The Quick (Hippuric Acid) Test:* The body contains no store of glycine, which is formed entirely by the liver. This is conjugated with benzoic acid to form hippuric acid. *Method.*—Sodium benzoate (5·9 G. in 30 c.c. water) is given by mouth as the bladder is emptied. The urine is collected at the end of four hours and the hippuric acid precipitated from it by conc. HCl, dried and weighed: normally 3·0 G. (as sodium benzoate) is excreted, the results being expressed as a percentage of this figure. An intravenous method may be used in cases of vomiting, 20 c.c. of 10 per cent. solution (2 G.) of sodium benzoate being injected intravenously.

(6) Changes in the *plasma proteins* are noted. In liver disease the albumin is diminished and the globulin increased, so that a diminution of the albumin-globulin ratio occurs: this is in proportion to the severity of the liver damage.

(7) *Serum alkaline phosphatase* is increased in obstructive jaundice; flocculation tests in the serum are increased in non-obstructive jaundice.

(8) In all cases THE URINE SHOULD BE EXAMINED for bile, and sometimes for urea, leucin and tyrosin. The ammonia-urea ratio is an important test. One of the main functions of the liver is to convert ammonium salts into urea. If the liver function is inadequate, the formation of urea is decreased, and the resulting excess of ammonium salts and diminution of urea in the blood is reflected in the urine. The normal ratio of ammonium salts to urea is determined in a twenty-four hours' specimen of urine, and is 4 per cent. In liver insufficiency, this may rise to 30–40 per cent. This estimation is rapidly performed and is a useful index by which to gauge from day to day the effect of treatment.

X-RAY EXAMINATION may reveal abscess, tumour, irregularity, enlargement or diminution of the liver. Gall-stones are dealt with in § 353.

PART C. DISEASES OF THE LIVER

Routine Procedure.—FIRST: Ascertain *what is the patient's Leading Symptom.* The symptoms of disorder of the liver we discussed in Part A. —*e.g.*, gastric disturbance, pain (or a feeling of weight or discomfort in the hepatic region), and jaundice. If there be severe and paroxysmal pain, turn first to biliary colic (§ 353).

SECONDLY: Learn the *History* of the patient's illness, eliciting the

facts in chronological order, and in this way ascertain also whether the disease be *acute* or *chronic*.

THIRDLY: THE EXAMINATION OF THE LIVER must next be made. The routine method is given in §§ 329 and 330.

Ascertain: 1. Whether the liver is *enlarged*, locally or generally, or *diminished* (by abdominal palpation and percussion in the nipple line), and whether there is any *pain*, *tenderness*, or other abnormality; 2. Whether there is any *fluid* in the peritoneum; 3. If there is any *jaundice*. 4. Examine *the urine* for bile pigments, urates, etc. 5. In certain cases the *liver function tests* and *X-ray examination* must be carried out.

Classification.—For clinical purposes, diseases of the liver may be conveniently divided into ACUTE and CHRONIC Disorders.

If the illness is one of long standing, and has come on insidiously, the reader should turn to **Chronic Diseases of the Liver** (§ 340).

ACUTE DISEASES OF THE LIVER

If the illness has come on more or less suddenly, and is attended by considerable malaise or other constitutional symptoms, it is one of the **acute diseases of the liver,** probably: I. ACUTE OR SUBACUTE HEPATITIS: I*a*. INFECTIVE HEPATITIS; I*b*. ACUTE OR SUBACUTE YELLOW ATROPHY; Ic. WEIL'S DISEASE. The less common acute diseases are: II. PERIHEPATITIS; III. ABSCESS; IV. ACTINOMYCOSIS; V. DISTOMIASIS.

I. *The patient, after a short spell of* MALAISE, VOMITING *and* DIARRHŒA, *becomes* JAUNDICED. *The disease is* ACUTE *or* SUBACUTE HEPATITIS, *and is due to interference with the function of the liver cells, due to bacterial, chemical or protozoal toxins. It may be mild* (CATARRHAL JAUNDICE), *or severe* (ACUTE YELLOW ATROPHY), *or due to* WEIL'S DISEASE.

§ **332.** I*a*. **Acute Infective Hepatitis**, previously known as *Catarrhal Jaundice*, occurs in sporadic or epidemic fashion, particularly in the autumn. It is milder in children than in adults, and one attack usually confers immunity.

Symptoms usually begin with a *pre-icteric stage*: (1) Anorexia is constant, and all solid food is refused for 2–3 days. (2) Nausea usually accompanies but vomiting is unusual. (3) Frontal headache, malaise and disinclination for any work are usually present. (4) A feeling of uneasiness or weight in the epigastrium is sometimes accompanied by actual pain. (5) Constipation is much more common than diarrhœa. (6) There is fever, sometimes only to 99°, more usually to 101° and occasionally higher for 3–4 days: this gradually settles to normal as the *icteric phase* is reached. (7) Jaundice occurs between 1 and 8 days from the start of the illness. It is accompanied by pale stools, bile-stained urine and often by pruritus. The depth of the jaundice is very variable, and so is its duration, which may be as short as a week, or may persist for even 2 months. (8) At the onset of jaundice the temperature usually settles to normal and the appetite simultaneously returns. (9) The liver is often enlarged and firm,

1–2 fingers' breadths below the right costal margin, and the spleen is usually palpable. (10) Occasionally skin rashes of macular, urticarial or even purpuric type occur. (11) The van den Bergh test gives at first a biphasic and later an indirect positive result. (12) Leucocytosis never occurs and a polymorph leucopenia is usual.

During the course of an epidemic, cases may reveal only the symptoms of the pre-icteric stage, and jaundice may never develop: even the serum bilirubin is not necessarily raised in such cases.

Etiology. Three related varieties are recognised. (1) *Primary infective hepatitis*, in which an agent (probably a virus), is transmitted from an infected person by droplet infection from the nose, and from the fæces and urine. The incubation period is usually 17–35 days, and more than one member of a family may be involved. (2) *Homologous serum jaundice* occurs when human serum containing an icterogenic agent is used for blood transfusion, or to convey immunity against measles, mumps or yellow fever. Convalescent serum from cases of measles and mumps, and pooled human serum used in the preparation of yellow fever vaccine, have produced liver damage, without or with jaundice, in a large number of subjects, after an incubation period of 56–239 days (average 101 days). This type of hepatitis has been transmitted from the serum and from the nose of persons in the pre-icteric and early icteric stages, to volunteers who were inoculated subcutaneously or intranasally. (3) *Post-arsphenamine jaundice* rarely occurs in the first two weeks after the initial dose, and this is probably chemical in origin. Much more often an icterogenic agent is transmitted by a syringe contaminated by blood from a previous patient, and produces hepatitis 12–19 weeks later: this agent is not readily destroyed when syringes are "sterilised" (§ 918), and can be transmitted to a third person by the subcutaneous inoculation of infected serum. Jaundice subsequent to the injection of gold salts and other chemicals may be of similar origin.

Whereas an attack of infective hepatitis provides almost complete immunity against a second attack, post-vaccinal yellow fever jaundice does not confer immunity against a subsequent attack of infective hepatitis, suggesting that infective hepatitis and homologous serum jaundice are due to related but separate agents: it is probable that the causes of homologous serum jaundice and post-arsphenamine jaundice are similar to one another, even if they are not identical.

Diagnosis. In *infective hepatitis*, pain is never severe and is often absent, bile is absent from the stools only for a short time, and they then contain bile but are pale, the spleen is often palpable and the gall-bladder is never enlarged: leucopenia is the rule. In *gall-stones* the onset is usually with severe biliary colic, jaundice is often deep and the stools persistently clay-coloured: the spleen is not palpable. *Cancer* occurs in middle-aged or elderly persons; jaundice is often insidious in onset and lasts for many months. Jaundice following acute infections and other abdominal inflammation may be due to *abscess of the liver*. *Post-arsphenamine jaundice*, and those varieties following *blood transfusions* and *human serum inoculations*, give a history of the cause.

Prognosis. Most cases clear up completely, but there is a danger of relapse: the malady usually terminates within 6–8 weeks. Rarely cirrhosis of the liver may ensue.

Treatment. The patient should be kept warm in bed for at least 2 weeks. Until appetite returns, the diet should consist mainly of milk and carbohydrates, avoiding irritating substances and fats. A large

amount of fluid should be drunk, and a rectal drip of saline may be given with 5 per cent. glucose. Insulin, 5 units, may be injected twice a day, and glucose taken by mouth. Methionine and casein digests do not benefit cases of infective hepatitis, but may modify attacks of post-arsphenamine jaundice. A brisk mercurial purge, followed by a saline twice a week, relieves the congestion of the intestines and the liver. To prevent relapse, patients should not be allowed to return to work until the indirect van den Bergh in the blood is under 1 unit.

§ 333. Ib. **Acute or Subacute Yellow Atrophy** (Severe Acute Hepatitis, Icterus Gravis) is a rare disease characterised by extensive necrosis of the liver cells, jaundice and cerebral symptoms, occurring especially in workers with trinitrotoluol, and usually ending fatally. Some cases are due to a severe form of infective hepatitis and presumably to a virus. A very severe form is associated with toxæmia of pregnancy.

Symptoms.—(i.) The premonitory symptoms may be slight, resembling infective hepatitis, and are associated with temporary enlargement of the liver. There is increasing tenderness over the liver. (ii.) In a few days or weeks severe symptoms set in, with deepening jaundice, headache, and delirium, and the patient passes into the typhoid state (cholæmia). (iii.) Hæmorrhages occur from the stomach, bowel, and kidney, and there may be petechiæ under the skin. (iv.) Fever is usually absent during the course of the illness, but at the end it may be high. (v.) With the onset of the severe symptoms the liver dulness begins to diminish rapidly. The spleen is usually enlarged. (vi.) The urine is characteristically altered, containing bile, albumen and blood, and showing diminished uric acid and urea, with increase of the ammonia coefficient and sometimes acetone. Leucin and tyrosin are sometimes found crystallising out on evaporating a few drops of urine (Fig. 99). (vii.) In the most severe cases, as in the toxæmias of pregnancy, early collapse, with tachycardia, prostration and death may take place before jaundice is apparent.

Diagnosis.—Acute Yellow Atrophy is not likely to be mistaken for any other liver disease after the acute symptoms set in. In phosphorus poisoning the liver is enlarged, and signs of irritant poisoning precede the onset of the jaundice.

Etiology.—*Predisposing Causes.*—(i.) Acute Yellow Atrophy is most common under middle age, though rare in children ; and (ii.) in women, especially during pregnancy, often accompanying eclampsia. (iii.) Workers in trinitrotoluol and carbon tetrachloride. (iv.) Dissipation and excesses predispose. *Exciting Causes.*—(1) It may complicate fevers, such as typhoid fever, streptococcal infections, and influenza. It is found in (2) delayed chloroform poisoning and poisoning with phosphorus or cinchophen (atophan) ; and (3) in some cases of secondary syphilis. It is more frequent since the introduction of intensive treatment with arsenical preparations (see post-arsphenamine jaundice, § 332). (4) It may follow the passage of a gall-stone. (5) In a large number of cases no cause can be found, but a virus infection is suspected.

Prognosis.—The disease is often fatal. After the severe symptoms set in the patient may die in a comatose condition within a week. Pregnant women usually abort. Recovery may take place, followed by cirrhosis of the liver (interstitial hepatitis).

The *Treatment* is very unsatisfactory. During the preliminary stage the disease is treated as under infective hepatitis (§ 332). Warm baths, diaphoretics, rest, milk food, large doses of bicarbonate of soda and glucose, and diuretics may be tried. In all cases of syphilis under treatment with arsenic preparations intravenously, the urine and, if necessary, the blood should be watched carefully for the presence of bile pigments, and the treatment intermitted if they are found. Intravenous injections of sodium thiosulphate and the use of B.A.L. (p. 718) may be successful in cases due to arsenic. Insulin subcutaneously, combined with glucose by the mouth or intravenously, may help recovery.

§ 334. Ic. **Weil's Disease** (Synonyms: Spirochætal Jaundice, Spirochætosis Icterohæmorrhagica, Leptospirosis) has a sudden onset associated with fever, toxæmic

symptoms, and, in severer cases, with jaundice and renal involvement. The incubation period varies from 6 to 12 days. The onset is with rigor, headache and frequently vomiting, followed by backache, joint pains, tenderness of the muscles and severe prostration. Sore throat is common. The blood pressure is low, the tongue furred, the face flushed and the conjunctivæ injected. The temperature generally oscillates between 102° and 104° F. and then begins to fall by lysis: it is generally 7 to 14 days before the temperature is normal. Jaundice appears from the 4th to the 6th day in about 50 per cent. of cases and may be intense.

The stools are light or even clay coloured. The urine is scanty, contains bile pigment and bile salts, albumin and sometimes red blood corpuscles. The van den Bergh reaction often gives a direct biphasic reaction and bilirubinæmia is increased. The blood urea is raised to 60–300 mgm. per 100 c.c. A leucocytosis is the rule, varying from 12,000 to 30,000 per c.mm., with neutrophil polymorphonuclears increased to 80 per cent. Skin petechiæ, epistaxis, melæna and hæmorrhage from other mucous membranes are not infrequent, and herpes is common.

The outstanding feature of the physical examination is the extreme tenderness of the muscles, especially those of the legs, neck and abdomen. Abdominal rigidity may suggest an acute abdomen. The liver is generally enlarged and tender and splenomegaly may be present. Later, nocturnal delirium, a typhoid state, muscular twitchings and convulsions may develop and the patient die with anuria and uræmia. In other instances cholæmia, associated with increasing jaundice, hiccough, Cheyne-Stokes' respiration and coma, terminates the picture. Meningeal symptoms predominate in some cases; the cerebrospinal fluid then contains polymorph leucocytes, lymphocytes, increased quantities of globulin and sometimes also leptospiræ. In about 50 per cent. of cases jaundice does not appear and kidney involvement is slight; the fever may only last 2 to 4 days, and unless the occupation of the patient suggests the need of laboratory investigations the condition will be missed.

Diagnosis.—Features of importance include an occupational relationship to rats or submersion in infected water, profound prostration, extreme tenderness of the muscles, jaundice appearing about the 5th day, hæmorrhages, albuminuria and leucocytosis. At the onset, *meningitis*, later, *infective hepatitis*, may be suspected: the latter does not cause leucocytosis. In the tropics *yellow fever* and *relapsing fever* complicated by jaundice may prove confusing. During the first week leptospiræ can be demonstrated by blood cultures or by the intraperitoneal inoculation of blood into guinea-pigs. The agglutination test becomes positive in the second week and from the third week onwards leptospiræ can be isolated from the urine.

Etiology.—Rats are carriers of the disease, the causative organism—*Leptospira icterohæmorrhagiæ*—being passed in the urine and so infecting water and fungal slime. Human beings are infected during bathing or immersion accidents or through abrasions of the skin; hence canal workers, bargemen, rat catchers, coal miners, sewer workers, fish curers and farm workers are liable.

Treatment.—Anti-leptospiral serum (60 c.c. or more) prepared from immunised horses or 30 c.c. of serum obtained from convalescents should be administered intravenously as early as possible. Intravenous dextrose solution (5 per cent.) combined with insulin is of special value. Penicillin, 40,000 units three-hourly for 4 days, should be given as early as possible—leptospiræ are very sensitive to this drug.

Preventive treatment consists of rat destruction, avoidance of bathing in infected water and the protection of skin abrasions in workers whose occupation brings them in contact with rat-infected slime.

The less common **Acute Disorders** of the Liver remain to be considered, viz., PERIHEPATITIS and ABSCESS OF THE LIVER.

II. *The patient complains of* PAIN AND TENDERNESS *in the hepatic region, aggravated by movement. There is* NO JAUNDICE, *and other hepatic symptoms are absent.* The malady is probably PERIHEPATITIS.

§ 335. **Perihepatitis** is inflammation of the capsule of the liver, which becomes opaque and thickened (sugar-loaf liver), and by its contraction may lead to considerable distortion of the shape of the liver.

Symptoms.—(i.) Acute attacks usually set in suddenly, with pain in the hepatic region, radiating to the shoulder, and there is tenderness, increased on movement, pressure, or cough. (ii.) Fever is absent as a rule, and the patient may appear to be in his usual health. (iii.) Friction may be felt or heard. (iv.) Unless some other disease is present, there is no jaundice. Recurrent attacks lead to thickening of the capsule, recurring ascites, necessitating repeated tapping, and occasionally jaundice. The puckered liver, with its thickened, rounded, distorted edge, can sometimes be made out. The history of a *Cause*, especially *syphilis*, is usually obtainable. It is sometimes part of an inflammation of the liver itself, or is associated with an abscess, tumour, or cirrhosis. Sometimes the inflammation extends from adjacent organs, as in pericarditis, pleurisy, or gastric ulcer, or it may be part of a general peritonitis.

Diagnosis.—The characteristic pain and the absence of jaundice differentiate it from many other liver diseases. Other signs of syphilis aid diagnosis. Cases of gall-stones or gumma of the liver may at times be mistaken for perihepatitis.

Prognosis.—Simple cases tend to recover. In those which have lasted for a long time a certain amount of cirrhosis of the liver ensues. Portal obstruction may ultimately result from puckering at the fissure, and considerable distortion of the liver may result in the same way.

Treatment.—The diet must be spare, and the patient must be kept warm. Salines are given, with blue pill and rhubarb. Externally, hot fomentations and poultices give relief, and if the pain is severe, leeches are recommended. The cause when known must be treated—*e.g.*, syphilis (§ 552).

III. *There is* ENLARGEMENT *of the liver, accompanied by* PAIN *and tenderness, and the boundaries of the area of dulness are* IRREGULAR ; *there are* SHIVERINGS, SWEATING, *and* INTERMITTENT PYREXIA. The disease is ABSCESS OF THE LIVER.

§ 336. **Abscess of the Liver.**—Solitary or multiple collections of pus may occur in the liver, due to septic infection, to suppuration of the bile channels (cholangitis), or portal vein (portal pyæmia), or more rarely to suppuration of pre-existing morbid conditions, such as hydatids or gummata. "Tropical" abscess occurs after amœbic infection of the colon, a common cause in the tropics ; it is usually solitary, whilst pyæmic abscesses are generally multiple.

Symptoms.—(i.) The onset is usually *acute*, except in the tropical form, with pain and tenderness of the liver, accompanied perhaps by a dry cough, shallow respiration and digestive disturbance. The pain is affected by respiration, and may be worst when the patient lies on the left side. (ii.) The liver is enlarged, and the enlargement may extend downwards, or more often upwards, even to the nipple. There may be fluctuation. (iii.) Jaundice is rarely present. (iv.) Constitutional symptoms are severe. There is usually high fever, continuous at first, then with increasing oscillations. Rigors and sweats are common. Later, the patient falls into the typhoid state, with emaciation, vomiting, diarrhœa, and delirium.

Besides the acute type just described, there is a variety with an *insidious* onset. As amœbic hepatitis gradually develops into frank abscess formation, there is general failure of health, and periods of continuous, remitting or intermitting fever, sometimes followed by intervals of apyrexia. Cough and dull aching over the liver and in the right shoulder may be present from the beginning. *Amœbic abscess of the liver* generally affects the right half of the liver and is usually associated with physical signs at the base of the right lung ; there may be a history of dysentery, while cysts of *Entamœba histolytica* may or may not be found in the fæces. Compensatory hypertrophy of the left half of the liver commonly occurs in destructive lesions implicating the right side.

Diagnosis.—(i.) The pain and pyrexia distinguish abscess from *hydatid* (when not in a suppurating condition). (ii.) A distended and *inflamed gall-bladder* may be palpable. Suppurative pylephlebitis, septic cholangitis, hepatic carcinoma and a breaking down gumma may simulate abscess. (iii.) Abscess is often mistaken for severe *malaria*. But malaria is amenable to quinine, the elevations of temperature are periodic, and each paroxysm has three stages. (iv.) Hepatic abscess may be diagnosed from other swellings of the liver by exploratory aspiration, revealing the chocolate "anchovy sauce" thick, tenacious pus. (v.) Physical signs suggestive of collapse or consolidation at the base of the right lung so frequently accompany liver abscess that their presence is an important aid to diagnosis. (vi.) X-ray shows upward enlargement of the liver (when the right lobe is involved), limited movement of the diaphragm, and sometimes local bulging due to a pointing abscess.

The insidious cases of liver abscess are always difficult to diagnose. No history of dysentery may be obtained and several examinations of the stools may be negative. In amœbic hepatitis there is often more fever than the total white count would suggest. With amœbic abscess the counts mostly range from 12,000 to 16,000 per c.mm. the polymorphonuclears 65 to 75 per cent. Where secondary streptococcal or staphylococcal infection supervenes, the leucocytes rise to 20,000–30,000 per c.mm. and the neutrophil polymorphonuclears equal 80–90 per cent. of the total cells ; these counts also hold for pyæmic abscesses. Always suspect hepatic amœbiasis in a patient with obscure pyrexia coming from a tropical country.

Etiology.—Hepatic abscess, single or multiple, may arise from—(i.) Suppuration in a pre-existing focus of disease—*e.g.*, hydatid, gumma, tuberculous abscess, actinomycosis, or malignant growth ; (ii.) ulceration of the biliary passages such as occurs in cholecystitis ; (iii.) ulceration of the alimentary canal. The abscesses are usually multiple, except in amœbic dysentery, where often one large abscess, consisting of necrotic liver tissue which is sterile except for the presence of the amœbæ, dominates the picture ; such an abscess may become secondarily infected with streptococci, etc. (iv.) Inflammation and pus-formation in the abdomen, especially in cases of old-standing suppuration of the pelvic organs and in appendicitis. (v.) Occasionally operations on the rectum or in any septic area produce an abscess in the liver, consequent on the conveyance of a septic embolus by the portal vein. (vi.) Portal pyæmia. (vii.) Trauma in a few cases.

Prognosis.—(1) The case mortality is high, except in tropical liver abscess. Death usually takes place in three weeks in cases with multiple abscesses. The pyrexia increases, and the patient dies in the typhoid state. The abscess may burst into the peritoneum, pericardium, or alimentary canal, with a fatal issue, or it may open externally and gradually heal by free discharge. Frequently the abscess, especially a "tropical" abscess, bursts into the right lung or the pleura. The patient develops a severe cough, with signs of consolidation of the right pulmonary base, and the abscess contents are brought up as a red-coloured sputum. Recovery may result, or the continued discharge may lead to death from exhaustion or lardaceous disease.

Treatment.—For multiple pyogenic abscesses, penicillin 50,000 units three-hourly subcutaneously and sulphadiazine 1 G. four-hourly by mouth should be tried. Where amœbic hepatitis (§ 517) or abscess is suspected give injections of emetine hydrochloride gr. i. daily for a period not exceeding 10 days. Absolute rest in bed is necessary. If the condition does not clear up, exploratory puncture of the liver should be made under local anæsthesia ; if pus is discovered it should be aspirated and aspiration should be repeated several times if needed. Incision and drainage is performed only when secondary bacterial infection is present ; penicillin and sulphadiazine should be given as well as emetine. If the stools still contain cysts, the treatment outlined for amœbiasis should be instituted (§ 308 (2)).

§ 337. IV. Actinomycosis of the Liver is a condition which may be mistaken for abscess of the liver. It is due to absorption of the ray fungus from the intestines, and starts as one or more foci in the liver substance, which slowly enlarge and may undergo suppuration.

The *Symptoms* consist of vague uneasiness referable to the liver, with gradually increasing enlargement—at first uniform, later unequal, the organ becoming prominent in one place. Exploration with a needle may yield no results; but if the tumour is laid open, the characteristic greenish fluid with yellow specks containing the ray fungus clinches the diagnosis. Actinomycosis appears to respond favourably to large doses of penicillin (Table XXX); otherwise maximal doses of potassium iodide (gr. 40–60 daily) should be given.

§ 338. V. **Distomiasis of the Liver** is commonly found in the Far East, due to *Clonorchis sinensis*, while more rarely *Fasciola hepatica*—the sheep fluke—may affect man. In these diseases the bile ducts are invaded with flukes, leading to thickening and dilatation of the ducts and cirrhosis of the liver. Mild infections may be symptomless, but the more severe cases present anorexia, epigastric pain, hepatomegaly, diarrhœa, wasting, œdema, ascites and jaundice. Secondary bacterial infection may produce fatal cholangitis or liver abscess. The *Diagnosis* is made by finding the operculated eggs in the fæces, associated with eosinophilia.

Treatment.—Carbon tetrachloride 3 c.c. in a gelatine capsule is sometimes effective; and favourable reports have been recorded as to the value of emetine injections (1 gr. daily for 10 days). A course of antimony sodium tartrate or organic compounds of antimony may be given intravenously.

§ 339. **Subphrenic Abscess.**—The *Symptoms* resemble those of tropical liver abscess. When occurring above the right lobe, the liver dulness is continued up in the axilla, perhaps as far as the level of the nipple, and is convex, or dome-shaped, upwards. The base of the right lung shows signs of congestion, and there are evidences of pleurisy at one or both bases.

Etiology.—The most common causes are appendicitis and ruptured peptic ulcer. Other causes are extension of hepatic abscess, empyema perforating the diaphragm, extension of pelvic abscess, and local tuberculous or (rarely) cancerous processes.

Diagnosis.—In a case of suspected abscess exploratory puncture may be performed, sometimes under general anæsthesia. The needle should not penetrate beyond 3½ inches, so as to avoid puncturing the portal vein. In a right-sided *empyema* of the chest the upper border of the dulness, when continuous with that of the liver, is concave, being higher towards the spine. In *hepatic abscess* the liver is tender and enlarged below the costal margin, but it is often impossible to distinguish subphrenic from hepatic abscess. A variety containing air so greatly resembles pneumothorax that it is called *pyopneumothorax subphrenicus.*

The *Prognosis* is fair if surgical treatment is carried out thoroughly and in time.

CHRONIC DISEASES OF THE LIVER

§ 340. **Routine Procedure.**—It will be remembered (§ 329) in the physical examination of a patient suspected to be suffering from hepatic disease that the *first* and most important question to investigate is whether there is *any alteration in size,* especially enlargement *of the liver* (by palpation and percussion). (2) For reasons which will be apparent below, the question next in order of importance is whether there is any *pain or tenderness* in the organ. And then (3) is there any *jaundice?* (4) Is there any *ascites?* (5) In every case of suspected liver disease the spleen (§ 356), the stools, and the urine should be carefully examined.

The numerous *fallacies* in the alteration of the size of the liver dulness must be carefully studied (§ 330).

Classification.—Chronic diseases of the liver are divided into those in which the **size of the liver is unchanged** and those in which it is altered,

either **diminished or enlarged**; the latter again being divided according to the presence or absence of pain over the liver.

A. The organ is **Normal in size** in :—
Functional derangement of the liver .. .. .. § 341
B. The organ is **Diminished in size** in :—
Portal cirrhosis or Chronic hepatitis .. .. .. § 342
C. The organ is **Increased in size** :—

(*a*) Without pain or tenderness :
I. Hypertrophic cirrhosis (bacterial and toxic) ; I*a*. Biliary cirrhosis ; I*b*. Cardiac valvular disease ; I*c*. Chronic syphilitic disease ; I*d*. Cirrhosis of biliary obstruction ; I*e*. Tropical cirrhosis .. .. § 343
II. Fatty Liver .. .. § 344
III. Von Gierke's disease .. .. § 345
IV. Lardaceous liver .. .. § 346
V. Hydatid of liver and other rare conditions .. .. § 347

(*b*) With pain or tenderness :
I. Chronic congestion .. .. § 348
II. Cancer of liver .. .. § 349
III. Abscess of liver, tumours and other rare conditions occurring sometimes in acute form .. .. §§ 336, 350

A. *The liver is* **normal in size.** *The patient complains of* LETHARGY, *vague digestive disturbances, sleepiness after meals, furred indented tongue,* CONSTIPATION, *headaches, and there is a frequent deposit of* URATES IN THE URINE *on cooling.* There is probably FUNCTIONAL DERANGEMENT OF THE LIVER.

§ 341. Functional Derangement of the Liver.[1]—The liver is the largest gland in the body and has many functions, which may become deranged together or separately. Usually one or two functions are more severely affected. See liver tests (§ 331).

The common complaint, "My liver is sluggish," is often equivalent to saying that the bowels do not act properly, but in some cases other parts of the digestive system may be at fault. The causes of this complaint may be temporary or continuous. Help may be obtained by consulting the following classification.

I. Temporary :
Acute dyspepsia, "bilious attack" (§ 281).
Migraine (§ 696).
Onset of colds, febricula.
Excess of food, alcohol, or exertion (§ 282).

Errors of diet, especially rich, sweet, greasy foods, and alcoholic beverages, *i.e.,* indigestible and excessive food rather than food with

[1] The introductory remarks at the head of this chapter may be referred to in this connection.

purin bodies. Alcohol *combined with sugar* (*e.g.*, port and other fruity wines) is especially injurious; or taken in the form of undiluted spirit, *e.g.* cocktails, particularly on an empty stomach, is more harmful than dilute alcohol at meal-times.

II. CONTINUOUS:

Constipation (§ 317).	Gastritis (§ 284).
Cholecystitis (§ 354).	Colitis (§§ 307, 310).
Chronic appendicitis (§ 249).	Acidosis (§ 384).

B. *The liver is* **diminished in size**; *if the surface can be felt it is* HARD AND UNEVEN (*hobnail*); ASCITES *is probably present, but no very distinct jaundice; the patient is subject to* HÆMORRHOIDS, *and* HÆMORRHAGES *from the stomach and bowel.* The disease is PORTAL CIRRHOSIS (CHRONIC INTERSTITIAL HEPATITIS).

§ **342. Portal Cirrhosis of the Liver** (Alcoholic Cirrhosis or Interstitial Fibrosis of the Liver) consists of a chronic hepatitis, with an increase of the interstitial fibrous tissue, leading to portal obstruction, and shrinkage of the organ. In the earlier stages a degree of fatty degeneration may give rise to considerable enlargement (§ 344), but later, with the formation of fibrous tissue, atrophy ensues. It is most common in men between 35 and 60 years old.

Symptoms.—(1) In the early stage of the disease the organ may be enlarged, though rarely much so; but in the second and third stages the liver dulness is diminished. The liver is small and hard, and the surface is often nodulated, hence it is known as the "hobnail," or "gin-drinkers'" liver. There is a feeling of uneasiness and weight in the hepatic region. (2) The onset is slow and insidious, extending sometimes over years. Gastric symptoms, such as capricious appetite, *morning sickness*, and other symptoms of alcoholic dyspepsia, are often complained of for a considerable time, together with nervous disturbances, such as drowsiness, muscle pains, especially in the neck, and headache after meals. These symptoms of chronic gastritis are followed by debility and emaciation. The patient's aspect is very characteristic, with dilated venules and capillaries in the cheeks. (3) Jaundice appears in the later stages of the malady in about one out of three cases. (4) Symptoms of portal obstruction occur (§ 260), and hæmatemesis is sometimes the first obvious symptom; the spleen becomes slightly enlarged, and ascites (which is present in 80 per cent. of the cases) may be very considerable in amount: when the ascites recurs after paracentesis, chronic peritonitis has probably supervened. (5) There is an increased tendency to hæmorrhage. (6) In the concluding stages of this disease, when the secreting tissue of the liver is destroyed, the patient falls into a comatose state, with muttering delirium (cholæmia), which resembles uræmia and the typhoid state, except that there is considerable pyrexia in the latter. This precise clinical resemblance is quite in keeping with the fact that the liver takes

part in the elaboration of urea, so that when its cells are destroyed the blood becomes charged with a number of nitrogenous products, which cannot be eliminated.

Etiology.—(1) Cirrhosis of the liver is due to chronic intoxication by poisons which are usually of exogenous origin. It affects men more often than women. (2) Alcohol undoubtedly predisposes to atrophic cirrhosis, especially when taken frequently in small quantities, or when taken *neat on an empty stomach,* the patient perhaps never becoming intoxicated. (3) Cirrhosis can occur in those who have never consumed alcohol: dietetic factors may play a part, and in Southern India toxic products from tapioca have been suspected. Syphilis, malaria, bilharzia and many bacterial infections may predispose. (4) In poisoning by T.N.T., carbon tetrachloride and tetrachlorethane, the process is subacute or even acute. (5) Splenic anæmia (Banti's disease, § 544).

Diagnosis.—*Cancer* of the liver is only difficult to diagnose from cirrhosis in the early stages; but usually it runs a more rapid course, and is accompanied by more pain, and more intense jaundice. The spleen is not usually enlarged in cancer. In *passive congestion* of the liver with ascites there are evidences of a cause, such as heart or lung disease. In the absence of ascites early cirrhosis may be mistaken for the other causes of liver enlargement. The enlargement of the spleen in atrophic cirrhosis may lead to the primary condition being overlooked. The liver is reduced in size in *starvation. Chronic peritonitis* with effusion may not be recognised as such until the organs can be palpated after paracentesis.

Prognosis.—The disease has a slower and more insidious onset than hypertrophic cirrhosis (below), and is in most cases a more serious condition. If the patient is seen before signs of portal obstruction supervene much can be done; if later, the prognosis is grave. The outlook is more favourable in patients who are young and where the general health is good. *Untoward Symptoms.*—Although restoration to comparative health has occurred after the development of ascites, with the onset and recurrence of rapid ascites the end is in view, the patient rarely living more than a few months. Pleurisy, renal disease, or tuberculous peritonitis are occasional complications.

Treatment in the early stages is practically the same as that employed for chronic congestion of the liver, and chronic gastritis (§§ 348 and 284). The habits of the patient must be corrected, and the diet reduced to the simplest elements; milk should be the staple food in advanced cases. A high-protein and a low-fat diet is best (§ 297, IV.): methionine and choline chloride (2 G. āā daily) have been advocated. Glucose and insulin aid. Alcohol must be avoided, and regular exercise taken. Treatment with liver extract, by mouth or by injection, has been tried with some success. A course of salines should be taken in the early morning, and rhubarb or mercurial pills at night. For hæmorrhages, vitamin K injections, calcium salts and hæmostatics such as coagulen Ciba are used. If portal obstruction and ascites have set in, see § 260. Patients sometimes

recover after repeated tappings, which give time for the establishment of the collateral circulation. Surgical measures have been devised to establish a collateral circulation, *e.g.*, "omentopexy," or stitching the omentum to the anterior abdominal wall. Relief of portal hypertension by anastomosing the portal vein to a neighbouring systemic vein is on trial.

C. We now turn to those chronic liver diseases in which **the size of the liver is increased.** These may be divided into two groups—those WITHOUT PAIN AND TENDERNESS are described immediately below. If the enlargement is attended WITH PAIN AND TENDERNESS, turn to § 348.

There are five diseases with **enlargement** of the liver **without pain and tenderness:** I. HYPERTROPHIC CIRRHOSIS; II. FATTY LIVER; III. VON GIERKE'S DISEASE; IV. LARDACEOUS LIVER; and V. HYDATID and other rare diseases. In INFECTIVE HEPATITIS (§ 332), CHRONIC CHOLELITHIASIS, and some other disorders, the liver is somewhat enlarged, but this is not their main feature.

Other rare causes of PAINLESS ENLARGEMENT of the liver are chronic blood diseases, noticeably LEUKÆMIA and SPLENIC ANÆMIA, ACHOLURIC JAUNDICE (§ 328), KALA-AZAR and MALARIA (§ 343, I*e*). TUMOURS (§ 350) may be unaccompanied by pain in the early stages.

I. *The* **liver is enlarged** *and* **painless**; *its surface is hard,* JAUNDICE IS PRESENT, *but little or no ascites, and there is a long history of failing health.* The disease is probably HYPERTROPHIC CIRRHOSIS.

§ **343. Hypertrophic Cirrhosis of the Liver** is a term employed in a generic or clinical sense to indicate a progressive enlargement of the liver due to an increase in the connective tissue of the organ with a tendency to jaundice. The condition may occur under at least four different aspects, due respectively to Syphilis, Gallstones, Chronic Heart disease, and Kala-azar. A rare variety of hypertrophic cirrhosis accompanied by pigmentation of the skin has been described under the name of hæmochromatosis. Glycosuria appears later; hence the name "BRONZED DIABETES." The pigmentation differs from that of Addison's disease in that it avoids the oral mucous membrane and appears on parts exposed to light rather than to pressure and friction. The pigment contains iron (§ 561).

I*a*. BILIARY CIRRHOSIS (Chronic Infective Cholangitis) is a condition occurring principally in young adults.

Symptoms. (1) There is a history of two or more attacks of acute hepatitis in preceding months. (2) The liver is uniformly, and often considerably enlarged, hard and sometimes rough. (3) The spleen is usually enlarged. (4) Recurring attacks of jaundice occur, with pyrexia even to 103° F., during which the urine contains bile and the stools are pale or clay-coloured. During these subacute exacerbations, the liver and spleen enlarge further, and the liver may become tender, with a feeling of a dull weight in the hepatic region. (5) In spite of the intense jaundice there are few or no signs of portal obstruction, and ascites is rarely, if ever, present. (6) Hæmorrhages, purpura and telangiectases may occur.

Etiology. The condition appears to be due to a subacute or chronic inflammation around the bile ducts, leading to partial obstruction. It is probably infective in origin; a similar condition may arise following chronic biliary obstruction, *e.g.*, with gall-stones in the common bile-duct.

Diagnosis.—From *portal cirrhosis* it is known by the absence of signs of portal obstruction (§ 260). *Fatty* and *amyloid* livers are not accompanied by jaundice. *Cancer* has a more rapid and painful course.

Prognosis.—Sometimes patients die within twelve months, with an acute onset of the typhoid state, but most live for a number of years, with signs of progressive liver damage. In children the general health may appear unaffected for a long period.

Treatment is as for Hepatitis (§ 332). Mercurial inunction of the abdominal wall, or calomel, gr. $\frac{1}{10}$ to $\frac{1}{4}$ t.i.d. for three days, with intervals of three days, continued for some months has good results. Glucose and insulin aid restoration of liver function. A prolonged course of penicillin should be tried. Drainage of the gall-bladder has cured some cases.

I*b*. Right-sided Heart Failure results, as we have seen, in very considerable congestion of the liver. Long-continued passive engorgement of the liver gives rise to changes known as the "nutmeg liver," accompanied by more or less enlargement of the organ; and this may be attended by a considerable degree of fibrosis. The diagnosis depends on the presence of cardiac valvular disease and other features (see Passive Congestion, § 348).

I*c*. Chronic Syphilitic Disease of the liver generally takes the form of a diffuse hypertrophic fibrosis; or it may be met with in the form of *gummata*. Hepatic fibrosis may result from both hereditary and acquired syphilis, though the gummatous form is commoner in the latter. In the inherited variety two forms of fibrosis occur. In one there is fine diffuse fibrosis between the individual cells (pericellular cirrhosis), and this variety is usually accompanied by an enlarged spleen. The liver is smooth and firm. In the other, coarse fibrosis with perihepatitis occurs, as in the acquired disease.

The *Symptoms* are variable. The liver is moderately enlarged; there is not much tendency to jaundice and portal obstruction excepting in the final stages. There may be actual pain, especially when the capsule of the liver is involved; but as a rule there are only indefinite sensations of illness, accompanied in the gummatous cases by a low form of intermittent pyrexia. In the gummatous form nodular projections may possibly be made out on the surface of the organ. The presence of such projections, accompanied by intermitting fever and a history of syphilis in a young or middle-aged adult, makes the diagnosis practically certain. In the absence of a syphilitic history the occurrence of pain and local tenderness at intervals points to syphilitic rather than to alcoholic cirrhosis, because *perihepatitis and the involvement of the capsule* are prominent features of syphilitic cirrhosis. In the diagnosis from cancer we have mainly to rely on the Wassermann reaction, the response to therapy and the (usual) absence of jaundice and ascites in syphilitic disease.

The *Prognosis*, as a rule, is good, if the nature of the disease be discovered and it be treated adequately with antisyphilitic remedies.

I*d*. Cirrhosis of Biliary Obstruction.—Hypertrophic cirrhosis has been produced experimentally in one half of the liver by ligature of one hepatic duct, and it is met with clinically in association with gall-stones, tumours or glands pressing on the bile-ducts. There is a history of repeated attacks of biliary colic, enlargement of the liver, with jaundice of some years' duration. The acholic stools aid the diagnosis of this form of hypertrophic cirrhosis.

I*e*. Tropical Cirrhosis.—Many parasitic infections involve the liver, but only a few produce actual cirrhosis. Malaria may induce hepatitis and biliary pigment stones, but it is doubtful if a true malarial cirrhosis ever occurs; kala-azar parasites, however, may produce it. Biliary cirrhosis is found in clonorchiasis and bilharzial peri-portal cirrhosis in *S. mansoni* and *S. japonicum*. Though hepatomegaly with occasional jaundice occurs, ascites is rare. And see Abscess of liver (§ 336).

II. ***The* enlargement of the liver *is* painless *and uniform; the surface is smooth and soft; there is* no jaundice or ascites, *and the* spleen is not enlarged; *there is a history of alcoholism, phthisis, or other toxæmia.*** The disease is probably Fatty Liver.

§ 344. Fatty Liver is a condition in which fat is deposited in the hepatic cells, commencing in the periphery of the lobules. It is nearly always associated with some other disease.

Symptoms.—(1) The liver is enlarged uniformly and is quite smooth. (2) Pain, jaundice, and portal obstruction are absent. (3) The accompanying symptoms are due to the cause of the fatty liver, and may consist, therefore, of debility, anæmia, etc. (4) The history of a *Cause* is important—viz., (i.) Chronic wasting disease, such as phthisis. (ii.) Fatty liver appears in association with fatty heart (*q.v.*) and general obesity. (iii.) It often accompanies chronic alcoholism; and a mixed degeneration with fat and fibrosis is not uncommon.

The *Diagnosis* from the painful enlargements of the liver is not difficult. In *lardaceous* liver there are also signs of lardaceous spleen or kidney. The *Prognosis* and *Treatment* depend upon the cause.

§ 345. III. **Von Gierke's Disease** is a rare cause of enlarged liver, usually seen in the young, due to excessive glycogen accumulation. Adrenalin does not produce the usual rise in blood sugar.

IV. *The* **enlargement of the liver** *is* UNIFORM *and* PAINLESS; *the surface is smooth and hard; there is* NO JAUNDICE, NO ASCITES; *the* SPLEEN IS ENLARGED; *there is a history of prolonged purulent discharge, phthisis, or constitutional syphilis.* The disease is LARDACEOUS DEGENERATION.

§ 346. **Lardaceous (Amyloid or Waxy) Liver** is a condition in which the liver tissue is replaced by lardaceous material, which starts in the capillaries and smaller arteries of the organ, leading sometimes to an immense enlargement.

Symptoms.—(1) The liver is enlarged uniformly and smoothly, and feels firm; (2) pain, jaundice, and portal obstruction are absent; (3) the constitutional symptoms are due to the causal condition, and to amyloid disease of other organs.

Etiology.—(i.) Long suppuration and purulent discharge, as from chronic osteomyelitis; (ii.) constitutional syphilis; and (iii.) tuberculous disease of the lungs or elsewhere. Amyloid liver has become much rarer since chronic suppuration has been obviated by improved surgical methods.

Diagnosis.—The presence or history of a cause renders the diagnosis of amyloid disease comparatively easy.

The *Prognosis* depends upon the amount of amyloid disease elsewhere. Diarrhœa, indicating amyloid changes in the intestines, abundant pale urine, with albuminuria, indicating amyloid disease of the kidneys, are untoward signs. If the cause is remediable, as by surgical treatment, the liver may decrease in size.

Treatment.—The indications are (i.) to remove the cause, and (ii.) to keep up the strength. The former is attained by anti-syphilitic treatment in the case of syphilis, and by surgical treatment in the case of long-standing discharges. Tonics, such as iron and quinine with cod-liver oil, are useful.

V. *The* **enlargement of the liver** *is* PAINLESS, *but* NOT UNIFORM, *and the upper margin of the liver dulness is perhaps* ARCHED; *there is no jaundice or ascites and the spleen is not enlarged; a thrill may be felt on percussion.* The disease is HYDATID CYST.

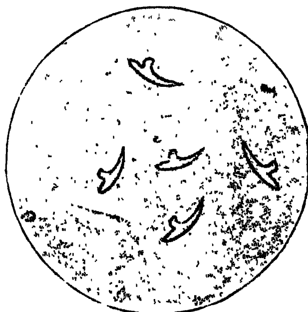


FIG. 85.—Hooklets, from a HYDATID CYST in man, magnified about 150 times. These form the crown of hooklets around the anterior end of the scolex, and are absolutely distinctive of hydatid fluid.

§ 347. **Hydatid of the Liver** depends on the presence in the liver of the parasite, *Echinococcus granulosus*, rare in this country, though common in Australia, New Zealand, the Argentine, Greece, and Iceland, where dogs live in close association with man.

Symptoms.—(i.) There is a slowly increasing enlargement of the liver, which is

smooth, globular, and elastic, sometimes fluctuating. The right chest may be bulged outwards, with dulness in the axilla. When the fingers of the left hand are laid on the tumour and tapped with those of the right hand, the "hydatid fremitus," or "thrill," is felt in some cases. (ii.) Pain is absent unless the tumour is very near the surface, when pain may be present, because *the capsule* is involved. (iii.) No constitutional symptoms appear unless the tumour presses upon the surrounding structures, or becomes inflamed and suppurates. (iv.) Rupture into the peritoneal cavity may be followed by anaphylactic shock and urticaria, and later by the growth of secondary cysts. Jaundice may occasionally be caused by cysts obstructing the bile-ducts.

Etiology.—The parasite enters the alimentary canal of man by means of food or water contaminated by fæces containing the ova of the *Echinococcus granulosus* (*Tænia echinococcus*), a tapeworm which infests the dog. The embryo is carried to the liver, where it encysts and grows, the liver tissues forming a fibrous capsule known as the adventitia. The cyst so developed has a lining membrane composed of an endogenous germinal-layer and an exogenous hyaline layer, and contains a clear fluid. The endogenous layer buds the tiny brood-capsules in which scolices or embryonic heads develop, each with a crown of hooklets. Daughter cysts and grand-daughter cysts may also be formed.

Diagnosis.—*Abscess* of the liver produces pain and fever, and on aspiration yields purulent material like anchovy sauce. *Pleural effusion* on the right side, leading to dulness in the axilla, may resemble hydatid. In such cases a *bulging* outwards of the lower ribs over the liver points to the presence of hydatid. A *renal cyst* has resonance in front, due to the colon. A history of residence in Australia, the Argentine, etc., should lead one to suspect hydatid in cases of slowly increasing enlargement of the liver, *with few other symptoms.* The symptoms of suppurating hydatid cyst of the liver are very like those of inflammation of the gall-bladder. X-rays are of value in diagnosis. Hydatid cyst fluid is pathognomonic, although exploratory puncture entails serious risk, as it may set scolices free, which subsequently form secondary cysts. The fluid is clear, opalescent, of low specific gravity, and contains a large excess of chlorides, no albumen (unless inflammation has taken place), and most characteristic of all—echinococcus hooklets (see Fig. 85 and Table LX). The blood sometimes shows eosinophilia, and the serum may give the complement fixation reaction with a suitable antigen in 70 per cent. of cases. Infested patients react to an intradermal injection of hydatid fluid (Casoni); it is a group reaction for infection with tapeworm, for in cysticercosis similar positive reactions are occasionally recorded.

Prognosis.—The patient may live for several years with no other symptoms than a slow increase in the size of the liver. The prognosis must be guarded even if the cyst is safely removed; other cysts may be present which will develop later. A cyst may remain quiescent for twelve years or more without losing its potentiality for mischief. The cyst may suppurate, giving rise to the symptoms of liver abscess, or pyæmia may be set up. When a cyst leaks into the surrounding tissues, anaphylactic symptoms may occur—collapse, vomiting, and urticaria associated with eosinophilia. Sometimes death occurs by the sudden rupture of the cyst into the pleura or peritoneum.

Treatment.—Hydatid cysts most often involve the inferior aspect of the right half of the liver and are generally accessible through an anterior abdominal incision. A transpleural route may be necessary for cysts impinging on the diaphragm. After opening the abdomen, packing off and locating the cyst, aspirate the fluid and inject 6 to 10 c.c. of pure commercial formalin. Subsequently the adventitia is incised and daughter cysts removed. The cyst cavity is filled with saline and the adventitia sutured together, where possible. Drainage is better avoided.

There are three diseases in which **enlargement of the liver** is attended **with pain and tenderness**: I. CHRONIC PASSIVE CONGESTION, II. CANCER

OF THE LIVER, and III. ABSCESS OF THE LIVER. In CHRONIC CHOLELITHIASIS and several ACUTE DISORDERS the liver may be slightly enlarged and tender.

I. *The* **enlargement** *is moderate, smooth, and uniform,* PAINFUL, *and* TENDER; *some jaundice and ascites may be present, the* SPLEEN IS SLIGHTLY ENLARGED, *and there are signs of congestion of the abdominal viscera.* The disease is probably CHRONIC CONGESTION OF THE LIVER.

§ **348. Chronic Passive Congestion** of the liver is a condition in which the enlargement is due to venous obstruction.

Symptoms.—(i.) The liver is tender, and a sensation of weight and fulness is complained of in the hepatic region. Expansile pulsation synchronous with the heart may be conveyed to the palpating hand in the early stages, but as the organ becomes firmer this is lost. (ii.) Signs of general venous obstruction appear. (iii.) Ascites develops, and the spleen is slightly enlarged. Some degree of jaundice may occur. (iv.) Gastro-intestinal disturbances are common.

Etiology.—Passive congestion is the result of any backward pressure due to obstruction or failure of the circulation. In most cases this is caused by heart or lung disease, and especially mitral stenosis.

The *Diagnosis* is often aided by the recognition of the heart disease on which it depends. In some cases of *ascites* with anasarca of the legs, we may find both *hepatic enlargement* and *albuminuria*, and a difficulty may arise as to which was the primary cause of the condition—heart, liver, or renal disease. The difficulty is increased if extensive bronchitis prevents accurate auscultation of the heart. In such cases, *the liver* may be excluded as the primary cause, if the dropsy in the legs clearly preceded the dropsy in the abdomen. The presence of hepatic enlargement is then a sign of great value as helping to exclude *renal* mischief, because enlargement of the liver is not a usual sequence of kidney disease, although it is a fairly constant result of *cardiac* valvular disease. In *paroxysmal tachycardia* the enlarged liver quickly decreases in size when the heart resumes its normal rate.

Prognosis.—The prognosis depends on the cause of the congestion; the state of the heart is generally the measure upon which the patient's chance of a longer or shorter life depends. In mitral stenosis an enlarged liver with ascites is less grave than in mitral regurgitation, because it normally occurs at an earlier stage in stenosis. It is most serious in aortic disease, and especially regurgitation, as it indicates mitral and tricuspid insufficiency.

The *Treatment* is that of the cause, and our attention must be directed to the heart and lungs. Purgatives and light foods are necessary in order to relieve the strain on the portal system. Leeches over the liver or venesection may be indicated.

II. *The* **enlargement of the liver** *is* IRREGULAR; *the* PAIN *and tenderness may be great;* JAUNDICE *and* ASCITES *are present; the spleen is not enlarged;*

the patient is advanced in years, feeble and emaciated. The disease is CANCER OF THE LIVER.

§ **349. Cancer** of the liver is rarely primary, and is usually secondary to disease elsewhere. It occurs after middle life and is rare before thirty-five.

Symptoms.—(i.) Pain is an almost constant feature of cancer of the liver; it is continuous, with exacerbations, and is independent of food or posture. A certain amount of tenderness develops. (ii.) The enlargement of the liver is irregular and may become an enormous size, and often umbilicated nodules may be made out. These are of a hard consistence, and increase rapidly. There is also less commonly diffuse cancer, in which there are no nodules, and in which the liver is only slightly and uniformly enlarged. (iii.) Jaundice is usually present, *sooner or later*, and is intense and progressive; an intense jaundice persisting over five to seven weeks in an old person should indeed always lead one to suspect cancer. Ascites generally occurs either from involvement of the glands in the fissure, or of the peritoneum. The spleen is not enlarged. (iv.) The general health of the patient is bad, and emaciation and cachexia may be present before any local signs are discovered. Cancer may be present in another part of the body. Fever may occur at intervals, and a polymorph leucocytosis (sometimes marked) is usual. Rectal examination may reveal malignant glands in the pelvis.

Etiology.—Cancer is liable to spread to the liver (*a*) via the portal blood stream or the abdominal lymphatics, from primary disease in the stomach, colon or other abdominal organs. (*b*) It may invade the liver via the systemic blood vessels from a primary site in the breasts, lungs, testicles, etc.

Diagnosis.—Jaundice is rarely entirely absent in cases of cancer of the liver: this and the cachexia alone may justify a diagnosis. The diagnosis from *cirrhosis* may be difficult when nodular enlargement cannot be made out, and when considerable ascites is present. In cirrhosis there is little or no pain and tenderness, the history of the illness is of longer duration, the spleen may be enlarged, and the jaundice is not so intense. The *inflammatory thickening* under the liver after a long history of gall-stones may resemble cancer, and can be distinguished only when time shows little or no increase in the enlargement. In doubtful cases, the abdomen should be thoroughly examined after removal of the ascitic fluid. *Syphilitic* liver has not so much pain and tenderness, is of slower growth, and rarely produces ascites.

Prognosis.—Cancer of the liver is usually fatal within six to twelve months, death taking place from exhaustion. Untoward symptoms are rapid growth, ascites, or respiratory difficulties due to extension of the disease to the lungs and pleura.

Treatment can be palliative only. Treatment of ascites makes the patient more comfortable. Morphia or opium is administered for the pain, and attention must be given to the relief of the symptoms of gastric

distress, and to aid nutrition. With rest and care there may be periods during which the disease makes no progress, and which hold out to the patient false hopes of his ultimate recovery.

III. **Abscess of the Liver** also produces considerable hepatic enlargement, which is PAINFUL and TENDER. It has already been described among the Acute Diseases, § 336; but sometimes it runs a very chronic course.

§ **350. Tumours of the Liver** other than CANCER (§ 349), HYDATID (§ 347), and GUMMA (§ 343, Ic.), are more rare. Their presence is manifested by *enlargement of the organ*, which may be regular or irregular, accompanied in some cases by constitutional symptoms. When, as in some cases of ACTINOMYCOSIS and FASCIOLA HEPATICA (*Distoma hepaticum*) (§§ 337, 338), they assume an inflammatory form, pyrexia is present. SARCOMA OF THE LIVER is occasionally met with—*e.g.* Lympho-sarcoma—but it is most often secondary to deposits elsewhere, and the liver condition is only a subordinate part of the case. The patient may be younger than in the other forms of malignant disease. Chondro-sarcoma, Melano-sarcoma, Tubercle, Angioma, Lymphadenoma, and Fibroma occur very rarely. Riedel's lobe is often mistaken for tumour (§ 263).

THE GALL-BLADDER

PART A. SYMPTOMATOLOGY

§ **351.** The cardinal symptoms commonly associated with gall-bladder disease are **pain** or **discomfort in the upper abdomen** and back, **flatulence, nausea** or **vomiting.** Occasionally **constitutional symptoms** are also present. Pain or discomfort is usually epigastric, often worse on the right side, and may be related to meals. It varies from a dull ache to acute paroxysms of colicky pain, as when a calculus becomes impacted in the neck of the gall-bladder or in the cystic duct. The pain is often referred to the lower right ribs, the angle of the right scapula or between the scapulæ. Flatulence in the abdomen may be severe; it produces a sense of fullness, so that the patient loosens the clothing. Nausea is rarely present before a meal, but after a few mouthfuls of food the patient may feel so distended and nauseated that he cannot eat more. Vomiting may be occasional, or in attacks, associated with the other symptoms. With colic it is usually severe. A characteristic feature of gall-bladder disease is the aggravation of the symptoms by food containing eggs, cream and animal fats, so that the patients avoid these foods. Pyrexia and other constitutional manifestations accompany catarrhal or suppurative processes in the gall-bladder. **Jaundice** is present when there is obstruction of the hepatic or common bile ducts.

PART B. PHYSICAL EXAMINATION

When examining the gall-bladder one must ensure that the abdominal wall is entirely relaxed. With the patient in the supine position and the knees drawn up, palpate gently with the fingers laid flat on the abdominal wall, the patient breathing gently all the time. Occasionally more satisfactory results are obtained by making the patient sit and lean

forward with the knees flexed, completely relaxed, whilst one palpates with the tips of the fingers under the right costal margin. The gall-bladder may be best felt when a deep breath is taken; when enlarged it is felt as a tender globular swelling coming forward at the tip of the ninth right costal cartilage. It usually remains just under the surface of the anterior abdominal wall, moves freely downwards with respiration, but cannot be moved laterally; when very large it is dull to percussion and may extend even to the right iliac fossa. With cancer of this organ, the surface becomes hard and nodular (and see § 263. 1). Even when the gall-bladder is not large enough to be felt, with inflammation the upper right rectus muscle shows rigidity. If the lower hepatic margin in the right hypochondrium be divided into outer, inner and middle segments, when the patient takes a deep breath he flinches and his face expresses pain on deep palpation of the middle segment but not with palpation of the other segments. In diseases of the gall-bladder it is essential to examine the back, as referred areas of tenderness may be met (*a*) over the 11th and 12th right ribs, (*b*) over the 5th–8th dorsal spines or (*c*) over the para-vertebral muscles between the scapulæ, especially over the right side. A friction rub is occasionally audible over the gall-bladder.

There are two forms of special investigation: (*a*) X-ray examination reveals gall-stones if opaque material such as calcium is present. Cholecystography is performed after giving iodophthalein B.P. by mouth or intravenously (Graham-Cole test). When it is taken by mouth, radiograms 12 hours later reveal the degree of filling; if a fatty meal is then given, a further plate reveals the degree of emptying. Filling may be promoted by giving two 1 oz. doses of glucose the day before. If the gall-bladder does not fill, the glucose is immediately repeated, a fat-free diet given, and more iodophthalein 24 hours after the first dose.

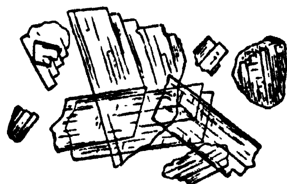


FIG. 86. — Cholesterol Crystals. Microscopic appearance presented by fragments of gall-stones in the fæces or from the duodenum.

Normal filling and emptying are occasionally compatible with a diseased gall-bladder, but as a rule improper filling and emptying, especially after the intravenous method, indicates a pathological condition. Non-opaque stones may be visualised only when they are surrounded by opaque substance (Fig. 87). (*b*) By introducing a long rubber tube into the duodenum, especially after a period of starvation, a sample of resting duodenal contents may be obtained; if then 30 c.c. of concentrated magnesium sulphate or 20 c.c. of hot olive oil or of peptone (10 per cent.) are introduced through the tube, a profuse flow of bile from the gall-bladder may be obtained within a few minutes and a sample aspirated; this is examined for micro-organisms (especially *B. coli* and those of the typhoid group), cholesterol crystals (which may be deformed when gall-stones are present) (Fig. 86), for lipoid globules (from a "strawberry gall-bladder"), and for cells. After magnesium sulphate desquamated cells from the duodenal wall may be present and must be differentiated from pus cells. If achlorhydria is present, the results must be viewed with caution.

PART C. DISEASES OF THE GALL-BLADDER

Gall-bladder Disease may be: A. **Acute**—I. ACUTE CHOLECYSTITIS, II. GALL-STONE COLIC; or B. **Chronic**—III. CHRONIC CHOLECYSTITIS, IV. CANCER OF THE GALL-BLADDER.

I. *The patient complains of* PAIN *in the* GALL-BLADDER REGION; *the pain is* PAROXYSMAL, *or* DULL AND CONTINUOUS, *and* RADIATES TO THE RIGHT SHOULDER. *There is* TENDERNESS *over the* GALL-BLADDER, *vomiting and some fever. The disease is* ACUTE CHOLECYSTITIS.

§ 352. **Acute Cholecystitis** may be catarrhal, suppurative, or gangrenous, according to the severity of the infection.



FIG. 87.—*Left*: a normally filled gall-bladder 13 hours after administration of the dye. *Right*: same type of gall-bladder, filled with typical facetted stones displacing the dye.

Symptoms.—(1) Local pain in the right hypochondrium and epigastrium. (2) Pain is spasmodic or dull and continuous, and radiates through to the back and right shoulder. (3) Tenderness in the right upper abdomen; this becomes localised below the tip of the ninth costal cartilage. (4) Rigidity of this area; if the muscles are relaxed, the enlarged gall-bladder may be felt. (5) Symptoms may be mild, like dyspepsia, or severe with vomiting, jaundice, rigors and much general disturbance.

Etiology.—*Predisposing*: (1) stagnation of bile; (2) calculi of solitary cholesterol type; (3) foreign bodies, worms and ova in the gall-bladder; and (4) previous attacks. The *exciting cause* is infection, which may come

from tonsils or teeth; or follow pneumonia, influenza or typhoid fever, gastric, duodenal or appendicular disease.

Diagnosis.—Absence of jaundice is not a point against cholecystitis. In *gall-stone colic* the pain is more severe, while local signs of tenderness, paralytic distension of intestines and palpable gall-bladder favour cholecystitis. Leucocytosis is rare with gall-stones, unless accompanied by cholecystitis. In perforated *duodenal ulcer* there may be a history of characteristic indigestion. In *acute pyelonephritis*, pus and *B. coli* are found in the urine. *Appendicitis* may cause difficulty; appendicitis and cholecystitis may co-exist. Right *diaphragmatic pleurisy* or *basal pneumonia, herpes zoster* and *intercostal neuralgia* must be excluded.

Prognosis.—The attack may subside or pass into chronic cholecystitis. If it proceeds to suppuration or gangrene, local or general peritonitis may supervene and life may be endangered.

Treatment.—The patient should be in bed on milk or light diet. Local applications of fomentations, a kaolin poultice, dry cupping or leeches may be used, or morphia may be required for the pain. Vomiting may be relieved by bismuth carbonate, hydrocyanic acid or an effervescing mixture. Salicylate of soda and hexamine with alkalies are useful as antiseptics. Penicillin injections have given good results. A drachm of magnesium sulphate taken in a dessertspoonful of water, fasting, in the morning, and followed after an hour by a pint of hot water or weak tea, and pure olive oil ♏120 between meals, act as a stimulus to gall-bladder evacuation. If fever and rigors persist, operation on the third or fourth day is indicated. In cholecystitis, even without gall-stones, drainage is not sufficient to cure, and the gall-bladder should be removed.

II. *The patient, usually an elderly female, is suddenly seized with* PAROXYSMS OF SEVERE PAIN *in the hepatic region, and in the course of twelve to twenty-four hours she may become* JAUNDICED, *the stools becoming clay-coloured.* The attack is one of BILIARY COLIC.

§ 353. **Gall-stones** and **Biliary Colic.**—Gall-stones are concretions which form in some part of the biliary passages, most commonly in the gall-bladder. CHOLELITHIASIS is the condition in which gall-stones are developed. When gall-stones move along any of the ducts, they give rise to Biliary Colic.

GALL-STONES may be *metabolic*, consisting of deposits of cholesterol or bile-pigment, or *infective*, of mixed composition. They vary in size from a sand-grain to a golf-ball. When solitary, they are round or oval in contour. The facets or flattenings of their surface are caused by the pressure of one against the other; this indicates that there has been more than one stone in the gall-bladder or bile-ducts. The colour varies from yellow to dark brown; their chief physical characteristics are the smooth "soapy" surface, the ready way in which they crumble between the thumb and finger (though sometimes they are very hard), and their lightness as compared with renal calculi. They generally consist chiefly of cholesterol mixed with calcium and bile pigment, but are sometimes pure cholesterol, pure bilirubin, or pure calcium carbonate. Cholesterol is contained and held in solution by bile salts in normal

bile. When from various causes the liver is unable to produce the bile salts in sufficient quantity, there is a high cholesterol content in the blood and bile, with eventual deposition of cholesterol and formation of gall-stones. Normal individuals can eat food containing cholesterol, because more bile salts are produced by the liver and hold the cholesterol in solution. With other individuals this capacity is defective. The foods which increase the cholesterol content of the blood are: egg yolk, butter, cream, liver, kidney, pancreas, brain and meat fats.

Biliary Colic.—Symptoms may be absent when the stone is at rest, but when it begins to move (i.) the pain is agonising; it starts in the epigastrium and shoots into the right hypochondriac region towards the spine and up to the right shoulder, but never passes downwards. The paroxysm is usually so severe that the patient is in a state of partial collapse, with vomiting, hiccough, subnormal temperature, and a quick, weak pulse. Sometimes there is a rigor, and the temperature rises a few degrees. Between the paroxysms of acute pain there is a constant dull aching and tenderness over the hepatic region. The attack lasts from a few hours to a few days. (ii.) The liver may be enlarged and if a stone becomes impacted in the hepatic duct the enlargement may be considerable. (iii.) Jaundice usually appears twelve to twenty-four hours after the paroxysm, and lasts from a few days to a few weeks. It is most intense when the stone is impacted in the ommon duct, and may give rise to severe pruritus.

The *Symptoms* which arise vary somewhat with the *position of the gall-stone* (Fig. 88). Thus: (i.) If a stone is impacted in the *common duct* there are biliary colic, jaundice, and sometimes a distended gall-bladder, and if the impaction continues the liver becomes enlarged. (ii.) If a gall-stone is impacted in the neck of the gall-bladder (*i.e.*, in the *cystic duct*), *biliary colic without jaundice* is present. In time the gall-bladder may be distended with mucus, and form a definite abdominal tumour (mucocœle), but more often the chronic irritation of many calculi leads to chronic fibrosis of the gall-bladder which prevents its becoming enlarged. Considerable distension of the gall-bladder is not usually associated with the presence of many gall-stones, but more often with cancer of the pancreas or chronic pancreatitis.

(iii.) Stone impacted in the *hepatic duct* is rare. It causes biliary colic and jaundice, but the gall-bladder is not distended. (iv.) Stones occasionally form in the *radicles of the hepatic ducts*, and give rise to indefinite symptoms, sometimes without pain, and usually without jaundice. (v.) Sometimes small particles of cholesterol (biliary sand) in the *gall-bladder* give rise to recurring paroxysms of pain, unaccompanied by other symptoms, eluding diagnosis. (vi.) The stones may become encysted, but more often, without surgical intervention, abscess and fistula result.

Diagnosis of Biliary Colic.—It is distinguished from the two other forms of colic in Table XIV, § 246. The severity of the pain and its paroxysmal character usually distinguish it from other acute diseases of the liver. *Pseudo-biliary colic* is sometimes met in nervous women. The diagnosis

from *cancer* of the liver may be very difficult. Both occur at the same age, and both cause jaundice; further, cancer of the gall-bladder may follow after years of trouble from gall-stones. In cancer, jaundice steadily becomes more intense. It must be remembered that in some cases gall-stones are passed without colic, but with jaundice; consequently, *recurring attacks* of jaundice in an elderly woman should lead one to suspect gall-stones. A radiogram may show gall-stones, but a negative plate is not conclusive. Negative shadows may be defined in the opaque gall-bladder with the Graham-Cole test (Cholecystography, p. 436). In all suspected cases the stools should be carefully examined for stones. *The presence of ascites* points to cancer, which rarely exists long without peritoneal effusion.

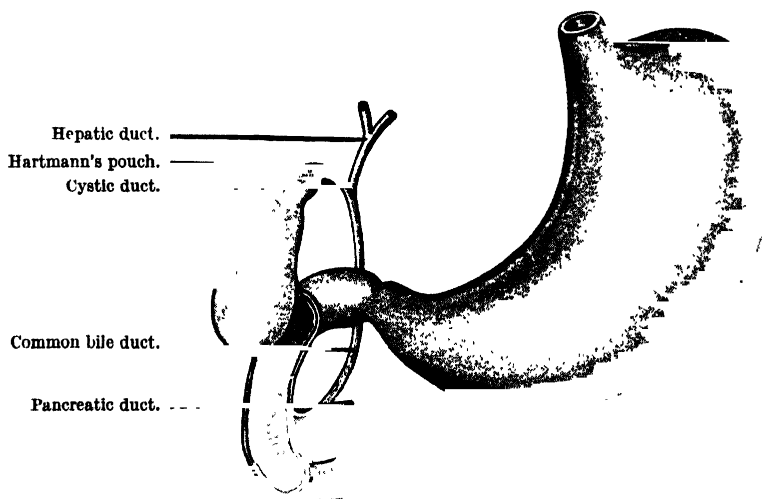


FIG. 88.—The STOMACH AND DUODENUM opened to show the ducts in connection with the Liver and Pancreas.

Gall-stones at rest in the Gall-bladder occur often in elderly women: they give rise to *Symptoms* the cause of which may be difficult to diagnose. They are the symptoms of cholecystitis (§§ 352, 354) which precedes or accompanies gall-stone formation. They consist of (1) flatulence, especially after fats, (2) pain referred to the right upper abdomen and shoulder, (3) subcostal ache, especially when chilled.

Etiology of Gall-stones.—(i.) They occur usually after the age of 50; (ii.) are much commoner in women, especially in multiparæ, and (iii.) in stout persons of sedentary habit whose diet is rich in fat and sugar. (iv.) There is often a history of gout, asthma or migraine. (v.) They may follow cholecystitis due to typhoid, coli or streptococcal infection, or any cause of stagnation of bile in the gall-bladder. (vi.) The colic is often

determined by a sudden strain, by motoring or by an overloaded stomach especially with rich fatty food.

Course and Prognosis.—The prognosis as to recovery from an attack of biliary colic is excellent, but recurrence may be expected. A stone usually forms in the gall-bladder and becomes impacted for a time in the neck of the cystic duct, giving rise to biliary colic without jaundice. It may then pass down the common duct, and cause jaundice. This rarely lasts more than a few weeks, but cases have been reported where it lasted two years. Impaction with infection is followed by: (i.) *Ulceration* of the ducts, with pyrexia, or abscesses of the liver and bile-ducts (cholangitis), and consequent subacute pyæmia; (ii.) *perforation* into adjacent tissues, leading, for example, to fatal peritonitis; (iii.) inflammation and *abscess* (empyema) of the gall-bladder, which may open externally, perforate into the peritoneum, or rupture into the intestines; (iv.) formation of *fistula* between the gall-bladder and the colon or duodenum, through which stones can pass of such a size that they may cause intestinal obstruction. (v.) *Cancer* may supervene in later years.

Treatment.—*During the attack* treatment aims at relieving spasm and controlling pain. If mild, a tablet of trinitrin may give relief. If severe, belladonna is the drug of choice: a dose of 15 minims of the tincture may be repeated after 2 hours, or a full dose of atropine given. A hypodermic injection of morphine or pethidine may be necessary for the pain, but morphine tends to increase biliary spasm. Chloroform inhalations are used in severe cases. Hot water with grains 60 of bicarbonate of soda to the pint aids the flow of bile, and hot turpentine stupes relieve pain. Sometimes an attack of pain is warded off by giving a hot bath (100° F.).

Between the attacks the diet must be supervised. Foods containing cholesterol must be omitted, therefore forbid most animal fats, especially if cooked. A little butter is allowed, but no cream or yolk of egg, no kidney, liver, brain, sweetbread or the fat of meat, pork, goose and duck. (For specimen diet see § 297, IV.) To flush out the biliary passages, the liver can be made to secrete more bile by administering bile acids, potassium salts, salicylates, and oil of peppermint: particularly powerful is dehydrocholic acid (decholin): Carlsbad Sprudel salt is popular, as it is rich in potassium salts. When it is desirable to cause the gall-bladder to contract and empty itself (gall-bladder drainage), give magnesium sulphate in doses of gr. 30 to 60 in concentrated solution before breakfast, and olive oil between meals. Where there is hyperchlorhydria, a tablespoonful of olive oil with a small dose of tincture of belladonna is useful, given before meals. Hexamine and sodium salicylate are excreted in the bile; the latter increases the excretion of bile salts and cholesterol; the hexamine sterilises the infected bile, when given in large doses with citrate of potassium in order to prevent irritation of the urinary bladder. Felamine is a useful preparation, in 5-grain tablets twice or thrice daily. Surgery is indicated where there is suppuration, when the gall-bladder

remains distended, the common duct is blocked, or biliary colic frequently recurs. The old practice of giving large amounts of olive oil does not remove gall-stones; the resulting masses passed in the fæces are aggregations of fatty acid crystals, not the gall-stones.

B. Chronic Diseases of the Gall-bladder

III. *The patient, a young adult or middle-aged, complains of* FULLNESS *weight or oppression* IN THE EPIGASTRIUM *about half an hour after meals,* WORSE AFTER GREASY *or* ACID FOOD. *Relief is obtained by belching, and cessation almost at once by vomiting. There is* CHILLINESS *or* SHIVERING *in the evenings, and a shoulder ache or stabbing* PAIN *in the* RIGHT SIDE *with a deep breath. The disease is probably* CHRONIC CHOLECYSTITIS.

§ **354. Chronic Cholecystitis** is one of the commonest of all abdominal diseases, and is often undiagnosed in the early stages when medical treatment is available. It may follow acute cholecystitis, it may precede or accompany gall-stones. Or, it may be chronic from the onset, brought on by sedentary habits which predispose to stagnation of bile and infection. The infection, usually borne by the blood-stream, is first seated in the wall of the gall-bladder. Cholesterol metabolism is interfered with, hypercholesterolæmia follows, and the mucosa of the gall-bladder becomes engorged with cholesterol (" strawberry gall-bladder "). Cholesterol stones form in the lumen followed by infection of the contents of the gall-bladder; at this stage are formed the mixed gall-stones of cholesterol, bile pigments and calcium.

Symptoms.—(1) Continual flatulent dyspepsia, fullness or oppression in the epigastrium, coming on soon after food; (2) worse after fruit, eggs, cooked fats, pork, pastry, pickles or heavy meals; (3) relieved by belching and ceasing almost at once after vomiting; (4) distension or tightness relieved by bending forwards, flexing the right thigh on the abdomen or loosening the clothing; (5) acidity or heartburn, sometimes a gush of saliva into the mouth; (6) chilliness or " gooseflesh," especially in the evenings. Attacks of " biliary fever," *i.e.*, shivering, nausea, vomiting, diarrhœa and faintness, with slight temperature, may occur at intervals for months or years, especially after exertion. (7) Aching in the right shoulder or stabbing pain with tenderness at the angle of the right scapula. Tenderness may occur in the areas supplied by the seventh to ninth thoracic segments, the areas which supply the sympathetic nerves to the gall-bladder and bile-ducts. (8) There may be congestion and œdema at the right base. (9) There is sometimes reflex gastric hyperchlorhydria, but the stomach juices are usually sub-acid. (10) Remote symptoms from the gall-bladder as a source of infection are chronic infective arthritis, fibrositis, phlebitis, anæmia or myocardial degeneration with palpitation, extrasystoles and breathlessness on exertion.

Etiology.—Chronic cholecystitis may occur at any age, but is frequent in the young. It follows (1) biliary stasis from sedentary habits, insufficient exercise or constipation; (2) infection, which takes place usually (*a*) by the blood-stream, but may spread (*b*) by direct extension from pre-existing hepatitis by way of the lymphatics, or (*c*) by ascent up the bile-ducts from the duodenum, and (3) disturbance in cholesterol metabolism.

Diagnosis.—Persistence of symptoms of flatulence ("wind") is characteristic of chronic cholecystitis. In cases with reflex superacidity of stomach contents, symptoms may resemble those of *duodenal ulcer.* The pain, coming long after meals or in the early morning, is relieved by food and alkalies; but with gall-bladder disease the pain is less regular and is made worse by fats. *Spastic gall-bladder* has much the same symptoms, but is relieved by belladonna (Newman). In *intercostal neuralgia*, the tenderness is in the abdominal parietes, not deep: and with a painful *slipping costal cartilage*, there is local tenderness of the costal margin. With X-ray, there may be (*a*) opacity in gall-bladder region; (*b*) irregularity and fixation of the hepatic flexure; (*c*) with cholecystography, non-filling or irregular filling points to a diseased gall-bladder; negative shadows of stones may be seen. A barium meal will demonstrate any lesion in the adjacent pylorus or duodenum. Duodenal intubation may show pus cells or bacteria (§ 351). The symptoms may resemble those of *psychoneurosis* and a careful enquiry into the environment and former history of the patient will help in diagnosis.

Course and Prognosis.—Gall-bladder disease must be thought of as a focus of infection in "toxæmic" states. If neglected, cholecystitis may lead to gall-stones, empyema or cancer of the gall-bladder.

Treatment.—Indications are (1) to prevent the stagnation of bile by exercise, plenty of fluids, magnesium sulphate, gr. 60 in water fl. oz. 2, first thing in the morning; (2) reduce bile cholesterol by a dietary of vegetables and carbohydrates, avoiding cream, egg-yolk, sweetbreads, brain, liver, kidneys and large meals; (3) treat infective foci by removal of diseased teeth or tonsils and attention to bowel, appendix, pelvic organs and nasal sinuses. Salicylate of sodium or hexamine combined with sodium bicarbonate and potassium citrate act as disinfectants of the biliary tract. Since the infection is intramural, drainage alone at operation will not effect a cure, and the gall-bladder should be removed. In older people palliative treatment should be recommended and a course of penicillin injections tried; in younger patients, operation.

IV. *The patient, a stout woman of sedentary habits, between fifty and sixty, who has suffered for years with "windy spasm" or mild colic, has a* CONSTANT OPPRESSION OR DISCOMFORT *in the* RIGHT HYPOCHONDRIUM, *loses weight and appetite, is* JAUNDICED *and has a palpable* TUMOUR IN THE GALL-BLADDER REGION. The disease is probably CANCER OF THE GALL-BLADDER.

§ **355. Cancer of the Gall-bladder** is uncommon. It is closely associated with cholelithiasis. Calculi are found in 70 to 90 per cent. of cases, and primary carcinoma of the gall-bladder occurs in 4 to 14 per cent. of all cases of cholelithiasis. It is much more common in women than in men (4 : 1).

Symptoms.—(1) The symptoms preceding the onset of carcinoma of the gall-bladder are those of the pre-existing cholelithiasis and cholecystitis. Biliary colic may occur, but usually there is only discomfort and heaviness in the right upper abdomen. (2) A tumour may be felt, at first round and smooth, but later nodular and hard. It moves with respiration. (3) Jaundice follows from pressure on the ducts by secondary glands or from catarrh. (4) Ascites occurs from pressure on the portal vein or secondary growths in the peritoneum.

Diagnosis.—The presence of a hard, nodular, progressively increasing tumour in the gall-bladder region of an elderly woman is suggestive. Gall-stones may cause inflammatory thickening round the gall-bladder, but enlargement of the gall-bladder is in favour of growth. Jaundice may come on suddenly with diarrhœa and vomiting, simulating infective hepatitis, but is progressive. Carcinoma of the stomach or hepatic flexure of the colon may cause confusion.

Treatment.—Extirpation by total removal offers the only hope of recovery. Medical treatment must be merely palliative.

THE SPLEEN

There is still some doubt as to the precise part played by the spleen, and symptoms may be altogether absent when it is diseased. Great diminution in size has been found *post-mortem* without any symptoms during life. The duties of the spleen are still uncertain; it does not appear to have an internal secretion as do the thyroid, suprarenal, and pituitary glands. One of its main functions is connected with the reticulo-endothelial system, of which it forms an important part. The reticulo-endothelial cells are widely distributed throughout the body. In certain diseases of the spleen, and after splenectomy, some of the functions of the spleen can be carried out by the other parts of the reticulo-endothelial system. In embryonic life the spleen is concerned with the formation of red and white blood corpuscles. In certain of the " blood diseases " in which it is enormously enlarged it resumes these functions. In the adult it is an important site of formation of lymphocytes and monocytes. It also deals with the removal from the circulation of old red cells, of pigments and parasites as in malaria. It enlarges during digestion, and owns muscle fibres which give it the power of rhythmical contraction, by which it can force its store of red blood cells into the circulation, *e.g.*, after hæmorrhage. In all probability the spleen is in some way necessary to the proper fulfilment of the digestive processes.

The spleen may show various congenital abnormalities. Of these the commonest is the presence of accessory spleens; less common are multiple spleens and a multilobular organ.

PART A. SYMPTOMATOLOGY

§ **356.** In addition to the local pain and discomfort due to enlargement, the symptom which is found to be most constantly associated with disease of the spleen is *anæmia*, the various causes of which are discussed elsewhere (§ 535). From this arise symptoms which include *extreme pallor* of the skin, great *weakness*, and *alterations in the blood-cells*. The size of the spleen is not necessarily a measure of the severity of the symptoms. Thus in "ague cake," for example, great enlargement takes place without any symptom beyond the inconvenience due to the size of the organ. In other instances a large spleen may, by simple pressure or by the formation of adhesions, give rise to signs of disease in the neighbouring organs, especially the stomach. Pain and local tenderness accompany acute enlargements, and there may also be pyrexia and vomiting. The liver and spleen are often enlarged together; one may precede the other, or both may be results of a common cause.

PART B. PHYSICAL EXAMINATION

The only physical sign which can be relied upon as diagnostic of splenic disease is enlargement of the organ, and this is most readily made out by **Palpation.** When the spleen is enlarged, the anterior edge, being free, makes its way downwards and forwards towards the umbilicus. The *notch* in the anterior border is so characteristic that it forms a strong point in diagnosis of any splenic tumour. METHOD.—Stand on the right side of the patient, who should be lying on his back. Pass the left hand across the abdomen, and lay it posteriorly over the eleventh rib on the left side; then place the right hand flat upon the anterior surface of the abdomen, with the tips of the fingers just below the eleventh rib. By gently dipping down into the abdomen, and tilting the organ upwards with the left hand during inspiration, the splenic edge and its notch may be felt. It is more readily palpated when the patient draws a deep breath. Normally, the spleen cannot be detected by palpation, and even slight enlargements may not always be appreciable. An enlarged spleen always has a space between its posterior edge and the erector spinæ behind, into which the fingers can be dipped—at any rate, in spare subjects. *Fallacies.*—Without being enlarged, the spleen is readily palpable when it is displaced downwards, or is "floating." It is sometimes displaced downwards in cases of deformed chest (*e.g.*, rickets), large pleural effusions, and emphysema.

It is important to notice the CHARACTER of the enlarged spleen: a *soft* spleen may be due to some recent cause, *e.g.*, septicæmia or typhoid fever; a *firm* spleen to a disease of longer standing, *e.g.*, pernicious anæmia. A

hard spleen indicates fibrotic changes in the organ. Also VARIATIONS IN SIZE should be recorded in relation to the left costal margin or the umbilicus.

The **Percussion** of the spleen is attended with some difficulty. The organ is situated in the left hypochondrium, between the upper border of the ninth rib and the lower border of the eleventh; and roughly between the mid-axillary and scapular lines (Fig. 41, § 108). It extends obliquely forwards and downwards nearly to the costal margin and lies wholly beneath the ribs: the upper third is overlapped by the lung. Percussion does not afford a very accurate means of investigation, but if it is desired to make use of this method of detecting splenic enlargement, it is best to percuss along the length of the 11th left rib and at the end of an expiration, because the spleen is then less covered by lung.

X-ray examination may be carried out.

Fallacies.—The dulness of *splenic enlargement* may be simulated by pleural effusion or consolidation of the left lung. The area of splenic dulness may be *diminished* by emphysema of the lungs, or distension of the stomach or colon by gas. The splenic dulness may be absent when there is a wandering spleen, or with congenital absence.

§ **357.** SPLENIC ENLARGEMENTS have seven chief characteristics: (1) The splenic *notches* are felt on its anterior border; (2) the mass moves with respiration if not bound down by adhesions; (3) it is dull to percussion because the resonant colon does not lie in front of splenic tumours, as it does in front of renal tumours. (4) It is palpable just under the anterior abdominal wall. (5) It is impossible to palpate above the organ, as it comes down beneath the left costal margin. (6) An enlarged spleen rarely crosses the midline above the umbilicus. (7) When an area of dulness is due to splenic enlargement, its outline *resembles in shape* that of the normal spleen. (8) It is distinguished from neoplasms of the peritoneum, stomach, intestines, etc., by its smooth and firm surface. Irregular enlargements of the spleen are rare, and can only be diagnosed after careful examination has excluded disease of other viscera.

Splenic enlargements or tumours may have to be diagnosed from the following conditions: (1) *Renal tumours,* and especially movable kidney, in which there is resonant intestine in front of the tumour, and absence of resonance in the flank; (2) *enlargement of the left lobe of the liver,* in which the dulness is continuous with that of the right lobe, whereas splenic dulness rarely reaches to the middle line; (3) *cancer of the cardiac end of the stomach,* in which the dulness is less absolute, and there is "coffee-ground" vomiting, etc., and the splenic notch is absent; (4) *ovarian tumour,* which (i.) will have grown from below upwards, (ii.) the hand cannot be pushed between the tumour and the pelvic brim as it can in the case of a splenic tumour, and (iii.) can be felt on vaginal examination; (5) *accumulation of fæces,* in which (i.) the tumour has an irregular outline, (ii.) doughy consistence, and (iii.) a course of purgatives and enemata will remove it; (6) *post-peritoneal tumour,* in which (i.) there is no notch, and (ii.) no resonance behind it; (7) *abdominal aneurysm,* when of sufficient

size to be mistaken for the spleen, is attended by pain in the back, and evident expansile pulsation; (8) *deep-seated abscess in the abdominal parietes* is tender, has a vague irregular outline, and is situated more superficially than a splenic tumour. In (9) *cancer of the splenic flexure* of the colon the mass varies from day to day and there will be intestinal symptoms; (10) *pancreatic* and *suprarenal* tumours and *perinephric abscess* may give rise to difficulty. (11) Rare causes of error are localised *tuberculous* masses and the thickened colon of *bilharziasis*.

PART C. DISEASES OF THE SPLEEN

§ **358.** The diseases of the spleen are all—if we except the relatively rare cases of wandering spleen and atrophy—comprised under the causes of **enlargement of the organ,** and its **diagnosis** therefore becomes a matter of considerable importance. Enlargement is detected by palpation aided by percussion as above mentioned. The mechanical effects of pressure, when the spleen is very much enlarged, are mainly dyspnœa and gastro-intestinal disturbance. These may be aggravated by attacks of perisplenitis, with acute pain locally, vomiting, pyrexia, and sometimes diarrhœa. Œdema of the base of the left lung is not uncommon.

The **Causes of Enlargement of the Spleen** are most readily differentiated according as they depend upon or are associated with the following:

I. Acute infections.
II. Chronic infections.
III. Portal obstruction or congestion.
IV. Blood diseases.
V. Parasitic and tropical diseases.
VI. Infancy and childhood.
VII. Irregularity of the surface of the spleen.

Method of Procedure.—As pointed out in Part A, advice is rarely sought for symptoms directly pointing to the spleen. Frequently the spleen is found to be enlarged when the patient is being examined for disease elsewhere. It should be remembered that in many diseases the detection of an enlarged spleen may be an important clue to the diagnosis.

Inquiry should be made as to the HISTORY. Thus residence abroad suggests malaria; prolonged suppuration, lardaceous disease; fever and rigors, the presence of some pyæmic cause.

The AGE of the patient is important (see VI. below); in childhood certain conditions are common which are rare in adults.

The TEMPERATURE aids the diagnosis of certain infections.

EXAMINATION OF OTHER ORGANS may render the diagnosis easy. The condition of the LIVER is of especial significance in several diseases. Thus a large liver, jaundice, and a normal spleen point to gall-stones or cancer, but if the spleen as well as the liver is large, these symptoms suggest cirrhosis or other obstruction. A very enlarged spleen with but slightly enlarged liver suggests some of the "blood diseases" which can be accurately differentiated only by an EXAMINATION OF THE BLOOD (§ 527).

I. **Acute Infections.**—Almost all acute infections are apt to be accompanied by slight enlargement of the spleen, and as far as the acute specific fevers are concerned this is usually of little clinical significance. The enlarged spleen often feels soft, is unaccompanied by local symptoms and is especially found with typhoid, abortus and typhus fevers. Sometimes, and particularly in TYPHOID fever, a splenic abscess may complicate the original condition. In such a case local symptoms of tenderness and pain will draw attention to the spleen. Again, these symptoms may arise in the course of some systemic infection, and be due to suppuration supervening in the area affected by an EMBOLISM or in some pre-existing cyst or tumour. Embolism due to cardiac disease causes (i.) acute sudden pain, and (ii.) local tenderness due to perisplenitis. Embolism due to pyæmia is usually known by the presence of the causal condition. In such diseases as leukæmia, in which the massive enlargement of the spleen is a prominent feature, the organ is liable to attacks of ACUTE CAPSULITIS, which may give rise to difficulty in diagnosis unless the possibility of their presence is borne in mind. A friction rub, due to localised peritonitis, may be audible during the acute attacks.

The *diagnosis* of the cause may be very difficult, but should be solved by patient investigation. Meanwhile, expectant treatment is to be adopted, and consists of rest in bed, attention to the bowels and hot applications to the spleen (for pain). If the attack does not subside and the local signs become worse, the advisability of surgical interference must be considered. Fortunately this is rarely needed, and the attacks tend to resolve in a few days, leaving adhesions which may lead to trouble later.

II. **Chronic Infections.**—(1) INFECTIVE OR MALIGNANT ENDOCARDITIS (§ 50) may give rise to embolism, which causes acute symptoms, or to a more chronic enlargement not wholly due to congestion, and due to the accompanying septicæmia. The symptoms in the latter case may be exactly similar to those of splenic anæmia (§ 544), and may occur when there is no suspicion of cardiac trouble. The importance of this lies in the fact that it is possible to remove the spleen with advantage to the patient in splenic anæmia, but the operation should not be performed in endocarditis. ABSCESS of the spleen may also occur in the course of this disease.

(2) SYPHILIS may cause a uniform enlargement of the spleen in the early stages of the toxæmia. Later, both spleen and liver may enlarge, and the diagnosis be difficult. Ascites and anæmia may supervene.

(3) TUBERCULOSIS may occur as miliary tubercle, as an abscess, as a capsulitis, or even as multiple tuberculomata. In no case is it likely to be diagnosed apart from the existence of tuberculosis elsewhere: X-ray examination of the lungs may reveal chronic miliary tuberculosis, which may resolve in course of time after splenectomy. It is rarely primary in the spleen, and is then an exception to the rule; if diagnosed it may be operated upon. In some cases of splenic tuberculosis there is a

marked polycythæmia instead of the anæmia which usually accompanies tuberculosis.

(4) CHRONIC SEPTIC SPLENOMEGALY resembles Splenic Anæmia except in that it may present a leucocytosis. It is especially common in tropical climates after dysentery or other intestinal disorders. The prognosis is good if the causal sepsis can be eradicated. It does not as a rule lead to hæmatemesis. The importance of the condition rests in the fact that it is liable to be confused with Splenic Anæmia and a bad prognosis given in consequence.

(5) In the absence of fuller knowledge, BILIARY CIRRHOSIS (HANOT'S DISEASE) may come under this heading. The spleen may be enlarged before the liver in some cases. The diagnostic signs are considered in § 343.

(6) AMYLOID disease of the spleen is becoming very rare. It is known by: (i.) There is usually a history of syphilis, phthisis, or of chronic purulent discharge; (ii.) the liver shows signs of amyloid disease, and diarrhœa may be present, due to involvement of the intestines; (iii.) the spleen may be much larger than is usual with acute or chronic infections.

III. **Portal Obstruction or Congestion.**—Any cause of portal obstruction, of whatever degree, will naturally lead to congestion in the whole of the splanchnic area, and in this the spleen will share. Thus the spleen is slightly enlarged in (1) CARDIAC and CHRONIC LUNG DISEASE, with backward pressure in the venous system. The obstruction may be more absolute, as in (2) THROMBOSIS of the INFERIOR VENA CAVA. In this case the enlargement of the spleen may reach a greater degree than in congestive conditions of the liver, and where the thrombosis affects only the splenic vein the hypertrophy may be extreme, and the symptoms conform to those of splenic anæmia (of which, according to some authorities, it is the chief cause) (§ 544). (3) CIRRHOSIS of the LIVER (§ 342) is associated with splenic hypertrophy. (4) In SYPHILITIC fibrosis, however, the liver and spleen are usually simultaneously affected. (5) One cause of splenic congestion and hypertrophy must be mentioned, although of great rarity —viz., TORSION of the splenic pedicle. This may occur when the spleen is displaced by its increased weight (in splenomegaly), or when it has an unusually long pedicle, as in splenoptosis and wandering spleen. It is unlikely to be diagnosed except by operation.

§ **359.** IV. "**Blood Diseases,**" or diseases of myeloid and lymphatic tissue. They merit individual remark, but for full descriptions the reader is referred to other paragraphs. In almost all of these the acute attacks of capsulitis above mentioned are apt to occur.

(1) PERNICIOUS ANÆMIA is often and (2) SIMPLE HYPOCHROMIC ANÆMIA sometimes associated with slight enlargement of the spleen. This rarely reaches a large size: the presence of a large spleen would be a sign that such a diagnosis requires revision.

(3) In SPLENO-MEDULLARY LEUKÆMIA (§ 543) the spleen is characteristically enormous, but it is to be remembered that in LYMPHATIC LEUKÆMIA

and in (4) CHLOROMA it may be just as large, even reaching to the pelvis. In the latter diseases some degree of enlargement is almost invariable. These diseases are diagnosed largely by the blood examination.

(5) LYMPHADENOMA (§ 572) is known by: (i.) One or more groups of enlarged lymphatic glands are present; (ii.) the splenic enlargement is usually not excessive until a late stage of the disease.

(6) SPLENIC ANÆMIA (§ 544) could hardly be diagnosed without the enlargement of the spleen, which usually reaches very considerable proportions. As will be gathered from the remarks made above, this disease is no doubt destined to be subdivided into several groups when further knowledge is available. In the tropics it may be simulated by kala-azar and other diseases. There is a form of splenic anæmia which is found particularly in infants, and tends to occur in twins. In this the prognosis is better than in the adult form; the blood changes differentiate it.

(7) ACHOLURIC JAUNDICE is associated with some enlargement of the spleen in the majority of cases. It is known by: (i.) it is often a disease of family incidence, (ii.) the presence of jaundice, and (iii.) the characteristic blood changes (§ 328).

(8) ERYTHRÆMIA is diagnosed by (i.) polycythæmia, which may reach a very high degree, and (ii.) the cyanosis, weakness, and paræsthesiæ to which it gives rise (§ 31).

V. **Tropical Diseases.**—MALARIA and KALA-AZAR are the most common. In acute malaria the enlargement is not very great, but after many attacks it may be enormous. A history of attacks of malaria occurring in a person who has been abroad leads one to suspect the cause of the splenic enlargement; but the diagnosis is made certain by finding the parasite in the blood. Anæmia is common, and periodic fever occurring on alternate days or every third day is suggestive of malaria. In kala-azar the spleen is usually large, and is rendered the more prominent by the emaciation of the subject. The diagnosis rests on the discovery of the parasite by blood culture on rabbit blood agar medium incubated at room temperature, or in the material obtained by liver, sternal or spleen puncture, while the formol-gel reaction in the serum is characteristic. Only occasionally is it possible to demonstrate Leishman-Donovan bodies by the microscopical examination of blood smears. TRYPANOSOMIASIS and RELAPSING FEVER may also cause splenic enlargement, while splenomegaly is not uncommonly associated with INTESTINAL BILHARZIA in Japan (*S. japonica*) and Egypt (*S. mansoni*).

Rarer **parasitic** causes are HISTOPLASMOSIS and TOXOPLASMOSIS.

VI. In **Infancy** and **Childhood**, RICKETS (§ 596) is a common cause of slight enlargement of the spleen. It may depend on the catarrh of mucous membranes often associated with rickets, especially as in children the spleen enlarges much more readily than in adults, and for causes inadequate in an older person. Congenital SYPHILIS and TUBERCLE may

be present in children, and are recognised by signs of the disease elsewhere; in syphilis the liver also is enlarged. A special form of splenomegaly associated with ANÆMIA in infants (§ 551. IV) has been mentioned above; there is also a special form of KALA-AZAR in infants, encountered in the Mediterranean basin. In cyanosis from CONGENITAL HEART DISEASE there may be marked enlargement of the spleen. Congenital ERYTHRÆMIA is also described.

VII. **Irregularity** of the surface of the enlarged spleen. This group includes quite a different class of disease to that above mentioned. The most important cause of enlargement is sarcoma, for there is some hope of cure if the spleen be removed early enough. It is rare, and usually occurs in children or young adults. It can only be diagnosed by exclusion. Other new growths are even more rare, and include lymph-angioma, fibroma, pulsating angioma (which may give rise to suspicions of aneurysm), secondary cancer, and cysts such as dermoids, and congenital polycystic disease.

HYDATID cyst in the spleen may be diagnosed by (i.) the presence of marked eosinophilia in a person who (ii.) has resided in an affected country, (iii.) the serum and intradermal reactions (§ 347), and (iv.) sometimes by the presence of cysts elsewhere; (v.) the cyst may present the characteristic thrill on palpation.

LYMPHADENOMA may give rise to irregular enlargement, and certain congenital malformations are irregular.

The *Treatment* and *Prognosis* of splenic enlargement depend, for the most part, on the primary condition. The treatment of lardaceous disease and of hydatid is given under Hepatic Disorders (§§ 346, 347). The treatment of "Ague Cake" consists of (i.) removal to a non-malarious district, and the administration of anti-malarial drugs; (ii.) unguentum hydrargyri iodidi dilutum, rubbed over the splenic area, is a remedy which may be of value; (iii.) violent movement must be forbidden, as the spleen may rupture. In chronic syphilitic splenomegaly arsphenamine therapy has been curative.

§ 360. **Wandering Spleen** (Floating, Dropped, or Dislocated Spleen, Splenoptosis) may be readily mistaken for enlargement of that organ when met with in the lesser degrees of displacement. But when the dislocation is, as generally happens, considerable, it is more often taken for a floating kidney. However, the presence of the notch, the fact that it can be made to recede upwards and that it comes down in front of the colon, aid the diagnosis. The condition is mostly met with in multiparæ with pendulous abdomen. It may be accompanied by nervous symptoms, though these are less constant than in dislocation of some of the other viscera. If troublesome, the condition may be relieved by removal of the organ, an operation which has been performed several times with good results.

Atrophy of the Spleen is, as a rule, unattended by symptoms. It is, as Bristowe said, a condition not infrequently met with. It may be congenital, but its commonest causes are: I. CIRRHOSIS of the spleen, due to an increase in the interstitial tissue, and usually secondary to cirrhosis of the liver, and II. CONTRACTION OF THE FIBROUS CAPSULE, usually of syphilitic origin. The syphilitic deposits in the capsule of the spleen sometimes take on a cartilaginous change, and form plates of cartilage. It is often only found at autopsy, being unattended by symptoms during life.

§ 361. The following are the indications for SPLENECTOMY: (i.) Rupture of the spleen, torsion or similar acute emergency. These constitute an absolute indication in all cases in which the patient is likely to survive the operation. (ii.) Tumours or

abscess of the spleen which cannot be dealt with by less radical measures. (iii.) Persistent attacks of peri-splenitis or other disabilities directly dependent on the size and weight of the organ. These are a sufficient indication in nearly all cases but do not allow of operation in leukæmia or erythræmia. (iv.) Cases in which splenectomy has been shown to exercise distinct benefit on the general disease present: *e.g.*, early cases of splenic anæmia, chronic infective splenomegaly and acholuric jaundice. Cases of pernicious anæmia are not benefited, nor does Hodgkin's disease fall within this group. (v.) Cases in which the circulation through the liver is embarrassed, as in cirrhosis, and in which diminution of the volume of blood passing through the portal vein may be of advantage. Banti's syndrome falls in this group, as well as a proportion of anomalous cases described as Hanot's cirrhosis.

Of the blood diseases only three—Splenic Anæmia or Chronic Infective Splenomegaly, Acholuric Jaundice, and Purpura Hæmorrhagica (§ 653) with a great diminution in the blood-platelets—demand treatment by splenectomy. Splenectomy is undertaken only when it is evident that disease is impairing, or likely to impair the general health and working ability of the patient. Then the operation should be done early, and particularly if there has been even a single attack of peri-splenitis. Peri-splenitis causes adhesions and one attack will certainly be followed by others. What is, in skilled hands, a fairly simple and safe operation will then become much more difficult and dangerous and the patient should not be allowed to drift on in ignorance of this fact. In no case should a patient be submitted to operation without adequate preparation, by transfusion if necessary. Pulmonary collapse at the left base is especially to be guarded against after operation—by deep breathing, by avoiding constriction of the chest with bandages, etc.

CHAPTER XIII

THE URINE

A PROPER understanding of the pathological processes in the kidneys is facilitated by recalling certain physiological facts. The unit of the kidney is the nephron, comprising the glomerulus, the absorbing tubules and the collecting tubule. Under the pressure of the blood in the glomerular capillaries, the glomeruli filter a fluid similar to blood plasma, minus its proteins. The greater part of this fluid is reabsorbed into the blood stream from the tubules ; a concentrated residue of urine is left, containing the waste products. To accomplish this, only a few nephrons are in action at a time for short periods. In diseased conditions there may be a generalised inflammation or degeneration of the nephrons (nephritis or nephrosis), or a patchy destruction of groups of them owing to narrowing or obliteration of their blood supply (arterio-sclerosis).[1] Such is the enormous reserve power of the kidneys that a large part may be destroyed and yet the remainder can carry out the necessary work ; but the reserve power is diminished. With further destruction each remaining nephron is called into continuous activity. To maintain the action of the glomeruli, the blood pressure rises. The blood is still cleared of waste products (*e.g.*, urea) to a normal extent, but, as the power of the absorbing mechanism of the tubules is diminished, a larger volume of the dilute urine passes down them. With still further damage to the nephrons, less fluid is secreted by the glomeruli, and so the blood now contains an excess of waste products (uræmia) and the volume of urine falls.

When the glomeruli are inflamed, albumen and even blood leak into the urine, as in acute nephritis. Some forms of renal disease are associated with general œdema and with intense albuminuria ; modern research suggests that the primary defects are not in the kidneys, but in the composition of the blood and in the semipermeability of the capillaries throughout the body (in which the renal capillaries share). The loss of such a large quantity of albumen from the blood may so lower the osmotic pressure of the blood plasma that fluid is attracted from the blood into the tissues. The cardinal features of renal disease are therefore best seen by examination of the urine, aided later by examination of the blood and the tissues. In practice it is not always possible to separate kidney diseases proper from disorders of other parts of the urinary tract, because changes

[1] Richard Bright in 1836 described the acute and chronic forms of inflammation of the kidneys, associated with albuminuria and hyperpiesia. Unfortunately the term chronic Bright's disease is still sometimes loosely and incorrectly applied to high arterial pressure and its associated symptoms, and so is better avoided.

in the urine are common to them all. It will be necessary, therefore, to refer to disorders of the bladder, prostate, and urethra for diagnostic purposes, though their treatment is often mainly surgical.

PART A. SYMPTOMATOLOGY

§ **368.** One of the chief functions of the kidneys is the elimination of nitrogenous waste. When this is interfered with by structural or functional disease, a toxic condition results, which is known as uræmia. Other functions are the removal of water, acid products and excess of sugar from the blood, and to maintain the optimum salt concentration in the tissues.

As a consequence of the deep-seated position of these organs, the local symptoms referable to the kidney are, except in cases of Tumour or Displacement, of subordinate importance. The most constant and CARDINAL SYMPTOM of kidney disorders is some **Alteration in the Urine**, which, as an indication of renal disease, corresponds to the physical signs in other organs, and is dealt with in PART B. of this chapter. The cardinal symptoms next in order of importance are **Pallor of the Surface** and **Dropsy. Pain** and **symptoms connected with the passing of urine** are often present. **General Symptoms,** due to toxæmia resulting from the retention of nitrogenous waste generally accompany these diseases.

Pallor of the Surface and Malaise are very constant features of all organic kidney diseases. To the experienced eye the pallor differs from that of anæmia in a manner somewhat difficult to describe. The skin has a "waxy" hue, a simile which is still further exemplified when dropsy is present. It affects the whole body, but is always most evident in the face. In chronic interstitial nephritis the pallor has a greyish hue. The diagnosis from other causes of pallor will be found in Chapter XVI, § 535.

§ **369. Renal Dropsy** is of *general* distribution, in which respect it differs from cardiac dropsy, which starts in the *legs* or most dependent parts, and from hepatic dropsy, which starts in the *abdomen* (§ 29). It is, however, most evident in the loose cellular tissue—*e.g.*, around the eyelids, where it is most marked on first waking in the morning. Towards evening the ankles become œdematous, or, as the patient may express it, "a ridge is present around the top of the boot." In severe cases the eyes may be almost closed by the swollen lids (Fig. 106), and at the same time there may be signs of dropsy in the serous cavities—the peritoneum, pleura, and pericardium. Œdema of the solid organs also occurs in severe cases, and death may be produced by pulmonary œdema. Œdema glottidis is another serious though less frequent complication.

Dropsy is not an equally constant feature in all diseases of the kidney. In *acute nephritis* it is often present, and it is seen in *subacute parenchymatous nephritis* and in *nephrosis.* But in *chronic nephritis* and *lardaceous kidney* it is comparatively rare; in the former it may occur late in the course of the disease, when it is generally due either to cardiac failure, to

recrudescence of the subacute nephritis, or to secondary inflammation of the serous membranes. In uncomplicated *pyelitis* and *neoplasms* dropsy is not present.

§ 370. **Pain in the Kidney.**—Many serious diseases of the renal substance are unaccompanied by any pain or local symptoms. A sense of dull aching in the loins may be present at the onset of acute nephritis and is frequent with pyelitis, pyelo-nephritis, pyonephrosis and oxaluria. Pain may be very severe when a renal calculus is present (Renal Colic, §§ 246, 408). Various tumours of the kidney are accompanied by pain, and perinephric abscesses are associated with lumbar pain and tenderness. A pain of gradually increasing intensity in the renal area on one side, which finally becomes very severe, but is relieved suddenly with the passage of a large quantity of urine, suggests Dietl's crisis. (§ 414.) A dull, dragging pain in the lumbar region, relieved by rest in the recumbent posture, occurs with movable kidney; it is usually on the affected side, and is liable to acute exacerbations resembling renal colic. The lumbar pain of renal disease must not be mistaken for the backache due to congestion of the female generative organs, nor for lumbago, in which the pain is usually of sudden onset, is not confined to one side, and may be accompanied by other signs of rheumatism. Less frequent causes of lumbar pain are dealt with in § 457.

Symptoms *connected with the passing of urine* are: increased frequency of micturition (§§ 422 and 456), incontinence of urine (§ 422), inability to pass urine (retention § 420, suppression § 421), the passage of large quantities of urine (§ 414) or the passage of blood (§ 406).

§ 371. A large number of **General Symptoms** are consequent on failure of renal function. These may be divided into (1) early symptoms, (2) late symptoms (uræmia).

(1) *Early Symptoms* ("incipient uræmia"). These are mainly due to hypertension and its associated vascular changes. To compensate for the renal damage the blood pressure rises so as to produce an increased filtration pressure in the glomeruli. Changes in the vessel walls follow; thickening of the arteries due to hypertrophy of the muscular coat is followed by degenerative changes in the vessel walls. In consequence the following symptoms arise: (i.) Breathlessness. At first present only on exertion, it is associated with hypertrophy of the left ventricle and an accentuated aortic second sound. Later, signs of left ventricular failure may occur (§ 55), the resulting pulmonary œdema aggravating the breathlessness. (ii.) Headache accompanies most forms of renal disease, especially that which terminates in uræmia: chronic interstitial nephritis is one of the most frequent causes of headache in advanced life. (iii.) Mental disturbances such as lack of power of concentration, forgetfulness and irritability may be present as early symptoms. (iv.) Vertigo, tinnitus and various neuralgias may also be complained of. (v.) Insomnia in the aged is another common symptom of chronic renal disease. The patient readily drops off to sleep, but as readily awakes, and may do so a dozen

times every night. (vi.) Hæmorrhages sometimes occur in chronic renal disease, a consequence of the high pressure, combined in most cases with a diseased state of the blood-vessels. Epistaxis may be the first symptom which leads to the discovery of chronic renal disease. Bleeding from the stomach or intestines, and purpura, sometimes occur. *Cerebral hæmorrhage is the most frequent cause of death* in chronic interstitial nephritis. (vii.) Ocular changes may be so characteristic that a diagnosis may be made by their presence. Albuminuric retinitis is diagnosed by typical alterations in the fundi, with loss of visual acuity—œdema and swelling of the retina, papillitis, flame-shaped hæmorrhages, and white areas of degeneration. Changes in the arteries may also be seen (Plate V). (viii.) Even though the patient remains at work, there is loss of mental and physical vigour, and wasting of muscular and subcutaneous tissues.

§ **372.** (2) *Late Symptoms.*—**Uræmia** is a term used to describe the symptoms which arise from retention within the body of those constituents which, under normal circumstances, are secreted in the urine. Originally intended to indicate the retention of nitrogenous products, it is now used to include a large variety of symptoms present in renal failure: as a result toxæmia ensues. There is a retention of nitrogenous bodies (urea, uric acid, creatinine); of acids (especially sodium acid phosphate), causing respiratory symptoms; of phosphate (with a rise in blood phosphate and often a lowering of calcium), producing neuromuscular irritability and even tetany; and added to these are the cerebral changes consequent on hyperpiesis—cerebral œdema, vascular spasm or occlusion (see hypertensive cerebral attacks, § 94) producing convulsions, amaurosis, headaches, drowsiness, etc. The term uræmia therefore is used to indicate a symptom complex which may show many different features, depending on the chemical state of the blood and the condition of the cerebral vessels.

Symptoms.—(i.) Persistent headache. (ii.) Restlessness, twitching and muscular tremors are frequent: the latter may be complained of by the patient or noticed by the doctor. True tetany is sometimes seen. (iii.) Drowsiness during the day, with sleeplessness or "cat-sleeps" (dropping off for a few minutes at a time) at night. Stupor and later uræmic coma often supervenes, with or without muttering delirium. Sometimes convulsions occur before death. (iv.) Uræmic dyspnœa is in part due to the cardio-vascular changes mentioned in § 371, and also due to retention of acid products together with an altered sensitivity of the respiratory centre. The various types are: (*a*) *Paroxysmal*; the attacks coming on chiefly at night, and resembling asthma ("uræmic asthma"). The patient sits up in bed gasping for breath, but there is no cyanosis, and the mind is clear. The breathing is often noisy, with a characteristic hissing quality (Addison). (*b*) *Continuous*, or continuous alternating with paroxysmal. (*c*) *Cheyne-Stokes' Respiration* (§ 28) may last for weeks. The pulse slows in the apnœic periods, and there is alternate contraction and dilatation of the pupil, the contraction occurring during

the period of apnœa. (v.) Gastro-intestinal disturbances such as thirst, anorexia, nausea, vomiting, and often epigastric pain may be present, and diarrhœa, sometimes with ulcerative colitis, may occur towards the end. These lead to still further (vi.) Wasting, which is often extreme in the terminal stages. (vii.) There is often a uriniferous odour in the breath and a metallic taste in the mouth. (viii.) Præcordial pain due to dry pericarditis is not unusual. (ix.) A severe grade of anæmia (even to 20 per cent. hæmoglobin) is common. This is mainly due to a toxic effect on the bone marrow, but is aggravated by hæmorrhages from the nose (epistaxis), gums, bronchial mucosa and gastro-intestinal and urinary tracts. (x.) The ocular changes (§ 371 and Plate VI) become more advanced. (xi.) A low form of bronchitis or pneumonia is a common complication of nephritis. (xii.) Renal disease may be complicated by various skin affections other than dropsy and the cellulitis which is liable to affect dropsical limbs. Amongst these may be mentioned eczema, urticaria, and various forms of erythema and purpura. Undoubtedly the most fatal is an epidemic form of exfoliative dermatitis.

An acute fulminating form of uræmia occasionally occurs. It may supervene at any stage of the foregoing or may come on abruptly in an apparently healthy person (small white kidney, § 402, 2 (*b*)). Its leading symptoms are (i.) low muttering delirium, (ii.) stupor, passing into coma, with or without convulsions, (iii.) a hissing type of respiration.

Etiology.—Renal failure and uræmia may occur in almost any disease of the kidney. In acute, subacute, and chronic glomerulo-tubular nephritis it is the usual mode of death ; in tuberculous, calculous, and polycystic disease, in hydronephrosis and consecutive nephritis, in active or passive congestion, and in lardaceous disease (rarely), mentioned in order of frequency, it is also apt to supervene. Moreover, complete suppression of urine may produce death associated with symptoms of what is called *latent uræmia* (§ 421), in those relatively rare cases of removal of a solitary kidney, or obstruction of both ureters. In severe alkalosis, as may be seen with the administration of large doses of sodium bicarbonate in the treatment of peptic ulcers, occasionally in cases of repeated vomiting and following a severe hæmatemesis, uræmia may occur, especially in elderly persons, even if the kidneys are fairly healthy (*extrarenal uræmia, gastric uræmia*).

Diagnosis.—The presence of any albumen with casts in the urine indicates renal damage. The earlier stages of renal failure are best diagnosed by performing one or more of the renal efficiency tests (§ 389) : in the later stages examination of the blood, especially for its urea content, will decide the diagnosis, a value over 120–150 mgms. per cent. usually being diagnostic. The differential diagnosis of uræmic coma is dealt with in §§ 711, 716.

Prognosis.—This depends on the extent of renal damage and how far it can be removed. In chronic nephritis where the kidneys are permanently and irremedially damaged, the prognosis is grave : whereas if

uræmia is secondary to obstruction (*e.g.*, enlarged prostate) or some other cause which can be removed, the prognosis is correspondingly improved. Untoward symptoms are a greatly reduced diurnal quantity of urine, severe anæmia, emaciation, drowsiness, uræmic dyspnœa, toxic myocardial changes, vomiting or diarrhœa, and a blood urea which is rising in spite of treatment. A safe rule is that once retinal changes are present, the patient will not survive more than 18 months.

Treatment.—This depends on the symptoms and signs present. With incipient uræmia, the treatment is that of chronic interstitial nephritis (§ 403). In the later stages the main object is to eliminate or neutralise the toxic effects. To aid elimination from the blood, sweating may be induced with hot packs, or a hot air or vapour bath: also a high colon washout may be used, but brisk purges are better avoided as they exhaust the patient too much. In view of its depressant effects, pilocarpine is no longer used to induce sweating. Provided œdema is not present, copious fluid drinks (at least 5 pints a day) with added glucose must be administered. For convulsions and coma, especially in persons under 30 with increased intracranial pressure as indicated by papillœdema, lumbar puncture is essential: in older persons with hyperpiesis, venesection (½–1 pint) should be used unless anæmia is present, and if need be, the subsequent transfusion of normal saline solution will compensate for the loss of fluid by venesection or purgation. Chloral and potassium bromide, morphia in small doses, or a general anæsthetic will help to control or to prevent the occurrence of convulsions. For uræmic dyspnœa, the same sedatives, combined with bicarbonate of soda (60–120 grains a day until the urine is faintly alkaline), are necessary: and for the neuromuscular irritability, especially in the presence of a low blood calcium, calcium salts administered—even uræmic convulsions may be benefited. It is unwise to restrict the fluid intake or to lower the blood pressure excessively in uræmia, as these defeat the attempt at compensation by the kidneys. In those cases where the heart shows evidence of failure, supporting measures must be undertaken.

PART B. PHYSICAL EXAMINATION

§ 373. The **Examination of the Urine** corresponds, in renal diseases, to the physical examination of other organs.

We examine it by (*a*) observing its *physical characters* (§ 374)—viz., its appearance (*i.e.*, its colour, and whether it is clear or cloudy)—its odour, reaction, specific gravity; the presence and characters of any deposit; and its diurnal quantity. (*b*) Then by *chemical analysis* (§ 379) we ascertain the presence or absence of albumen, the presence or absence of sugar, and other substances, according to circumstances. (*c*) A *microscopic examination* (§ 391) has to be made of any deposit which may be present. (*d*) The *kidney efficiency* tests consist in part of examination of the urine, and in part of examination of the blood. The blood examinations are usually

conducted by skilled laboratory workers (§ 389). They are of special value in the detection of kidney disease where albuminuria is slight or sometimes absent. It is important in all cases—not only in cases of suspected renal disease—to observe *and to record* the condition of the urine when the patient is first seen, even when the symptoms do not suggest renal disease.

(a) Physical Characters of the Urine

§ **374. Appearance.**—The colour of the urine depends upon the proportion of pigments present. The chief pigments are urochrome and urobilin, the antecedents of which are the blood and bile pigments; but there are many others.

The urine varies from a pale yellow to a deep amber, according to the DEGREE OF DILUTION of the pigments; and, as the latter are fairly constant in quantity, a *dark urine* is commonly associated with a smaller diurnal quantity and a higher specific gravity than a pale urine. The urine is dark in excessive perspiration, acute nephritis, pyrexial states, and with diminished fluid intake, as in diarrhœa or vomiting. On the other hand, in certain diseases with *polyuria* the urine is *pale*, as in chronic nephritis, and in diabetes. With a large intake, in diabetes insipidus, and other conditions, the urine may be as colourless as water.

The colour of the urine may be altered by MORBID PRODUCTS—*e.g.*, *a dark orange colour to brown* is due to the presence of bile pigments or urobilinogen (§ 383). A *red* colour, which may be a dark red or porter colour or only a mere "smokiness," is due to the presence of blood (§ 382). *Blackish brown* colour may be due to melanin and certain oxyacids, which cause the urine to darken on exposure (§ 386). A *bright green* urine may be associated with chloroma. Intense *grass-green* fluorescence follows the ingestion of fluorescein dye (fluoresceinuria). *Milky* urine is found with chyluria and multiple myeloma. Various DRUGS affect the colour of the urine. A *dark olive-green* or *black colour* may be due to the absorption of carbolic acid—as when this is used for dressings; or it may appear after administration of creosote, salicylates, salol, tar, resorcin, or naphthol. The colour is explained by the presence of hydroquinone, which turns crimson on the addition of ferric chloride. A *reddish-brown colour* may be due to rhubarb, senna, or chrysophanic acid taken internally—all these turn red on the addition of alkali. A *bright yellow colour* follows the administration of mepacrine and santonin. A *colourless* urine is said to result from tannin taken by the mouth, and a *reddish* hue from hæmatoxylin. A *coloured* urine, from the presence of eosin, methylene blue, or other dye, may result from coloured sweets or cakes or certain proprietary pills. *Black* urine may follow the ingestion of black cherries or bilberries.

Urinary Deposits and Cloudiness will be described in § 390.

§ **375. Reaction.**—The urine should be tested soon after being passed. In normal urine an *acid reaction* is usual from the presence of acid phosphate of sodium. On standing decomposition takes place, the urea being transformed into ammonium carbonate $(NH_2)_2CO + 2H_2O = (NH_4)_2CO_3$, and hence the reaction becomes alkaline. The same change takes place within the bladder in many cases of chronic cystitis. *Alkalinity* occurs in normal urine on waking and after meals—the "alkaline tide"—due to the disodium phosphate (Na_2HPO_4) replacing the acid salt, or when alkalies are administered. A *neutral* reaction may occur under the same conditions. The reaction of the urine is often expressed in terms of pH values, and varies in health from pH 5·5 (acid urine) to pH 8·0 (alkaline urine), the neutral point being at pH 7·2.

§ **376. Specific Gravity.**—The average specific gravity of the urine in health varies between 1015 and 1025. It depends chiefly upon the percentages of urea and salts (especially chlorides) present. Extractives and pigments play only a small part; and —since the salts are fairly constant—the specific gravity, *in the absence of sugar*, gives

a fair measure of the urea present in a given sample. The specific gravity is low in granular, lardaceous, and polycystic kidney disease; high in acute and subacute nephritis, passive congestion and with glycosuria. The specific gravity is most conveniently measured in the specimen passed on waking, as it has been collecting over a period of 8–10 hours. The instrument, a urinometer (Fig. 89), must not touch the sides of the vessel, and the graduated stem should be read along the surface of the fluid, not at the place where it is raised along the stem by capillarity. When enough urine is not obtainable, and a glass bead urinometer is not accessible, mix the urine with one, two, or three times its own bulk of water and multiply the last two figures of the specific gravity by two, three, or four, respectively. For example, a mixture of one ounce of urine with *three* ounces of distilled water gives a specific gravity of 1005; the specific gravity of the urine is 1020($0{\cdot}005 \times 4 = 0{\cdot}020$).



FIG. 89. — URINOMETER, made of metal, and with flanged foot.—The flanges steady it while in the urine, and form a stand when not in use.

§ 377. The normal **odour** of freshly-passed urine is described as "aromatic"; it is very different from the ammoniacal odour of decomposing urine. The resinous portions of copaiba, cubebs, and other balsams are excreted by the urine, and impart their characteristic odour to it. Turpentine gives an odour said to resemble violets. It may smell of volatile sulphides due to the presence of some bacteria, notably *B. coli communis*, and also when cystinuria is present, especially after the urine has stood for awhile.

§ 378. **The Diurnal Quantity** varies considerably within the range of health. Normally, 40 to 50 ounces (1½ litres) are passed per diem, but the quantity depends upon the amount of fluid drunk, the action of the skin, and the activity of the renal circulation. In order to estimate the quantity of urea, and for some other purposes, it is necessary to collect all the urine passed in twenty-four hours—say, for example, from 8 A.M. Monday to 8 A.M. Tuesday. The patient should pass water *at* 8 A.M. *on Monday morning*, and this should be thrown away. All that is passed after that hour, together with what is passed *at* 8 A.M. *on Tuesday*, should be collected in one clean vessel. During the whole time it is necessary to pass water *before* going to stool, and to add this to the total collected. At 8 A.M. on Tuesday, after passing urine and adding it to that previously passed, the whole should be stirred and measured. A specimen from this should be put into a clean bottle (say, 10 ounces), and this should be labelled with the name of the patient, the date, and the total quantity passed in twenty-four hours, and sent for examination immediately. A few drops of chloroform or toluene will preserve it.

(b) *Chemical Examination of the Urine. Abnormal Constituents.*

In disease the most important abnormal substances for which the urine has to be tested chemically are albumen, sugar, blood, bile, aceto-acetic acid, acetone, and pus.

§ 379. **Albumen** is the most frequent of the pathological constituents of the urine. The variety of "albumen" usually present is serum albumen and serum globulin. Their relative amounts are of no clinical significance. The chief tests for albumen are: (1) boiling; (2) cold nitric acid; (3) Esbach's Test.

1. *Boiling.*—After testing with litmus, adjust the reaction (by the addition of alkali or 2 per cent. acetic acid) until slightly acid; then boil.

A generalised white precipitate forms on boiling if albumen is present, and is not dissolved by further addition of acetic acid. It is always best to boil the upper part of a column of urine to compare it with the lower.

The *Fallacies* of this test are: (i.) Phosphates may be precipitated by heat alone if the urine be faintly acid, neutral, or alkaline, but the acetic acid dissolves these whereas it increases the albuminous precipitate. (ii.) Excess of acid redissolves the albumen; undue natural acidity may have the same effect; all of which prove how essential it is to adjust the reaction correctly in the first place. (iii.) In acid urines a cloud sometimes appears, not on boiling only, as albumen would do, but when the acid is added, due to mucus. (iv.) Copaiba and other resins may give a precipitate insoluble in acid, but their odour is characteristic. (v.) If the urine is not quite clear, it may be necessary to filter it. If turbid from bacteria, add a trace of NaOH, and a deposit of phosphates occurs which carries the bacteria down with it. Filter and acidify again before testing.

2. *Cold Nitric Acid Test.* Pour some strong non-fuming nitric acid into the bottom of the test-tube, hold the tube in a very sloping position, and let the urine gently flow upon the top; a haze of precipitated albumen will appear at the line of junction. It is necessary to wait a few seconds for the haze to appear, when the albumen is very small in quantity; and the tube should be gently heated at the junction.

The *Fallacies* of this test are not serious. (i.) Mucin, or urates, may form a precipitate, but it occurs *above* the line of junction; (ii.) in a very concentrated urine, a haze of tiny crystals of nitrate of urea may form, but this may readily be dissolved by heat; (iii.) copaiba and other resins give a haze in a similar position, but the odour is characteristic; (iv.) the haze due to the presence of albumoses disappears on heating, and reappears on cooling; (v.) both pus and blood contain albumen, and if present in the urine, give this reaction, apart from the presence of free albumen.

3. *Esbach's Test.*—Add Esbach's solution [1] to the urine by a pipette. A precipitate indicates the presence of albumen. Alkaloids and albumoses may also be precipitated, but disappear on heating.

The *quantitative estimation* of albumen may be roughly determined by boiling as above, setting aside the test-tube for twenty-four hours, and reading off the proportion. It may be more precisely calculated by means of Esbach's albuminometer, a tube graduated for measuring the percentage of albumen. Urine taken from twenty-four hours' collection is poured into the tube up to the mark U, and the reagent is added up to the mark R. After mixing the tube is set aside for twenty-four hours, and the precipitate falls to the bottom. The level to which this reaches is then noted, and the number on the glass indicates the grammes per litre of albumen present. *Fallacies.*—(1) This method is not reliable if the specific gravity of the urine is over 1015. The urine should be diluted to 1015 or below, and a calculation made afterwards by multiplying the result by the number of times of dilution. (2) If the patient is taking alkaline salts, the urine must be first acidified before adding the reagent.

§ 380. **Mucin** is precipitated, as above mentioned, by adding dilute acetic acid which precipitates it in the cold; the precipitate is not redissolved by excess of acetic acid. Mucin is dissolved in alkaline urine. Excess of mucus indicates irritation of the bladder or genito-urinary tract, or a vaginal or uterine discharge.

§ **381. Sugar** is present in normal urine to the extent of 0·1 per cent., but the reagents used to detect an abnormal amount do not give a reaction with this normal trace of sugar. Glycosuria (sugar in the urine) is most

[1] Picric acid, 1 part; citric acid, 2 parts; water, 100 parts.

commonly due to the presence of glucose (dextrose), as in Diabetes Mellitus (§ 416), but may be due to lactose in certain cases.

QUALITATIVE TESTS FOR GLYCOSURIA (glucose, lactose or pentose).

(1) *Benedict's Test.*—The reagent consists of copper sulphate 17·3 G. sod. cit. 173 G. sod. carb. (anhyd.) 100 G. aq. dest. ad. 1000 c.c. Add 8 drops urine to 2 c.c. of the reagent and boil for 2 minutes. If a reducing sugar is present a red or greenish-yellow precipitate forms. When the test is done in this manner, a reduction is only given by a reducing sugar.

(2) *Fehling's Test.*—Fehling's solution consists of an alkaline solution of potassio-tartrate of copper, so prepared that 10 c.c. is reduced by 0·05 gramme of glucose. As it will alter on keeping, it should be boiled before using, to make certain that no precipitate forms before adding the urine (it is better to keep the copper solution and the alkali solution in separate bottles, mixing them just before using). Add to it a few drops of urine and boil again: and then continue adding till equal quantities of urine and Fehling are used. If on further boiling the solution is still clear, no noteworthy quantity of sugar is present. The Fehling's solution must always be in excess, and the boiling must not be too prolonged. If glucose or some other readily oxidisable substance is added, the blue cupric hydrate on gentle heating is reduced, and falls as a *red* or *yellow* precipitate of cuprous hydrate (Cu_2O, H_2O), which on longer boiling becomes red cuprous oxide (Cu_2O).

Fallacies.—(i.) The urine to be tested must be freed from albumen, and (ii.) it must not be ammoniacal. (iii.) Other reducing agents may occasionally give the reaction. After the administration of chloroform, chloral, morphia, and some other drugs, a reaction is obtained resembling that due to sugar, but is due probably to the presence of glycuronic acid. Lactose, uric acid and urates, ammonium salts, hippuric acid, creatinine, oxyacids and the products of certain drugs, such as carbolic or benzoic acids, may be sources of fallacy. To avoid these it is best to control by the Fermentation Test, or to filter a few drachms of the urine through a charcoal filter seven or eight times, to remove all reducing substances other than sugar.

(3) The *Clinitest Method* avoids the need of external heat and is useful to diabetic patients when travelling.

Lactose is only present in the urine during the later months of pregnancy, and during lactation. To distinguish between glucose and lactose, two tests may be used: (1) *Fermentation Test.*—Glucose is the only substance occurring in urine which is fermented by yeast. Lactose is not fermented. See that the urine is acid. Pour it into a test-tube, and insert a piece of German yeast; invert the tube over a saucer of water (or mercury) and place in a warm place (*e.g.*, on the mantelpiece). Have a control tube beside it with normal urine and a piece of yeast. If glucose is present, bubbles of CO_2 form and collect at the top of the tube. (2) *Phenyl-Hydrazine Test.*—To a third of a test-tube of urine add enough phenyl-hydrazine hydrochloride to cover a sixpence, sodium acetate to cover a shilling, and a few drops of glacial acetic acid; boil in a water-bath for half an hour. Cool by placing the tube in cold water. In the case of glucose a mass of yellow crystals forms, which under the microscope appear as fine yellow needles arranged as in a "wheatsheaf." In the case of lactose, yellow balls with fluffy edges are seen.

Pentosuria, due to a rare error of metabolism, causes a reduction of the alkaline copper solutions, but does not give the fermentation test, and the crystals found with phenyl-hydrazine hydrochloride are different from those produced by glucose and lactose. It may be tested for by Bial's reagent.

QUANTITATIVE ESTIMATION.—An approximate estimate of the amount of glucose present in urine may be formed if it is remembered that the specific gravity of a 1 per cent. solution is 1003. Thus, if from the depths of pigments present, it is judged that the specific gravity should be 1010, whereas the urinometer shows it actually to be 1031, the amount of sugar present is approximately $\frac{1031 - 1010}{3} = 7$ per

cont. (1) *Fehling's Method.*—The urine should be a sample taken from the total collection in twenty-four hours. Fill a burette with urine, diluted if necessary, and have 10 c.c. Fehling's solution in a porcelain dish, diluted with water. Boil the solution, and while boiling run in drops of urine, stirring all the while. Urine must be added from the burette till the fluid is just colourless; this is difficult to decide unless the dish be tilted so that it shows against the white background apart from the red precipitate at the bottom. Read off the amount of urine required for complete reduction, and calculate. Suppose 60 c.c. of urine, which has been diluted twenty times, are required to decolorise the 10 c.c. Fehling's solution (representing 0·05 gramme glucose), then 60/20 = 3 c.c. urine contain 0·05 gramme glucose. From this the percentage of glucose present can easily be calculated. Carwardine's Saccharimeter may be employed if an ordinary laboratory burette is not accessible.

(2) *Benedict's Method* is a modification of Fehling's method. The cuprous oxide formed on reduction reacts with potassium thiocyanate and forms a white precipitate of cuprous thiocyanate. 25 c.c. of Benedict's *quantitative* solution are measured into a conical flask; 4 G. of anhydrous sodium carbonate are added, and the urine (diluted if necessary) is run into the boiling solution until the blue colour is discharged. The solution must be kept boiling between each addition from the burette. 25 c.c. of Benedict's solution are reduced by 0·05 gramme glucose. The advantage of using Benedict's solution is that (as in qualitative testing for sugar in urine) it is not reduced by many other reducing substances present in urine.

If lactose is the reducing sugar present, when calculating the results remember that 10 parts of lactose have the same reducing power as 7 parts of glucose.

§ **382. Blood** in the urine imparts a characteristic smoky colour and may be present largely (*a*) in the form of red blood corpuscles (hæmaturia), some usually being broken up by the acidity of the urine, or (*b*) only in the form of free hæmoglobin (hæmoglobinuria). A darker colour of different shades may also be imparted to the urine by Methæmoglobinuria, Hæmatoporphyrinuria, Alcaptonuria, and Carbolic Acid. The most delicate test for hæmoglobin is the spectroscopic test (see Plate IV).

Chemical Test for Blood.—Add a few drops of freshly-prepared tr. guaiaci to the urine (which has been previously boiled and cooled) and shake; then add excess of ozonic alcohol or ozonic ether. A blue line appears at the junction of the fluids. The same reaction may be obtained by using filter or blotting-paper. Allow a drop of each of the reagents to fall on the paper beside a drop of the urine, noticing the colour at the junction of the three drops. *Fallacies.*—Saliva gives the same reaction, and so do iodides, in patients taking these salts. Pus gives a green-blue colour with guaiacum alone. Tincture of guaiacum must be freshly prepared, and it is best to dissolve a little of the resin in rectified spirit just before use.

Hæmoglobinuria is always present with hæmaturia, because the corpuscles break up. Its presence *alone* is rare, and can only be proved by examining the centrifugalised deposit of absolutely fresh urine under the microscope and finding *no red cells*, although hæmoglobin is present. Some of the hæmoglobin is converted into methæmoglobin. (See also § 409.)

Methæmoglobinuria.—The characteristic smoky colour of the urine in hæmaturia of renal origin depends largely on methæmoglobin, a substance formed from hæmoglobin by the action of acid urine. (See § 409.) It is recognised by the spectroscope.

Hæmatoporphyrinuria (Iron-free Hæmatin in the Urine).—The urine has a dark cherry-red colour like port wine, but gives no guaiacum reaction, as no iron is present. Usually albumen is absent. It is found in rare congenital conditions, and after excessive amounts of sulphonal and trional, and is an indication for at once stopping the drug and giving alkalies freely. It is known by its spectroscopic bands

(Plate IV). If these cannot be detected, the hæmatoporphyrin should be extracted with acetic ether or amylic alcohol, after adding a few drops of acetic acid.

Hæmosiderinuria occurs in some forms of severe acute hæmolysis. The tobacco-yellow deposit gives in part the reactions for iron and consists of pigment partly free and partly incorporated in leucocytes and epithelial cells.

§ 383. **Bile** is present in the urine in cases of obstructive jaundice, and can be detected even before the skin becomes yellow. Both bile pigments and bile salts are present early in jaundice, but later only the pigments are present in many cases, probably because the liver ceases to manufacture the salts. A greenish-orange colour of the urine betrays the presence of bile if in more than slight amount.

Bile pigments may be tested for by: (1) *Gmelin's Test.*—Run fuming nitric acid down the side of a test-tube containing urine. As the bile-pigment oxidises, rings of colour form red, violet and green at the top; the green indicates bile. (2) *Maréchal's Test.*—Add a few drops of very diluted solution of iodine to the surface of the urine in a test-tube; a green reaction is obtained. *Bile Salts* are tested for by *Hay's Test.* Sprinkle flowers of sulphur on the surface of the urine in a wide-mouthed vessel (not a test-tube). If bile salts are present, the sulphur sinks, instead of floating as on normal urine, because the surface tension of a fluid containing bile salts is lowered. For the same reason urine containing bile salts gives a yellow or greenish froth when shaken in a test-tube.

Urobilinogen is present in small amounts in normal urine, but in cases of hepatic deficiency or of hæmolytic anæmia is in excess of normal, giving the urine a brighter yellow or yellowish-brown tinge. It rapidly disappears in urine on standing, being converted to urobilin.

To test, fresh urine must be used. Use Ehrlich's aldehyde reagent (p. dimethylaminobenzaldehyde 10 G., conc. hydrochloric acid 100 c.c., distilled water to 300 c.c.). To 10 c.c. urine add ½ c.c. reagent, and if urobilinogen is in excess of normal a cherry-red colour develops within three minutes: this is hastened by gently warming.

Urobilin is absent in normal urine, but is slowly formed from urobilinogen on standing: when large quantities of urobilinogen are passed in pathological conditions, some urobilin probably accompanies it.

To test.—The colourless chromogen is converted into urobilin by 2 drops of liq. iodi mit. (B.P.): after acidifying with HCl, the spectroscope will detect an absorption band between the green and blue.

§ 384. **Acetone** and **Aceto-acetic acid** (often called diacetic acid) are present together in the urine in cases of ketosis. Small amounts are detected by Rothera's test and large amounts by Gerhardt's test.

Rothera's Test.—Add to 5 c.c. urine a small crystal of sodium nitro-prusside and a few drops of liq. ammon. fort., shaking well: a permanganate colour appears and gradually deepens. The sensitiveness of the reaction is greatly increased by saturation of the urine with crystals of ammonium sulphate.

Gerhardt's Test is performed by adding a few drops of a strong aqueous solution of ferric chloride, until in excess of the amount required to precipitate the phosphates, when a Burgundy-red colour appears. This same colour is given by salicylates. When urine gives a positive Gerhardt's test, Rothera's reaction develops very quickly due to the large amount of ketone bodies present.

Diacetic acid and acetone have long been known to be present in the urine in many cases of diabetes mellitus. One or both are also present in starvation and inanition, prolonged vomiting and gastro-intestinal diseases which prevent assimilation, severe acute diseases of the liver (such as acute yellow atrophy, delayed chloroform poisoning and eclampsia), in febrile states, in cases with an ill-balanced diet with a large excess of fat and insufficient sugar, and also in sea-sickness and cyclical vomiting in children (§ 271), even before the vomiting commences. In ketosis there is a deficiency in the utilisation of carbohydrates; the subsequent incomplete oxidation of fats leads to the formation of oxybutyric acid, aceto-acetic acid and acetone. **Acidosis** signifies a decrease in the fixed bases in the blood and tissues; though the blood remains alkaline, the amount of base present, as measured by the bicarbonate reserve, is diminished. It is therefore not always due to diacetic acid, although this is the commonest cause. Acidosis may also be caused (1) by the presence of other acids (as in acute rickets); (2) in kidney disease, by interfering with acid excretion; (3) by taking excessive quantities of acids by the mouth—HCl, acid phosphate of sodium—and ammonium chloride which is converted into urea and HCl. The normal ratio of acid to base in the blood and tissues is upheld by the elimination of CO_2 by the lungs and of acid by the kidneys, by the neutralisation of acid by ammonia, and by the intake of bases with food. In acidosis (whether due to ketosis or to other causes), much of the nitrogen excreted appears in the urine as salts of ammonia instead of the normal urea. Hence, the ammonia nitrogen/total nitrogen coefficient is increased from the normal 5 to as much as 30 to 50 per cent.

The clinical *symptoms* of acidosis are: (1) hyperpnœa or dyspnœa (air-hunger), without cyanosis. If it is due to ketosis, there is (2) an ethereal odour to the breath, and (3) the presence of ketone substances may be shown in the urine. Chemical tests reveal the presence, but not the degree of the ketosis. The normal urine is rendered alkaline after a dose of about 60 gr. of bicarbonate of soda, but in ketosis the urine may not become alkaline till about 1 oz. has been taken.

Treatment of Acidosis and Ketosis.—Bicarbonate of soda is given with the idea of raising the bicarbonate reserve of the blood. Water aids the elimination of the acids. Glucose must be administered in large quantities by mouth, or in the form of a 5 per cent. solution per rectum every four hours. In severe cases, the most rapid results are given by intravenous injection of 250 c.c. of sodium bicarbonate (4 per cent.) and dextrose (5 per cent.), and should be used with insulin. When the ketosis occurs with diabetes mellitus, see §§ 416, 417.

Alkalosis is the reverse of acidosis, and signifies an increase in the basic constituents of the blood (particularly $NaHCO_3$). If it occurs slowly, it is compensated for by a retention of acids, especially H_2CO_3; if rapidly, the blood actually becomes more alkaline, and so a condition of *alkalœmia* results. The usual *symptoms* are headache, nausea, vomiting, abdominal pain, thirst, mental irritability; in severe cases symptoms of tetany ensue (§ 778), ushered in with tingling of the fingers and toes; coma may lead to a fatal issue. On examination the patient looks ill, usually has a flushed dehydrated face, a furred tongue and suffused conjunctivæ; the urine may contain a trace of albumen and in severe cases acetone occurs. *Causes.*—(1) After excessive vomiting, as with pyloric and high intestinal obstruction; (2) after excessive doses of alkalies by mouth, especially $NaHCO_3$; (3) with hyperpnœa due to mountain climbing, forced breathing at rest or exposure to a hot moist climate, with excessive loss of CO_2, and therefore of H_2CO_3 from the blood.

Treatment.—The cause must be removed, vomiting allayed, the lack of chlorides compensated by large doses of normal saline solutions by mouth, per rectum, or intravenously, and acid administered in the form of ammonium chloride (50–100 gr.) or dilute hydrochloric acid by mouth, or calcium chloride (15–30 gr.) intravenously.

§ **385. Pus** in the urine is detected by the microscope: chemical tests should not be used, as they only show the presence of large amounts of pus. The presence of one or two leucocytes in the deposit of a fresh

specimen of urine is normal. When pus comes from the *kidney*, the urine is, at any rate when first passed, acid, and the pus is uniformly disseminated through the urine, and remains so for some time. When it comes from the *bladder*, the urine is usually alkaline or neutral, and the pus soon collects into a creamy layer at the bottom of the glass.

When in *very considerable quantity*, pus may be detected by the addition of an equal quantity of liq. potassæ to the deposit. A ropy gelatinous mass is formed, which pours from one test-tube to another like a fluid jelly. Another test is by the addition of a few drops of tinct. guaiaci, when a greenish-blue colour appears. *In small quantities* it is essential to examine the deposit microscopically for pus cells.

§ 386. **Other constituents** sometimes met with in urine are albumoses, homogentesic acid, melanin, indican and skatol.

Albumosuria occurs in many different conditions, *e.g.*, with abscess formation, during the resolution of pneumonia, with acute yellow atrophy of the liver, with asthma and with some cases of nephritis. It is only clinically significant in the form of *Bence-Jones' protein*, which is present in about half the cases of multiple myelomatosis or Kahler's disease.

Tests.—Slightly acidify 50 c.c. urine with 33 per cent. acetic acid. To 5 c.c. urine in each of 3 test-tubes, add 0, 1, and 2 drops of 33 per cent. acetic acid and place in a water bath with a thermometer. When Bence-Jones' protein is present, the urine suddenly becomes turbid at a temperature between 40–55° C. On boiling, in at least one tube the protein redissolves. Bradshaw's test depends on a dense white ring forming at the junction of urine and concentrated HCl in a test-tube. (Other proteins when in large quantities give a faint white ring.)

Alcaptonuria is a condition where the urine darkens from the surface downwards on standing exposed to the air or on addition of alkalies, due to the presence of *homogentesic acid*. It is due to an inborn error of metabolism, and has no known clinical significance. Its only importance lies in the fact that it reduces Fehling's solution and simulates glycosuria. (See § 653. XI.)

Melanuria occurs when there are extensive deposits of melanotic sarcoma. Fresh urine is usually colourless (melanogen) but becomes dark on standing (melanin).

Test.—To 5 c.c. of urine add 3–4 drops of freshly prepared sodium nitro-prusside solution, and 10–12 drops of 40 per cent. caustic soda and shake. Then add sufficient 33 per cent. acetic acid to acidify, when a Prussian-blue colour develops.

Indicanuria.—Indican is found where there is undue intestinal putrefaction; usually where bacterial infection is present above the cæcum. *Test.*—To 5 c.c. urine add 5 c.c. Obermayer's reagent (ferric chloride 2 parts, hydrochloric acid 1000 parts) and 2 c.c. chloroform. Shake a few times and in the presence of excess of indican the chloroform assumes an indigo-blue colour.

Fallacy.—Iodides and bile pigments also give this reaction.

Normal Constituents of the Urine.

Normally the urine consists of water containing about 4 per cent. of solids by weight, of which urea comprises from 2·5 to 3 per cent. of the total urine, amounting to about 30 grammes per diem.

§ 387. **Urea.**—A healthy male adult, weighing, say 140 pounds, excretes daily an average of 1200 to 1500 c.c. urine (42 to 53 ounces), containing 30 grammes of urea. These figures vary widely in health, and are much less for a lighter person taking less food. If the kidneys are acting well, the urea output is dependent on the intake of nitrogenous

food, but it is considerably diminished after vomiting or diarrhœa. A specimen for estimation should be taken from the urine of twenty-four hours, mixed and measured (§ 378). Deficient elimination of urea (with a rise in blood urea) occurs sooner or later in nearly all renal diseases (see Uræmia, § 372), in certain hepatic diseases, in myxœdema, Addison's disease, and melancholia. (See Kidney Efficiency Tests, § 389.)

Estimation of Urea.—On an average mixed diet, the urea excretion is about 30 grammes per diem, *i.e.*, just over 2 per cent. in the urine. For accurate results it is necessary to determine the *total nitrogen* in the urine; but since the greater proportion is in the form of urea, it is most convenient to estimate the urea excretion.

The apparatus usually employed is Gerrard's Ureameter or some modification of it. It is fitted up as shown in Fig. 90. 25 c.c. of freshly-prepared solution of sodium hypobromite are placed in the wide-mouthed jar (A). Then a small tube containing 5 c.c. of urine (B) is carefully introduced so that it stands up against the side of the wide glass jar, which is tightly stoppered. Next, the reservoir (C) is filled with water; the stopcock (E) of the cylinder (D) is opened and the reservoir raised till the water in the cylinder is at zero and level with that in the reservoir: the stopcock (E) is closed. Then the jar (A) is tilted so that the urine mixes with the hypobromite. Effervescence occurs as the liberated nitrogen enters the cylinder and displaces water, driving it into the reservoir. After allowing to cool for ten minutes, the reservoir is moved until the water in it and the cylinder are level; then the amount of gas is read. The cylinder is graduated in percentage of urea.

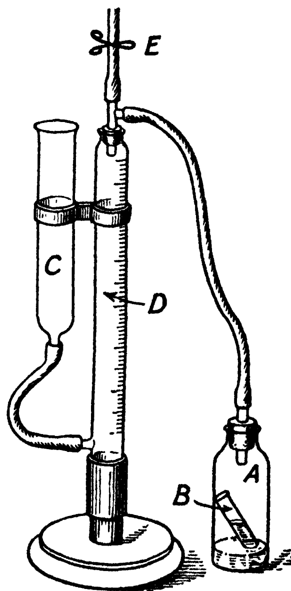


FIG. 90.—GERRARD'S UREAMETER.

§ 388. **Uric acid,** either free or combined in the form of urates, is normally present in a sample from a day's collection of urine to the extent of 0·04 grammes per cent. (or 0·7 grammes excreted per diem). Uric acid and urates separate out as a cloudiness or deposit (§§ 390 and 393) when there is high acidity. This dissolves on warming with alkali. Their chemical quantitative estimation is difficult.

CHLORIDES.—The chlorides found in the urine are principally salts of sodium, and vary in *health*, according to the food taken, from about 11 to 15 grammes daily. In *disease*, the chlorides are increased during convalescence from fevers, during the absorption of œdema or other forms of serous exudations, and in diabetes insipidus. Except in malaria, they are diminished in acute fevers, especially in pneumonia (reappearing 2 or 3 days after the crisis), in renal diseases with albuminuria, during vomiting and in anæmic conditions.

Test.—Add a few drops of HNO_3 to the urine, and an equal bulk of 3 per cent. solution of $AgNO_3$. A curdy precipitate follows if the chlorides are normal in quantity; if the urine only becomes milky, they are diminished.

PHOSPHATES (§ 393) occur in two groups: the alkaline phosphates, salts of potassium, sodium and ammonium; and the earthy phosphates, salts of calcium and magnesium. The former are readily soluble; the latter are readily deposited when the urine is neutral or alkaline, especially when heated.

Tests.—In an *alkaline* or neutral urine, the earthy phosphates form a cloudy precipitate, which is increased on boiling, but disappears on acidifying the urine. If present in an alkaline urine the deposit is distinguished from pus by being dissolved by acetic acid. The microscope enables us to distinguish between pus and phosphates, and is indispensable when, as often happens, the two deposits occur together.

SULPHATES are also normally present in the urine, and there is an increase with increase of protein in the diet or in fever. Two forms exist: (*a*) as potassium or sodium sulphate (*inorganic sulphates*); (*b*) as combinations of cresol, phenol, indol, skatol, etc. (*organic or ethereal sulphates*). A relative increase of the latter group occurs when phenol or allied substances are given as drugs, and with intestinal putrefaction.

§ 389. **Kidney Efficiency Tests.**—Certain tests yield invaluable evidence as to the condition of the kidneys. These have usurped the position of the older tests for the amount of urea in the urine, which was formerly regarded as the chief source of information. Several of these tests should be confided to the skilled laboratory worker. This is true especially of the examination of the blood for protein and non-protein nitrogen.

1. *Estimation of Blood Urea.*—The most accurate method is by means of urease in the soya bean, but for practical purposes a much more rapid method is by means of sodium hypobromite. *Ambard's Method.*—Into a 25 c.c. measuring flask deliver approximately 10 c.c. of 20 per cent. trichloracetic acid. To this add 10 c.c. of blood, and make up to the 25 c.c. mark with water. Filter off the coagulated protein, and take a measured quantity (10–15 c.c.) of the clear filtrate. Pour this into the urea apparatus by squeezing some air out of the rubber bag, filling the cup with the solution, and introducing this into the apparatus by releasing the pressure on the rubber. Add a few drops of phenolphthalein and sufficient strong caustic soda to

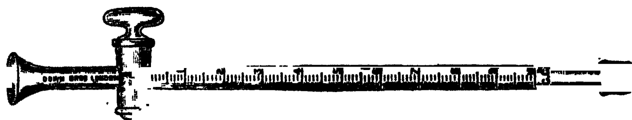


FIG. 91.—AMBARD'S APPARATUS FOR ESTIMATING BLOOD UREA. (½ scale.)

make the contents alkaline. By pressing on the rubber bag, drive all the air from the apparatus to the top of the tap. Then fill the cup to the level of the neck with sodium hypobromite, and release this into the apparatus, taking care that no air enters. Invert the apparatus several times till all the nitrogen has been evolved, and measure the quantity of this by releasing the bag from the apparatus in a cylinder of water, and measuring the quantity of gas when the levels of fluid are the same inside and outside the apparatus. Suppose 13 c.c. of filtrate were used, and evolved 0·8 c.c. of nitrogen. This amount of filtrate was derived from $\frac{13 \times 10}{25}$ c.c. of blood. Knowing that 1 gram of urea liberates 354 c.c. nitrogen at N.T.P., the amount of urea present in the blood is readily calculated. Normally the blood has 20 to 50 mgms. of urea per 100 c.c. In renal diseases it rises considerably higher; about 100 mgms. per 100 c.c. blood may be taken as definitely pathological. A high blood urea may occasionally occur without renal disease (§§ 372 and 387); then the prognosis varies with the cause. In doubtful cases the urea excretion should be measured.

The urea concentration test and the urea clearance test will help to decide whether the condition is due to disease of the kidney. Usually there is urea retention in the blood with chronic interstitial nephritis but not with subacute parenchymatous nephritis.

2. *The Urea Concentration Test* introduced by Maclean is a valuable guide to the function of the kidney. The bladder is emptied, and the patient drinks 15 G. urea dissolved in 100 c.c. water. The urine passed at the end of 1, 2 and 3 hours is separately collected, measured and the urea percentage in each specimen estimated. The volume in the first hour may be increased by the diuretic effect of urea, and if it exceeds 120 c.c. the result should be discarded. The second sample is often the more important because of this diuresis. Above 2·5 per cent. urea in any sample means normal concentration. In chronic interstitial nephritis there is less; serious cases show a concentration of only 1·0 to 1·5 per cent. urea. The fallacy of this test is that it may not show even severe grades of renal insufficiency in certain cases, particularly in subacute parenchymatous nephritis, because the urea is concentrated, owing to the small output of fluid. This is partly overcome by estimating the total urea excretion in each hour. In at least one period 10 per cent. of the ingested dose of urea is secreted by the normal kidneys. More accurate estimation is by the urea clearance tests.

3. *Van Slyke's Method for Determining Urea Clearance.*—The test is best performed between breakfast and lunch. Before the test, vigorous exercise must be avoided, and the previous meal must have been moderate and without coffee. Just prior to the test, and again 1 hour later, a glass of water is given. At a noted time, the bladder is emptied; at the end of the first hour a sample of blood is taken for blood urea estimation; at about the end of the first hour, urine is passed, the time recorded, and the whole specimen put into a labelled bottle. At the end of a second hour urine is again passed, and the whole specimen put into another labelled bottle.

For persons over 16 years, and of average build, the following methods are used to calculate the results (in others, correction factors have to be applied).

$$\text{The Maximum Clearance} = \frac{\text{per cent. urea in urine (U}}{\text{per cent. urea in blood (B)}} \times \frac{\text{Vol. of urine in c.c.}}{\text{per minute (V)}}$$

is used where the output exceeds 2 c.c. per minute, the normal values being 64–99.

The Standard Clearance $= \frac{\text{U.}}{\text{B.}} \sqrt{\text{V.}}$ is used where the output is less than 2 c.c. per minute, the normal values being 40–68.

4. *The Phenolsulphonophthalein test* can also be used to detect whether the kidney function is impaired. After the patient has drunk 300 c.c. water, inject intramuscularly 6 milligrammes of the dye, and examine the urine 70 and 130 minutes after. If 40 to 50 per cent. of the dye is excreted in an hour, and 70 to 90 per cent. in two hours, the kidneys are not diseased.

(c) *The Urinary Deposit.*

§ 390. Cloudiness of the Urine (naked-eye examination).—In healthy urine there is no deposit, but many of the normal constituents, and some abnormal substances, may become evident as a sediment or turbidity after the urine has cooled.

(1) A bulky pinkish turbidity and deposit in an acid urine, which form when the urine cools, indicates the presence of *urates*. It is the commonest of urinary deposits. (2) *Uric Acid* is evident to the naked eye as a sandy deposit resembling red cayenne pepper. (3) A white flocculent turbidity in an alkaline or neutral urine indicates the presence of *phosphates*, which are cleared at once by the addition of a few drops of 2 per cent. acetic acid. (4) *Calcium oxalate* gives a typical "powdered-wig"

deposit of fine white points seen on the surface of a mucous cloud. (5) A fine cloud of *vesical mucus* is normally present in the urine, although it is only visible when the entangled débris and epithelial cells are sufficiently plentiful. (6) *Pus* forms a deposit which resembles phosphates to the naked eye, but it is readily distinguished under the microscope. (7) *Prostatic threads* ("floaters") are elongated fine white threads which float in the urine and indicate chronic prostatitis. (8) Urine is sometimes cloudy from the presence of *bacteria*, and this cloudiness cannot be cleared by boiling or the addition of acids.

§ 391. **Specimens** of the deposit must always be examined **microscopically** in cases of suspected renal disease. The urinary deposit is best examined after the urine has stood for some hours in a conical glass, or after the specimen has been centrifugalised. Take a pipette, close it at the top with the right forefinger, pass it to the bottom of the glass, allow a small quantity of the sediment to enter, withdraw the pipette, wipe its exterior with a cloth, place the point on a slide, then surround the pipette with the palm of the left hand, the warmth of which will cause a drop to exude. Cover the drop with a cover-glass, and examine first under a ⅓ or ½ inch objective, then under a ⅙ or higher. The deposit normally contains foreign substances, such as cotton and woollen fibres, etc., and a few bladder (and in women nearly always a few vaginal) epithelial cells, which are recognised by their large and nucleated appearance. Inquiry should always be made as to the sex of the patient, and in women if any leucorrhœa is present. If so, it is very desirable to draw off a specimen of urine by the catheter.

The urinary deposit may contain ORGANISED SUBSTANCES (§ 392), or CRYSTALLINE and inorganic substances (§ 393).

§ 392. The **Organised Constituents** of the urinary sediment are of far more serious import than the crystalline substances. They comprise TUBE-CASTS (which are the most important), EPITHELIAL CELLS, PUS CELLS, BLOOD CELLS, spermatozoa, and certain rarer structures such as bacteria, fat cells, etc.

Tube-casts and renal **Epithelial Cells** are present in all renal maladies attended by shedding or destruction of the renal epithelium. The casts are composed of blood cells or renal epithelial cells moulded together in the convoluted tubules during the absorption of water. When tube-casts are abundant in the urine microscopic examination of the sediment permits of their ready detection. But if, on the other hand, they are present only in small numbers, they may be easily overlooked, especially when, as in chronic interstitial nephritis and in amyloid disease, the urine is abundant and of low specific gravity, so that any suspended matter is deposited only slowly and incompletely. Moreover, these are the exact instances in which the casts are apt to be of the hyaline variety, and their almost transparent character renders them inconspicuous objects in the microscopic field. To detect the presence of casts a fresh specimen of urine is often essential, as they rapidly disappear in alkaline or decomposing urine.

THE SEARCH FOR TUBE-CASTS must be conducted with great care if the risk of a false conclusion is to be avoided. One of the best methods, after settlement or centrifugalisation of the deposit, is to examine it with a moderately low power of the microscope, using a narrow diaphragm and shading the light so as to have the field

only feebly illuminated. Any suspicious-looking object can be brought into the centre of the field and examined with a stronger lens. In this way casts may be detected which in a strong light would readily be missed, and if several slides have been prepared and examined in this manner the detection of any casts present in the urine is rendered fairly certain. But the examination should be repeated on several occasions in any urine containing albumen before a negative conclusion is finally arrived at. The addition of a few drops of methylene blue to the urine before centrifugalisation is of assistance. The casts do not stain at first, but in those containing cells the nuclei stain; and the casts stand out against the pale blue background of the fluid.

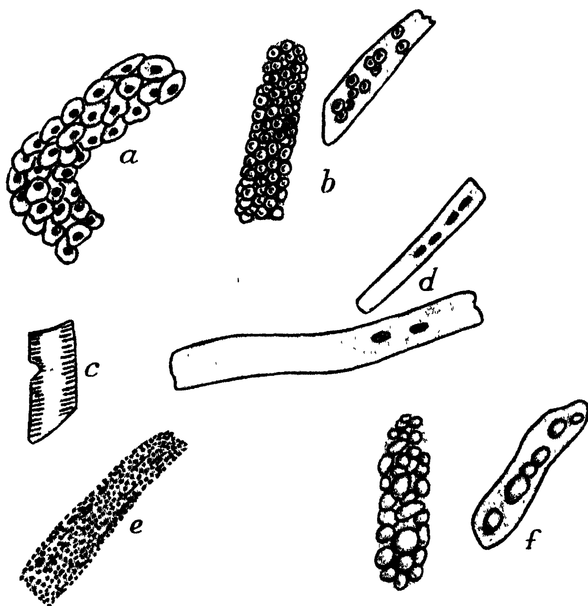


FIG. 92.—RENAL TUBE-CASTS.—*a*, epithelial casts; *b*, blood casts; c, waxy cast; *d*, hyaline casts; *e*, granular cast; *f*, fatty casts.

The clinical importance of tube-casts in the urine is that, with but few exceptions, they definitely indicate disease of the renal epithelium. Thus, when found in a urine containing albumen, they indicate that the albuminuria is a result of some structural change in the kidney. Similarly in cases of pyuria and hæmaturia the detection of tube-casts not only suggests that the pus and blood are of renal origin, but that the kidney is affected. It must be remembered that more than one part of the urinary tract may be diseased at one and the same time. In the urine of patients who are jaundiced, tube-casts may often be found without, either at the time or subsequently, any evidence of renal disease.

In general terms, *epithelial casts* and *blood casts* are indicative of the earlier and more acute stages of nephritis. *Waxy casts* are not peculiar to lardaceous kidney, but occur in other forms of long-standing renal disease. These and *fatty casts* indicate that the inflammatory process is passing to a degenerative stage. *Granular casts*

are more abundant in chronic renal disease. *Hyaline casts*, which must not be confused with waxy casts, occur in all forms of nephritis, both acute and chronic. The relative proportion of epithelial, granular, and hyaline casts (Fig. 92) is affected by the condition of the urine. (In highly acid and in alkaline urine the casts tend to be hyaline; in acid urine, granular.) Tube-casts in abundance always form a serious symptom, but one or two hyaline casts occur in normal urine. They are more abundant in the acute than the chronic forms of renal disease. Their *absence* does not count for very much, as they may be easily missed or undergo disintegration in the urine. The continued presence of hyaline and granular casts is more serious than the temporary appearance of other types.

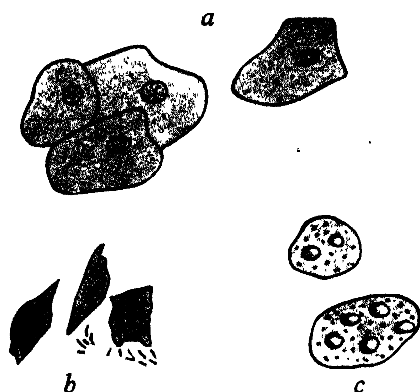


FIG. 93.—RENAL EPITHELIUM—*a*, normal; *b*, disintegrating; *c*, fatty.

Renal Epithelium (Fig. 93).—The detection of renal epithelium in a urinary deposit has much the same significance as the presence of tube-casts. The cells are *spherical* and rather smaller than bladder or vaginal epithelium. They may be seen isolated or in small groups. In acute nephritis they may be found in an unaltered condition, but in chronic disease they become degenerated, and may thus appear crowded with fat globules. BLADDER OF VAGINAL EPITHELIUM (Fig. 94) is met with as collections of squamous cells; transitional, spindle-shaped. and other forms of epithelium may also be derived from the bladder. TAILED EPITHELIUM may be derived from the pelvis of the kidney: the

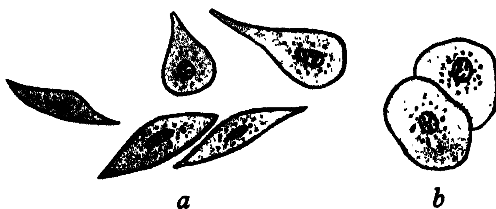


FIG. 94.—TAILED EPITHELIUM (*a*) from the pelvis of the kidney; and BLADDER EPITHELIAL CELLS (*b*).

male urethra and the prostate gland yield epithelium practically identical with this.

Pus Cells, under the microscope, are of globular form with a diameter about one-third larger than that of a red blood cell: they are opaque and granular, but when treated with acetic acid they clear, and a nucleus is seen (Fig. 95, *d* and *e*). Pus cells may or may not accompany bacilli in the urine.

Red Blood Cells.—The detection of red blood cells in a urinary deposit

is conclusive evidence of the presence of blood. When only in small numbers, they may be seen microscopically, but do not give the chemical reactions (§ 382). In most fresh urines they are readily distinguished, as they retain their bi-concave form and the outline shows a double contour (Fig. 95, *a*). But sometimes the cells become much changed. Thus in a very dilute urine they are apt to become distended by imbibition, and then are seen as circles having sharp delicate outlines (*c*). In other instances they become crenated, shrunken, and deformed (*b*).

Spermatozoa may occasionally be found in the urine. Each has a minute oval or pear-shaped head, from the larger extremity of which there passes a long and delicate tail. The total measurement of the spermatozoon is about $\frac{1}{800}$ inch in length.

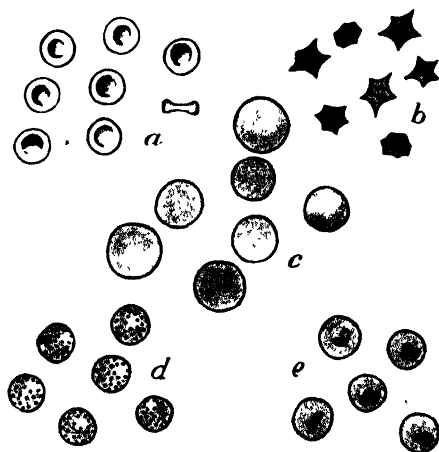


FIG. 95.—Various appearances of RED BLOOD CORPUSCLES and PUS CELLS in the URINE.—*a*, normal red blood corpuscles; *b*, crenated; *c*, in hypotonic solution; *d*, pus cells; *e*, pus cells + acetic acid. In very pale, watery urine the red corpuscles may be so pale as to escape detection (*c*). They may then be revealed by adding a solution of iodine in potassium iodide.

Micro-organisms.—Numerous organisms are found in the urine, especially when decomposition has occurred within the bladder. *B. Coli* is much the commonest (§ 412): other organisms found are *B. proteus*, *B. pyocyaneus*, *B. subtilis*, streptococci, staphylococci and occasionally gonococci, and tubercle bacilli (see § 411). The *Typhoid bacillus* may be abundant in cases of typhoid fever, and long after health is restored it may remain a potent source of infection to others.

The *Tubercle bacillus* may be found in tuberculous disease of the bladder or of the kidney, and is therefore a sign of great value. In appearance under the microscope it resembles the smegma bacillus. Its presence should always be suspected when pus cells are abundant and yet the urine remains sterile on culture. Its special staining reaction is given in § 921. It is difficult to find in the urine early in the disease and the deposit of a 24 hours' specimen should be examined. In obscure cases a guinea pig should be inoculated or a culture made (§ 919).

§ 393. **Crystalline and Inorganic Deposits** in urine are usually of less serious import than the organised substances above noted.

In ACID URINES we meet chiefly urates, uric acid, oxalates, and, more

rarely, stellar phosphates, cystin, xanthin, hippuric acid, tyrosin, and leucin.

In neutral or ALKALINE URINES we meet chiefly triple phosphates (occasionally ammonium urate and calcium carbonate).

In urines of EITHER REACTION amorphous deposits of potassium or ammonium urate and phosphates and carbonates of the alkaline earths may be met.

1. URATES, chiefly of sodium, potassium, or ammonium, when in excess are deposited as an amorphous brick-coloured deposit after the urine has become cold. On heating or on the addition of caustic potash, the deposit clears, both tests distinguishing urates from phosphates. The characteristic forms are shown in Fig. 96.

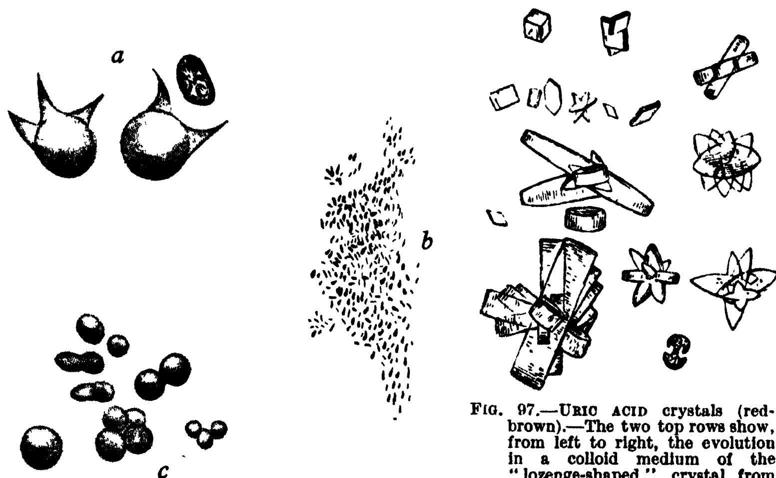


FIG. 96.—URATES.—*a*, "Hedgehog" crystals of sodium urate; *b*, amorphous urates; *c*, ammonium urate crystals (found in alkaline urine).

FIG. 97.—URIC ACID crystals (red-brown).—The two top rows show, from left to right, the evolution in a colloid medium of the "lozenge-shaped" crystal from the primary rhombic prism. In the lower right-hand corner is the "dumb-bell" form occasionally met with.

An occasional deposit of urates in a concentrated urine is of no importance. When they are *constantly* present a calculus may form in the kidney or bladder.

2. FREE URIC ACID is deposited when the urine is very acid or poor in salts and in pigment, and is therefore found chiefly in dilute pale urines with deficiency of salts. The red deposit of uric acid closely resembles cayenne pepper to the naked eye. It may be detected in the urinary deposit under the microscope by the *colour* and *shape* of the crystals. It occurs in the form of *red-brown crystals* (the only coloured crystals commonly found in the urine) (Fig. 97). Uric acid assumes many different shapes, owing to the presence of the colloid substances in the urine. This deposit is soluble in caustic potash, insoluble in dilute acetic acid, the converse of phosphates.

In health uric acid is increased with a highly nitrogenous diet, after much exercise, after meals, and during the "alkaline tide" of the morning. It is also increased after any excess of purin intake, in most fevers, in liver diseases, and during and after acute gout. It is diminished in chronic gout, especially just before the acute exacerbations and in chronic nephritis.

3. PHOSPHATES occur as a white deposit or flocculent turbidity in FEEBLY ACID, NEUTRAL, or ALKALINE urine, in three different forms, which in order of frequency

are (1) *Amorphous phosphates of calcium* form the thick white deposit that is apt to be mistaken for pus, but which is more readily shaken up in the urine. These and all other phosphates are soluble in acetic acid (which distinguishes the deposit from pus). (2) *Triple phosphate* of ammonium and magnesium (Fig. 98) is found in urine which has undergone alkaline fermentation. In markedly ammoniacal urine " feathery phosphates " are found. (3) *Basic magnesium phosphate* occurs in large rhombic plates, not grouped, but scattered. (4) *Neutral or dicalcium phosphate* occurs in neutral or alkaline urine as " stellar phosphates " (Fig. 100). They decompose on the addition of ammonia. The constant presence of phosphate deposits may be

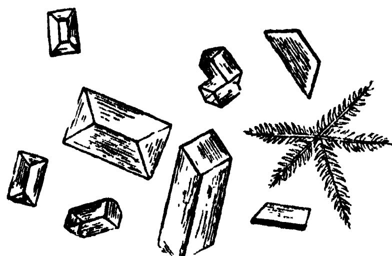


FIG. 98.—TRIPLE PHOSPHATE—" house-top " and " feathery " crystals.

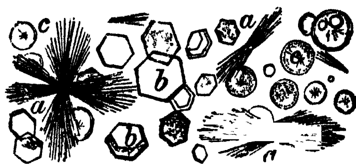


FIG. 99.—*a*, TYROSIN, in bundles of needle-shaped crystals; *b*, CYSTIN (clear six-sided plates) is a rare urinary deposit due to an inborn error of metabolism. It may form renal calculi; *c*, LEUCIN, spherical crystals with concentric markings, found in the urine in rare cases of acute yellow atrophy of the liver.

associated with symptoms of phosphaturia (§ 423), and usually does not indicate excess eliminated, but only alkalinity of the urine. *Monocalcium phosphate* occurs chiefly in acid urines.

4. OXALATES are chiefly met as *calcium oxalate* (Fig. 101). They are soluble in hydrochloric acid, insoluble in acetic acid or caustic potash. The presence of crystals of calcium oxalate is not necessarily indicative of an excess (OXALURIA, § 423); their presence may suggest the nature of a calculus.

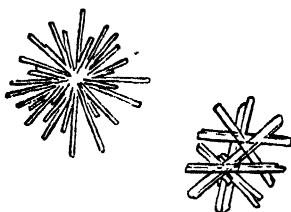


FIG. 100.—NEUTRAL OR " STELLAR " PHOSPHATE.



FIG. 101.—CALCIUM OXALATE—" envelope " and " dumb-bell " crystals.

5. *Calcium Carbonate* is a rare deposit, consisting of tiny spheres and dumb-bells, or of amorphous granules, effervescing and dissolving in acetic acid. The *Carbonates of the Alkaline Earths* are rarely found as tiny amorphous granules or concretions. Calcium sulphate and carbonate may take part in the formation of vesical calculi, especially in the aged, but otherwise they have no clinical significance. Their presence indicates the composition of a calculus.

When a patient is taking crystalline drugs, such as potassium acetate and sodium phosphate, or even liquor ammoniæ, crystals without pathological significance sometimes appear in the urine. After giving sulphonamide drugs, characteristic crystals are seen. Moreover, after a reagent has been added to urine (*e.g.*, Esbach's solution for estimating albumen), and it has been set aside, crystals may appear which have no clinical significance.

6. *Certain rare and less important deposits*, which occur chiefly in acid urines, are as follows: *Hippuric Acid* occurs as four-sided prisms, either scattered or in groups. It is present after the ingestion of benzoic acid in large doses, cranberries, and other fruits. *Calcium Sulphate* occurs either as amorphous granules, or, very rarely, as long colourless needles or elongated tables with truncated ends. It is detected by being insoluble in ammonia and acids. *Leucin* occurs as laminated spheroids, and *Tyrosin* as bundles of acicular crystals (Fig. 99). Both occur occasionally in the urine in phosphorus poisoning, acute yellow atrophy of the liver and other causes of liver destruction. *Cholesterin* (Fig. 86) is only occasionally found among urinary deposits. It forms laminated plates with longitudinal striæ, and a notch at one end. *Cystin* occurs as hexagonal plates soluble in ammonia (Fig. 99), in large amount in congenital cystinuria.

PHYSICAL EXAMINATION OF THE KIDNEYS

§ **394.** A dull "sickening" pain is usually felt on firmly compressing the kidney with both hands, but there is no tenderness in a healthy organ. Tenderness may be elicited in cases of calculus and other forms of pyelitis, perinephric inflammation, abscess, or tumour of the organ, and in "dropped kidney" in neurotic subjects. Kidney tumours tend to grow forwards, where there is least resistance, pushing the resonant colon *in front* of them. When, therefore, the palpating hand encounters resistance and swelling in the lumbar region *posteriorly*, it is probably due to a peri- or extra-renal, rather than to a renal condition (see Fig. 41). The diagnosis of renal swellings from other abdominal tumours has been given in § 263. An extra-renal tumour may press the kidney backwards, so that the apex of the tumour may be due to the displaced kidney.

In the majority of renal disorders the physical examination of the kidney is of secondary importance to the examination of the urine. The kidneys are situated on either side of the spine, about 3 inches from the middle line; the right is slightly lower than the left, owing to the position of the liver just above it. The upper end of the right kidney reaches to the *lower* edge of the eleventh rib; the left kidney reaches as high as the *upper* edge of the eleventh rib. The kidneys lie partly in the hypochondriac and partly in the lumbar regions, and are therefore much higher than is commonly supposed, with reference to the anterior abdominal wall. The lower end of the right kidney is 1 inch and that of the left kidney 1½ inches *above* the level of the umbilicus.

Palpation.—Even in normal conditions the lower border of the right kidney may be palpable in thin people. In those whose abdominal walls are lax—in women who have borne children, for instance—it is surprising how frequently the right kidney can be palpated. The patient should lie on the back, with the abdominal muscles relaxed. The physician, standing on the right of the patient, should place his left hand beneath the patient's back, close under the ribs, just external to the quadratus lumborum. The right hand is laid flat over the anterior surface of the abdomen, in the mid-clavicular line, with the fingers pointing upwards, just below the liver. Pressure backwards, as if to meet the left hand, is made by the right hand. The patient should then be asked to draw a deep breath, and as he does so the rounded lower edge of the kidney is felt to slip between the opposing hands. When the ligaments of the kidney are relaxed—*movable kidney*—the fingers of the right hand may be able to palpate the upper border of the organ, and to retain it during expiration. A kidney is said to be "*floating*" when it can not only be readily palpated, but can be pushed below the umbilicus or freely moved about in the abdominal cavity.

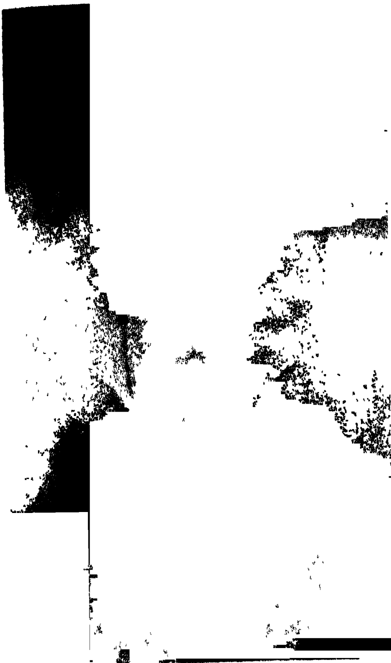


FIG. 102.—NORMAL RETROGRADE PYELOGRAPHY.

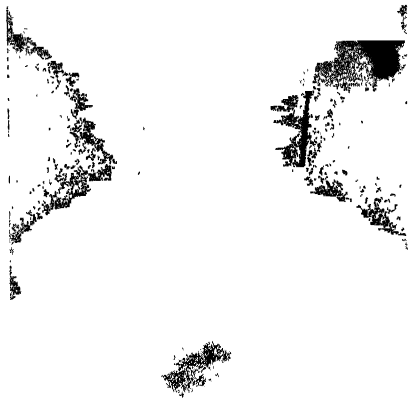


FIG. 103.—LARGE BILATERAL BRANCHED RENAL CALCULI ("coral calculi"), with ureteric catheters in position.



FIG. 104.—BILATERAL POLYCYSTIC KIDNEYS, demonstrated by retrograde pyelography. (X-Rays kindly supplied by Dr. J. Russell-Reynolds.)



FIG. 105.—BILATERAL HYDRONEPHROSIS in a child, with demonstration of renal pelves with iodoxylum (excretion urography).

Percussion does not enable us to define the margins of the kidney, for the organ is too deeply seated. The feature of primary importance in this connection is its relation to the colon, which is pushed forward by enlargement or tumour of the kidney. Consequently the anterior surface of such growths is always resonant, there being dulness at the side which is continuous with that at the back; whereas with enlargements of the spleen or gall-bladder there is dulness anteriorly and resonance at the side.

Radiography is often of great help. In cases of doubtful renal calculus a *radiogram* usually settles the diagnosis. By *pyelography* an outline of the pelves of the kidneys, and of the ureters can be obtained. In retrograde pyelography, a 10–15 per cent. solution of potassium iodide is injected through a ureteric catheter, and on X-ray examination an opaque shadow is thrown where the solution has penetrated (Fig. 102). With *excretion urography* iodoxylum B.P. (uroselectan B.) is injected intravenously in doses of 15–20 c.c. of a 75 per cent. solution in water, and X-ray examination 5, 10, 30 and 50 minutes later gives information as to the secreting power of the kidneys and a picture of the outline of the whole renal tract (Fig. 105). Care must be taken that no drop enters the tissues round the vein. Diodone (B.P.) is a new preparation for intravenous or intramuscular injection.

Cystoscopy reveals the condition of the bladder and of the ureteric orifices: the orifices may be the seat of congestion or ulceration. The previous injection of indigo-carmine or methylene blue may make the differences of the flow from the orifices more obvious (*chromo-cystoscopy*). Through the cystoscope the ureters may be catheterised and a separate specimen of urine obtained from each kidney.

PART C. URINARY DISORDERS, THEIR DIAGNOSIS, PROGNOSIS, AND TREATMENT

§ 395. Routine Procedure and Classification.—*First*, having ascertained that the patient's LEADING SYMPTOM refers to the urinary apparatus; and, *secondly*, the data of his ILLNESS, particularly as to whether it is of an ACUTE or CHRONIC nature; we proceed, *thirdly*, to examine the urine. The ROUTINE EXAMINATION of the URINE in everyday practice consists of Inspection, Reaction, Specific Gravity, Tests for Albumen and for Sugar. The subsequent more detailed examination depends upon circumstances. As stated, the examination of the urine stands in relation to renal disease, as the local signs do to diseases of other organs. Few diseases, certainly no common disorders of the kidneys, are unattended by some change in the urine. On the other hand, the LOCAL EXAMINATION of the kidney, by palpation, percussion and by radiography, is more difficult, but should never be omitted in any case which is at all obscure.

Classification.—We will deal with urinary disorders under their respective cardinal symptoms as follows:

Albuminuria	§ 396
Hæmaturia	§ 406
Pyuria	§ 410
Alterations in the specific gravity	§ 413
Polyuria	§ 414
Glycosuria	§ 415
Retention of urine	§ 420
Suppression of urine	§ 421
Incontinence of urine	§ 422
Presence of various deposits	§ 423
Renal enlargements	§ 424

§ 396. **Albuminuria.**—The causes of albuminuria come under these groups:

A. The ALBUMEN IS ASSOCIATED with BLOOD and CASTS and the disease is **acute**: Acute nephritis (§ 397).

B. The ALBUMEN IS PERSISTENT and is ASSOCIATED with CASTS; BLOOD is present microscopically, and ŒDEMA may be marked; the disease is **subacute**: Subacute parenchymatous nephritis: § 398.

(a) With marked œdema.
(b) Without marked œdema.

C. The ALBUMEN is ASSOCIATED with CASTS; BLOOD is USUALLY ABSENT and the disease is **chronic**: § 399.

(a) Secondary contracted kidney.
(b) Chronic interstitial nephritis.
(c) Amyloid disease (§ 404).

D. The ALBUMEN is NOT USUALLY ASSOCIATED with CASTS or BLOOD; it may be INTERMITTENT, and the condition is usually **chronic**—I. physiological; II. orthostatic; III. kyphotic; IV. toxæmic; V. pregnancy; VI. drugs; VII. endogenous poisons; VIII. chill to the surface; IX. mild renal congestion; X. chronic gout and arteriosclerosis; XI. urinary calculi and crystals; XII. leaky kidney or residual albuminuria; XIII. anæmia; XIV. obscure causes (§ 405).

E. The ALBUMEN IS ASSOCIATED with BLOOD, and CASTS are SCANTY or ABSENT—Hæmaturia (§§ 406–409).

F. The ALBUMEN IS ASSOCIATED with PUS—Pyuria (§ 410).

A. *The illness came on recently and is* **acute**; *the urine contains a considerable quantity of* ALBUMEN *and* TUBE-CASTS: *it is or has been* "SMOKY" *from the presence of blood. The disease is* ACUTE NEPHRITIS.

§ 397. **Acute Nephritis** (Synonym: Acute glomerulo-tubular nephritis; formerly called Acute Bright's Disease).—In this disease the inflammation begins and predominates in the glomeruli and to a less extent in the tubules (the parenchyma) of the organ. The condition usually lasts five or six weeks, and may terminate in recovery or pass into a subacute condition. The disease exists in two forms: (a) acute diffuse glomerulo-tubular nephritis; (b) acute focal glomerulo-tubular nephritis.

(a) **Acute diffuse glomerulo-tubular nephritis** is due to an intoxication of the kidney, usually one to three weeks after an acute hæmolytic streptococcal infection. The diffuse involvement causes temporary renal failure.

Symptoms.—(1) The albumen is often in considerable quantity, and the urine may even "go solid" on boiling. (2) The other characters of the urine are: (i.) It is scanty, sometimes only 10 or 20 ounces a day, or less. Consequently, the specific gravity is high, although the diurnal quantity of urea is diminished. (ii.) It varies from a turbid or "smoky" to a dark brown hue from the presence of blood. (iii.) Epithelial, hyaline, and

blood casts, free renal epithelium, and red and white blood-corpuscles are present. (3) Dropsy is usually moderate in extent and severity, and is first noticed in the loose areolar tissue below the eyes, in the legs and back and in the genitals. There may be collections of dropsical fluid in the serous cavities. (4) There is a waxy pallor of the skin. (5) A degree of malaise, with discomfort and even pain in the loins or abdomen, may be present, but there is only a slight elevation of temperature for about four or five days: mild anæmia is common. (6) Uræmic symptoms may come on early—*e.g.*, (i.) occasional vomiting, (ii.) headache, (iii.) drowsiness, (iv.) some shortness of breath, (v.) the blood urea is often raised. (7) In the course of a few days the blood pressure may become high, and the second aortic sound accentuated.

Etiology.—(1) Acute infection is the commonest cause. The micro-organism is usually streptococcus hæmolyticus, commonly found in the tonsils, rarely in the respiratory passages, nasal sinuses or middle ear, or in the skin (*e.g.*, erysipelas). This explains its common occurrence with scarlet fever. Other acute infections are influenza, typhoid, malaria, cerebro-spinal fever, staphylococcal infection and trench fever. (2) Hidden foci of sepsis, cholecystitis, empyema, etc. (3) Sudden chill may predispose. (4) A family tendency is common.

Prognosis.—Acute nephritis will terminate (1) usually in complete recovery in a few weeks, when treatment and hygienic surroundings are good (80 per cent. of cases). This is usual with children; with adults complete recovery is not so common, unless the original focus of infection rapidly subsides. (2) Partial recovery. If the disease lasts longer than two months, it develops into the condition known as large white kidney (Subacute Parenchymatous Nephritis, § 398). (3) Death may occur from uræmia, from dropsy into the serous cavities, or from other complications. The chief *complications* are: (*a*) Uræmia; (*b*) hypertensive encephalopathy; (*c*) inflammations of the *serous* membranes, such as pleurisy, pericarditis, or peritonitis, which are usually latent—*i.e.*, attended by little or no pain; and (*d*) infections of the *mucous* membranes, such as bronchitis, broncho-pneumonia, gastro-enteritis; (*e*) œdema of the lungs or of the glottis; (*f*) cardiac dilatation and left ventricular failure with pulmonary œdema; (*g*) erysipelas, cellulitis, and various other *skin diseases* are very prone to attack patients with acute nephritis. The prognosis, therefore, of acute nephritis is grave in proportion to (i.) the persistence of œdema, oliguria, gross albuminuria, and hypertension beyond two weeks; (ii.) the development of uræmic symptoms; and (iii.) the nature and severity of the complications.

Treatment.—The indications are to relieve the kidney by giving it as little to do as possible; to increase the action of the skin and bowels; and to lessen local congestion. (i.) The diet should consist at first of 1½–2 pints of fluid daily, with added glucose (see diet in § 297, VIII, Stage I) unless the blood urea is over 100 mgms. per cent., when more fluid is essential. After 3–4 days the hæmaturia has often diminished, the

blood pressure fallen, and the volume of urine increased ("critical diuresis"), when further quantities of fluid and more carbohydrate may be added (Stage II). About the 10th–14th day it is usually possible to give a still more liberal diet (Stage III). It is unwise to restrict the diet for too long a period as this undermines the patient's resistance. (ii.) To obviate the danger from exposure to cold, the patient must be kept strictly confined to bed till all red blood cells have disappeared from the urine. Cases of scarlet fever should be kept in bed during convalescence, because they are so apt to develop this disease. Diaphoretics may be needed, liquor ammoniæ acetatis, warm baths, wet packs, and hot-air baths. Mild purgatives are indicated; saline purgatives are especially useful when there is much dropsy. Diuretics are contraindicated in the early stage only. Alkalies such as sodium or potassium bicarbonate, citrate, acetate, and bitartrate may be given to prevent an acid urine still further damaging the inflamed renal epithelium. (iii.) Local depletion by wet or dry cupping is especially indicated when the volume of urine is low. Counter-irritation over the kidneys, with poultices, antiphlogistine, or leeches, has a similar effect. Digitalis can be given if the heart is feeble. The effect of penicillin injections is on trial. During *convalescence*, tonics, especially iron, must be given. In the treatment of renal disease two drugs are contraindicated—cantharides and turpentine. Mercury is generally added to these and mersalyl should certainly be used with caution. For the treatment of Uræmia, see § 372.

In scarlet fever albuminuria frequently comes on between the sixteenth and twenty-sixth day, at which time also acute nephritis may supervene. To avoid this risk, scarlet fever patients should be kept in bed a month, and the urine kept alkaline (Osman).

(*b*) **Acute focal glomerulo-tubular nephritis** arises during the height of the acute stage of an infection, again usually due to streptococcus hæmolyticus. Only a certain number of the glomeruli are involved; probably the micro-organisms produce minute emboli in them, and so signs of renal failure are usually absent.

Symptoms.—(1) The condition is commonest in children. (2) Hæmaturia and cylinduria are present; the bleeding may be profuse; the amount of albumen is such as can be accounted for by the bleeding, or little in excess of this. (3) Dull aching in the loins is common. (4) There is no renal failure; symptoms and signs of uræmia are absent, there is no œdema, no rise in the blood urea or blood-pressure. (5) Relapse may occur with recurrence of the original infection. The *prognosis* is almost invariably excellent; chronic nephritis ensues rarely. The *treatment* is as for the diffuse variety, except that there is no need for restriction of fluids, and not so much need to restrict the protein.

B. *The albumen is* PERSISTENT *and is* ASSOCIATED WITH CASTS ; BLOOD *is present microscopically and* ŒDEMA MAY BE PRESENT ; *the disease is* **Subacute parenchymatous nephritis.**

§ 398. When the symptoms and signs of acute nephritis do not subside within 6–8 weeks, the disease has entered the subacute phase. A large number of patients are first seen at this stage, occasionally because they have neglected to obtain advice, or more usually because there is no initial acute infection and the disease has been insidious from the commencement. There are two extremes of this clinical condition: (*a*) In

the usual variety œdema is the most prominent feature; (*b*) in rare cases œdema may be largely absent: intermediate cases are often seen. In either case, if the disease persists, it usually passes into the stage of secondary contracted kidney (§ 400).

(*a*) *The illness has been present for two or more months, and the general symptoms of renal disease pronounced;* GENERALISED DROPSY IS MARKED; *the* URINE IS SCANTY, *and* ALBUMEN *and* CASTS ARE ABUNDANT. *The disease is* SUBACUTE PARENCHYMATOUS NEPHRITIS (nephrotic type).

Subacute Parenchymatous Nephritis (synonyms: Large White Kidney, Subacute Glomerulo-tubular nephritis, formerly called Chronic Parenchymatous Nephritis) usually develops insidiously.

Symptoms.—(1) The albuminuria is considerable, $\frac{1}{4}$ to $\frac{1}{2}$ of the volume of the urine: the daily loss may be 20–30 G. (2) The other characters of the urine are: (i.) the diurnal quantity is diminished, (ii.) the specific gravity tends to be high, (iii.) it is often turbid with urates, (iv.) all forms of casts are met with (§ 392), (v.) blood is rarely absent but is usually only detected microscopically: relapses temporarily increase the amount of blood. (3) Generalised dropsy is a marked feature. At first it is most marked in the face, giving a general puffiness: soon it appears in all the loose cellular tissues of the body, and the serous cavities become involved, causing a general anasarca. The amount of œdema varies at different times, so that the patient may lose or gain many pounds in weight in the course of a few weeks. (4) A marked degree of anæmia is present sooner or later, the hæmoglobin falling to $\frac{1}{2}$–$\frac{1}{3}$ the usual values. (5) Lassitude and digestive disorders are common. (6) The blood pressure and the blood urea are very little raised. (7) The blood proteins are markedly lowered (below 5 per cent.), but the blood cholesterol is high. (8) After many months the œdema may subside. It is rare for the condition to be cured: more often the blood pressure and the blood urea are found to increase simultaneously with the disappearance of the œdema, renal function tests show progressive impairment of function, and the condition becomes chronic (secondary contracted kidney).



FIG. 106.—A case of Subacute Nephritis with Anasarca.

(*b*) *The illness is subacute, the urine containing* ALBUMEN, TUBE CASTS *and* RED CELLS: *the patient is* PALE, *shows occasional* PUFFINESS OF THE FACE, *and renal function tests show* PROGRESSIVE IMPAIRMENT OF RENAL FUNCTION. *The disease is* SUBACUTE NEPHRITIS WITHOUT MARKED ŒDEMA.

This variety is more commonly overlooked than the nephrotic variety, on account of the slight degree of œdema. The *symptoms* are: (1) The urine invariably contains a small quantity of albumen, and hyaline and granular casts. Microscopically, red cells are almost invariably present, indicating that the inflammatory process is still active. (2) Periodically, slight œdema may appear, especially in the face. (3) Symptoms of general debility, lassitude, anæmia, and headaches are present. (4) The blood pressure is raised, the systolic pressure being commonly 20–50 mm. above normal. (5) The blood urea is raised and kidney efficiency tests give a poor result. (6) The nephrotic type may supervene later.

Etiology of Subacute Nephritis.—The cause is usually not known. However carefully a septic focus is sought, according to Ellis it is rarely found. The insidious onset over a period of weeks or months makes the search more difficult. The average age of onset is later than in the acute varieties. Occasionally tertiary syphilis is causal.

Diagnosis of Subacute Nephritis.—When the insidious form occurs in young women it is often mistaken for *simple anæmia;* in all such cases, examine the urine for albumen and tube casts. In the later stages it may be mistaken for *chronic interstitial nephritis*; but in that disease the patient is usually older, and see Table XXII (p. 490). In certain cases which present *both renal and cardiac* symptoms, it may be very difficult to say *which condition is the primary* one.[1] In such cases it is important to note the following points: (i.) If there is a *history* of rheumatic fever and previous attacks of dropsy, it is probable that the cardiac condition is primary. (ii.) The presence of *other than mitral* systolic murmurs points to cardiac disease; a mitral regurgitation murmur *alone* might be due to the cardiac failure following renal disease. (iii.) The *urine*, when there is any difficulty in diagnosis, is in both cases scanty and albuminous. Many tube-casts, and an appreciably raised blood urea, point to renal disease; the rapid clearing up of the dropsy and improvement of the urine after a short period of rest in bed points to heart disease. (iv.) A *hard pulse* favours kidney disease, but an irregular soft pulse is found with cardiac failure secondary both to renal and to cardiac disease.

Prognosis.—It is rare for the disease to be arrested, but the prognosis is better in the non-œdematous variety. It is not uncommon for acute relapses to occur. A useful guide to the prognosis is furnished by successive renal function tests every six months. Death occurs with complications of uræmia, or as with acute nephritis. The prognosis is grave in proportion to (1) the amount of dropsy and albuminuria; (2) diminution of urine and of nitrogen excretion; (3) the height of the blood pressure. When this rises progressively the outlook is grave, whereas if the pressure remains the same or falls, the prognosis improves correspondingly. (4) Uræmic symptoms.

[1] It is well to bear in mind that when both cardiac and renal disease are present, they may be associated in three ways: (*a*) Cardiac disease may produce renal disease (§405. IX). (*b*) Renal disease may produce cardiac disease, as when acute or chronic nephritis lead to cardiac hypertrophy and failure. (*c*) They may both be the result of a common cause—*e.g.*, gout.

If a source of infection is found and removed, the outlook improves. When the stage of contraction sets in, life may be prolonged with care.

Treatment.—So long as subacute inflammation is present, the patient should be confined to bed, and this is essential when œdema is present. The main objects are (i.) to reduce or abolish œdema, (ii.) to remove septic foci, (iii.) to relieve the kidneys as far as possible. (i.) *Diet.* When œdema is present, the kidneys do not secrete sodium chloride and water, the urinary volume remains at a low figure, but on the other hand an enormous amount of albumen (often 10–25 grammes daily) is lost in the urine. The rational line of therapy is to limit the amount of fluid ingested to that which the kidneys can secrete per day, and salt should be avoided as far as possible: at the same time the protein intake should be of a high order, and this is of additional value for the urea so produced is a valuable diuretic (Maclean). A diet meeting these needs is given (§ 297. VIII. B.). If benefit is to be obtained with such a diet it should show itself in 6–8 weeks, and it is useless to continue it otherwise. In this case an ordinary diet can be resumed, with some restriction of total fluid and salt intake. It is unwise to use a high protein diet if the blood urea is already raised, and in any case weekly blood urea estimations are desirable: in these circumstances the Karell diet is often used (§ 297. VIII. B.). When there is little or no œdema, these strict diets are unnecessary and may be harmful by lowering the patient's resistance. The Stage III diet of acute nephritis is then useful. *Diuretics* may aid the elimination of fluid. Maclean found a remarkable diuretic effect can be obtained with 15 G. of urea dissolved in water, 2–3 times daily. Thyroid gr. 3–5 a day helps to reduce œdema. Another method is to give citrate and bicarbonate of potassium in the ratio 1 : 2 ; these act by raising the alkali reserve of the blood, antagonising the sodium salts in the tissues and acting as diuretics.

So long as the urea elimination tests give good results, these salts should be given in frequent small doses sufficient to make the urine strongly alkaline to litmus and to give a faint pink tint to 4–5 drops of phenolphthalein (*i.e.*, to pH 8·2). Often 200–300 grains a day are necessary and after a week a profuse diuresis frequently sets in and eliminates the anasarca.

Intramuscular injections of mersalyl are now used more freely than was once thought possible: the toxic effects on the renal tubules are usually very transitory, but need careful watching. This drug must never be given intravenously in nephritis, for in cases with a low plasma albumen sudden myocardial failure may occur.

Effusions in the serous cavities may have to be tapped if they are producing pressure symptoms, but scrupulous care must be taken to avoid infection. *Decapsulation* of the kidneys has been performed in resistant cases. (ii.) Septic foci must be searched for and carefully removed. The author has had encouraging results from penicillin therapy, even when a definite focus of infection cannot be found: but the penicillin must be given within 2 weeks of the onset of clinical symptoms. (iii.) To prevent further renal damage, chill and exposure must be avoided, the

bowels regulated, and all alcohol forbidden. (iv.) Although concentrated plasma transfusions are usually disappointing in that the transfused protein is rapidly eliminated, when anæmia is severe a blood transfusion is often helpful.

Nephrosis is a term used to describe a special type of parenchymatous nephritis. The essential lesion is a lipoid degeneration of the kidney tubules, without any signs of inflammation. Thus, in addition to marked œdema and albuminuria, there is hypercholesteræmia and slight myxœdema; but there are no cardio-vascular changes nor is there any nitrogen retention. At autopsy, the masses of lipoid in the tubules form the so-called "myelin" deposits.

C. *The* ALBUMEN *is* ASSOCIATED WITH CASTS. BLOOD *is usually* ABSENT *and the condition is* CHRONIC. *The disease is* CHRONIC NEPHRITIS.

§ **399.** There are three anatomical varieties of **chronic renal disease** attended with more or less albuminuria, which, when occurring in their typical forms, present well-marked clinical distinctions, as shown in tabular form on p. 490. In the condition of SECONDARY CONTRACTED KIDNEY, we are dealing with the end result of progressive acute and subacute nephritis with gradual destruction of the kidneys and replacement by fibrous tissue. In CHRONIC INTERSTITIAL NEPHRITIS there is no such history of previous acute or subacute nephritis; the onset is usually insidious, hypertension and arterial disease are often marked, and pathologically there is found considerable increase in the interstitial tissue of the kidneys and hyperplasia of the middle coats of the arteries. In the AMYLOID (Waxy) Kidney the vessels are primarily involved, the lardaceous degeneration beginning in the middle coat. Pathologists make many sub-divisions, but these represent the three clinically recognisable groups of chronic renal changes attended by albuminuria.

Following an attack of ACUTE *or* SUBACUTE NEPHRITIS *the patient complains of the symptoms of* INCIPIENT URÆMIA. *There is a small quantity of* ALBUMEN, POLYURIA *is present,* DROPSY *is slight or absent. The condition is one of* SECONDARY CONTRACTED KIDNEY.

§ **400. Secondary Contracted Kidney** (Synonym: Chronic Diffuse Nephritis). *Symptoms:* (1) Urinary changes: (i.) diminution of the large amount of albumen which was present in the early stage; (ii.) the volume of the urine rises; (iii.) the specific gravity falls, and the urea content is considerably reduced. (2) The dropsy disappears as the diurnal quantity of the urine increases. (3) The blood urea and the blood pressure rise progressively, and the left ventricle hypertrophies. (4) In the terminal stage of renal failure the urinary volume falls and uræmic symptoms ensue. In children a condition of renal rickets may accompany this form of nephritis. The treatment is as for chronic interstitial nephritis.

§ **401. Chronic Interstitial Nephritis** used to be regarded as a single clinical entity, but now different varieties are becoming recognised. It is convenient to reserve this term for a composite group of cases distinguished by *persistent albuminuria* and *cylindruria*, often with hypertension,

in which the progressive renal destruction is not due to a previous attack of acute or subacute nephritis. In one variety the primary cause is arterial disease associated with hypertension, benign or malignant, and recent experimental and clinical evidence regards the chronic renal destruction as being due to the diminution of blood supply to the kidneys. In another variety the kidneys are primarily at fault, due to congenital or acquired lesions: here the renal destruction often causes the liberation of a pressor substance which produces hypertension, the resultant arterial disease causing a "vicious circle." The clinical symptoms produced in these different types are influenced in part by the age of the patient, as when chronic nephritis causes renal rickets and renal dwarfism. A classification which includes most of these forms of chronic interstitial nephritis is:—

Type 1. Chronic Nephritis develops in a *middle-aged* or *elderly patient* suffering from *hypertension* (hypertensive nephritis).
(*a*) Benign hypertension.
(*b*) Malignant hypertension (Nephrosclerosis).

Type 2. Chronic Nephritis occurs in a *young person*, previously in good health, and *may* or *may not* be associated with *hypertension*. It is probably due to some congenital abnormality in the kidneys (renal dysbiotrophy).
(*a*) Chronic type.
(*b*) Rose Bradford type with acute symptoms.
or Chronic Nephritis occurs in a *young or middle-aged person* as a result of a *congenital cystic defect* (Congenital Cystic Kidneys). § 424. V.

Type 3. Chronic Nephritis occurs as a result of renal destruction by other causes, *e.g.*, renal calculus, hydronephrosis, chronic pyelonephritis.

1 (*a*). *The patient is* MIDDLE-AGED OR ELDERLY *and has suffered from* BENIGN HYPERTENSION *for years. The* DIURNAL QUANTITY OF URINE *increases, and* ALBUMINURIA *and* CASTS *appear: later signs of* INCIPIENT URÆMIA *develop. The disease is* CHRONIC NEPHRITIS *with* BENIGN HYPERTENSION.

Benign hypertension is a condition of hypertension which for long periods is non-progressive, or very slowly progressive, and associated in the majority of cases with no renal changes and no albuminuria (§ 94). In a small number of cases, albuminuria and casts appear later, due probably to renal arterio-sclerosis.

Symptoms.—(1) The symptoms of benign hypertension are fully described in § 94. When impairment of kidney function follows (i.) the albuminuria is small in amount, and many samples of urine may be examined without finding any. In cold weather, however, when there is deficient skin action, there is generally a trace, especially after a chill or any cause which produces renal congestion. The other characters

of the urine are: (ii.) The diurnal quantity is increased (perhaps to 100 ounces). The patient has to get up at night several times to pass large quantities of water. (iii.) The specific gravity is low (1002 to 1010), owing chiefly to the increased quantity of urine. (iv.) The urine is clear, pale, and contains but few casts, and these are chiefly hyaline or granular (Fig. 92). (2) Dropsy is usually absent. If dropsy occurs it is due to secondary cardiac failure. (3) The patient may look robust, but sometimes he has a greyish pallor. (4) The pulse indicates persistent high blood pressure, associated with hypertrophy of the left ventricle, and with a thickened condition of all the arteries. (5) There develops a condition of chronic or incipient uræmia (§ 371), due to the deficient elimination of nitrogenous and other substances.

Diagnosis.—Often the patient has been known to have hypertension for many years. Although degenerative retinal changes associated with retinal arterio-sclerosis are present, *papillœdema never occurs.*

Prognosis.—The disease is very slowly progressive, even when renal damage is present. The older the patient the better the outlook, and he is more likely to die of heart failure or apoplexy than of uræmia, even when renal disease is well established. With proper care and treatment, the patient may live for five or ten years. The amount of albumen is no criterion as regards prognosis in chronic interstitial, as it is in parenchymatous, nephritis.

1 (*b*). *The patient has suffered from* HEADACHE *and other symptoms of* HYPERTENSION *for months or years.* VOMITING, PRECORDIAL PAIN *and* ALBUMINURIA *develop, with* PAPILLŒDEMA. *The disease is* CHRONIC NEPHRITIS *with* MALIGNANT HYPERTENSION (MALIGNANT NEPHRO-SCLEROSIS).

§ **402. Malignant Nephrosclerosis** (Synonym: Chronic Focal Glomerular Nephritis) is accompanied by widespread cardio-vascular changes. There is no history of antecedent acute or subacute nephritis; often the patient has been in normal health and not known to have hypertension before symptoms arise. In other cases there has been benign hypertension for years, the condition developing into malignant hypertension in the course of a few months (see § 94).

Symptoms.—(i.) Headache, especially on waking, with vomiting, is associated with attacks of precordial pain, breathlessness, and nocturnal paroxysmal dyspnœa. An epileptiform convulsion may be the first symptom. (ii.) The blood pressure is very high, with figures of 240 mms. (systolic) and 130 mms. (diastolic) or more. A figure of 290/180 is not unusual in association with hypertensive cerebral attacks (§ 94). (iii.) The left ventricle is hypertrophied, the brachial and radial arteries contracted and hardened, and in the late stages stress and failure of the left ventricle are shown by attacks of œdema of the lungs, by tachycardia, premature beats, pulsus alternans and gallop rhythm. (iv.) Failing vision is due to changes in the fundus oculi: the fundi show papillœdema, contracted silver-wired arteries and patches of retinal degeneration, especially at the

maculæ. Whereas in the early stages of malignant hypertension there is little evidence of renal damage, later this becomes a prominent feature; when this occurs, (v.) the specific gravity of the urine becomes more and more fixed around 1012, albuminuria is marked, and the urinary deposit contains some red and white blood cells, and a number of hyaline and granular casts; (vi.) the blood urea may be normal when the patient is first seen, but later rises to 300 mgms. per cent. or more in spite of treatment; (vii.) symptoms of incipient uræmia become more marked, with loss of appetite and considerable loss of weight, thirst, impairment of mental and physical vigour, and tremor and twitchings of the muscles; hiccup is often very troublesome. The *Diagnosis* depends on the very high blood pressure, the retinal changes (and especially the papillœdema), the moderate or considerable albuminuria and the absence of a previous history of nephritis.

Etiology.—(i.) The disease may occur at any age, but most often between 30 and 50 years of age. (ii.) A family history of hypertension is very common.

Structural Pathology. (See § 94.) The kidneys are a little smaller than normal. There is fibrinoid necrosis especially of the afferent glomerular arteries, with acute focal degeneration of the glomeruli, and severe degenerative changes in the tubules.

Prognosis.—The course of the disease is relatively rapid once the kidneys are involved; patients rarely live more than two years after diagnosis. The greater the degree of hypertension, of albuminuria and especially of papillœdema when first seen, the worse is the prognosis.

2 (*a*). *The patient, who is* UNDER 30 YEARS OF AGE, *is found to have* ALBUMINURIA. *There is no previous history of acute or subacute nephritis.* HYPERTENSION *may or may not be present. The course of the disease is slow, but* URÆMIA *supervenes after a course of years. The disease is* CHRONIC NEPHRITIS *probably due to a* CONGENITAL DEFECT *in the kidneys.*

This little understood group has recently been separated from the heterogeneous group of chronic interstitial nephritis, and merits a separate description.

Symptoms.—(i.) The patient, who is usually 15–20 years of age, is found on routine examination to have chronic albuminuria. The amount of albumen is never large. The urine contains some red cells, and occasional granular and hyaline casts. (ii.) For months or years the patient may otherwise appear to be in perfect health, but sooner or later lassitude, attacks of pallor, and occasional headaches make their appearance. (iii.) From the time the patient is first seen there is often polyuria, the renal function tests give a poor result, with deficient urea concentration and urea clearance, and often a blood urea raised above the normal. (iv.) In some the blood pressure is raised, but it may be normal throughout. (v.) In cases before puberty, signs of renal infantilism or of renal rickets (§ 596) may be present.

Diagnosis.—In the earlier stages it may be difficult to distinguish

postural or orthostatic albuminuria, especially as the amount of albumen in both cases is reduced by rest in bed. Impaired renal function tests prove the kidneys to be diseased.

Etiology.—These cases are believed to be due to an inborn tendency to renal degeneration (dysbiotrophy); rarely they are familial, and at autopsy it is not unusual to find such congenital abnormalities as double ureters.

Prognosis.—The course is very slowly progressive, and it is common to find the patient living 5 to 10 years after the disease is discovered. It is often surprising how long a young person will live with a blood urea persistently raised at a level of 100 mgms. per cent. or more. The rapidity of the renal failure is best judged by periodic renal function tests.

2 (*b*). *The patient is commonly* 15–26 YEARS OF AGE, *and has not previously suffered from acute or subacute nephritis. He may be* SUDDENLY TAKEN ILL *with* URÆMIA, *and is found to have a* VERY HIGH BLOOD PRESSURE. *The disease is probably* CHRONIC NEPHRITIS (*Rose Bradford type*) *with* SMALL WHITE KIDNEYS.

The cause is unknown, but it does not follow acute or subacute nephritis. Often the first symptom is an attack of uræmia in an apparently healthy person. There is no œdema, the blood pressure is very high, the respiration assumes a hissing quality, and the urine is normal in quantity, of low specific gravity, with a few casts and more albumen than is met in the cases with red granular kidney.

§ **403.** 3. *A patient with* HYPERTENSION *and* ALBUMINURIA *also has symptoms of chronic* PYELONEPHRITIS, *a* RENAL CALCULUS *or* OTHER DISEASE OF THE KIDNEYS. *The disease may be* CHRONIC NEPHRITIS *associated with a* SURGICAL KIDNEY LESION.

As already discussed in §§ 87, 94, any lesion of a kidney which produces chronic renal destruction may cause the liberation of a pressor substance which produces hypertension and subsequent chronic nephritis in the sound kidney. Such lesions are chronic atrophic pyelonephritis, renal tuberculosis, renal calculus, hydronephrosis, or a renal tumour. When unilateral lesions are present, the progressive hypertension and the chronic nephritic lesions in the sound kidney may be controlled by surgical removal of the diseased kidney, and in certain cases the blood pressure may return to normal. In future it will therefore be necessary to investigate for these several conditions (*q.v.*) as part of the routine investigation of cases of hypertension and chronic nephritis. The best results are obtained by removing a unilaterally diseased kidney affected by chronic atrophic pyelonephritis.

Treatment of Chronic Nephritis.—The first aim is to discover the cause whenever possible. Septic foci should be eliminated, and drugs which damage the kidneys (such as phenol, cantharides, mercurial salts) avoided. When unilateral renal disease is present, such as chronic pyelonephritis or a calculus, considerations of nephrectomy arise. General measures include the avoidance of chill or of excessive mental or physical exertion, the administration of a suitable dietary (§ 297. VIII C.) when the blood urea is raised, and exercising extreme moderation with alcohol. Pregnancy is not permissible, as it increases the renal damage, and abortions

or a macerated fœtus usually result. Otherwise the treatment is that of the main complications: (i.) Heart failure will need rest in bed, the relief of hypertension, and of insomnia (§ 62). (ii.) Hypertension may be relieved by mannitol nitrate, gr. ½ or sodium nitrite, gr. 1, together with small regular doses of calomel; (and see § 94): malignant hypertension may require surgical treatment. (iii.) Anæmia does not necessarily respond to iron salts, and these may be injurious by leading to constipation: in certain cases transfusion of concentrated red cells may stimulate a hypoplasic bone marrow. (iv.) Treatment often resolves itself into the treatment of uræmia. In *chronic* uræmia keep the diet low, give enemas or high colon douches, and encourage the action of the skin. The treatment of *acute* uræmia—muttering delirium, convulsions, coma—is fully described in § 372.

There is **abundant albumen** *with the passage of* LARGE QUANTITIES *of urine, but little tendency to dropsy and uræmia; the patient is anæmic; there is a history of prolonged* SUPPURATION, *or of* SYPHILIS; *and there may be evidences of lardaceous disease elsewhere.* The disease is AMYLOID KIDNEY.

§ **404. Amyloid Kidney** (Waxy or Lardaceous Kidney) is generally part of a widespread lardaceous disease. With more efficient modern surgical methods, amyloid degeneration is becoming a very rare condition.

Symptoms.—(1) The albumen, though it may be small in quantity in the early stage, is marked when the condition is established. (i.) The diurnal quantity is greatly increased, even to 150 ounces; (ii.) the specific gravity is very low, but the urea is not diminished till the later stages; (iii.) the colour is pale and clear; (iv.) all varieties of casts may be found, including amyloid and fatty casts. (2) There

TABLE XXII.—CHRONIC ALBUMINURIA OF RENAL ORIGIN.

	Quantity of Albumen.	*Tendency to Uræmia.*	*Quantity of Urine.*	*Tendency to Dropsy.*
Subacute Parenchymatous Nephritis.	Large.	Moderate.	Diminished or normal.	Great.
Secondary Contracted Kidney.	Moderate.	Great.	Increased.	Slight.
Chronic Interstitial Nephritis.	Varies with the cause.	Great.	Increased.	Very slight.
Amyloid Kidney.	Very great.	Slight.	Greatly increased.	Slight.

is great pallor of the surface and anæmia, but there may be no dropsy, till near the end. In cases with great cachexia dropsy *may* occur early (§ 29). (3) Other evidence of lardaceous disease is present—enlargement of the liver and spleen; consequently hæmorrhages may occur from different parts. Amyloid disease of the bowel causes intractable diarrhœa, and when it attacks the suprarenals the blood pressure is low.

It is important for the *diagnosis* to ascertain the history of a *cause*—namely, (*a*) prolonged suppuration, either from a chronic abscess, chronic phthisis, or caries. (*b*) Syphilis is an important cause.

Prognosis.—The patient may live long, dying from exhaustion from diarrhœa,

or other complications; rarely from uræmia due to supervention of acute nephritis. With careful treatment patients may live for many years, or even recover if the disease is seen early; but the prognosis is bad in proportion to (1) the amount of albuminuria, and (2) the extent of the involvement of other organs. The prognosis is good if the cause is removed.

Treatment.—Alkalies have been reputed not only to prevent, but also to improve, the lardaceous process—*e.g.*, the tartrates and citrates of the alkalies. Iodide of potassium or of iron should be given, particularly in syphilitic cases. The most troublesome complication is diarrhœa. The only remedies of any use are liquor ferri pernitratis, ℳ 15; or pil. plumbi cum opio, gr. 5, continued every four hours until the diarrhœa ceases. The *preventive treatment* consists in the adequate treatment of syphilis in its early stages, and in curing prolonged suppuration.

D. *The* ALBUMEN *is* NOT *usually* ASSOCIATED *with* CASTS *or* BLOOD; *the condition is often* INTERMITTENT *and tends to be chronic.*

§ **405.** The chief causes to be reviewed are :

I. PHYSIOLOGICAL.—Albumen occurs regularly in the urine of new-born infants in the first week of life, apparently due to the kidney not having gained its normal semi-permeability. Albumen is also present for some hours after any severe exercise, such as running or rowing. Probably the albuminuria is due to a temporary vaso-constriction of the renal vessels during exercise.

II. ORTHOSTATIC or FUNCTIONAL albuminuria occurs in the adolescent, as a result of the upright position. It therefore disappears at night, when asleep and in the horizontal position; occasionally it continues to be excreted for the first hour after lying down. It is commonest (i.) in young people; (ii.) it shows a familial tendency; (iii.) it occurs in tall people, during the periods of most active growth; it is associated (iv.) with a tendency to attacks of pallor, faintness and fatigue states; and (v.) with a low systolic blood pressure and often a low pulse pressure. *Diagnosis:* casts and other evidences of renal failure are absent, renal efficiency tests give normal findings and, if the patient voids urine an hour after going to bed, the morning specimen is free of albumen. The albuminuria often disappears with full doses of calcium salts. Mild cases of nephritis also may show albuminuria when the patient stands upright for long, but other evidences of renal disease are present.

III. KYPHOTIC albuminuria is due to a kyphotic stance causing pressure on the left renal vein. It disappears on correcting the cause.

IV. Any form of TOXÆMIA may produce albuminuria. The milder cases probably recover completely, with no permanent renal damage; more severe cases tend to progress to acute or chronic nephritis. In hyperpyrexia albuminuria is invariably present. A small quantity of albumen is common in febrile conditions, *e.g.*, in pneumonia, typhoid, diphtheria, diarrhœa and vomiting of infants, the reaction stage of cholera, secondary syphilis, tuberculosis, streptococcal infections and in any septicæmia. The albumen may be accompanied by some casts in the severe forms, but it disappears completely within 2 or 3 weeks of the subsidence of the fever.

V. PREGNANCY.—The cause of the albuminuria is almost certainly a toxæmia, although mechanical pressure may play a part. The condition is more common in primiparæ. *Mild cases* occur after the sixth month; there is albuminuria (up to 0·2 per cent.), and an excess of renal epithelium and occasionally of casts. The blood pressure is slightly raised (systolic up to 150 mm.), but there are no signs of renal failure, such as œdema, retinitis, or urea retention. The condition rapidly disappears in the puerperium, but may recur in subsequent pregnancies. *Severe cases* may begin as mild cases, or they may start suddenly, often with eclampsia. The blood pressure is considerable (up to 230–240 mm.), and there is usually evidence of renal failure with œdema, oliguria, retinitis, headaches, drowsiness, muscular twitchings, etc. The condition may respond to medical treatment or may need artificial termination of pregnancy. It often passes into chronic nephritis, and recurs in subsequent pregnancies.

VI. DRUGS and POISONS are closely allied to the previous group. Common causes are mercury, arsenic, phosphorus, phenol, cubebs, copaiba, salicylic acid, quinine, lead, cantharides, turpentine, alcoholism, and vegetable or animal poisons such as mushroom or fish poisoning. EXCESSIVE PROTEIN INTAKE may act in a similar manner. This cause is recognised by: (i.) the presence of the drug in the urine; (ii.) there may be a history of the administration of the drug; and (iii.) the albuminuria usually disappears when the drug is stopped.

VII. ENDOGENOUS POISONS such as jaundice, diabetes, and acute gout may all cause temporary albuminuria.

VIII. CHILL TO THE SURFACE.—This is recognised by: (i.) The amount of albuminuria is never great, and it does not last for more than a few days; (ii.) the urine is otherwise normal, or may deposit urates; (iii.) the patient is healthy, or complains only of slight bronchial catarrh or coryza.

IX. Mild RENAL CONGESTION causes albuminuria in: (i.) right-sided heart failure; (ii.) rapid catheterisation of a distended bladder (and see Hæmaturia, § 406); (iii.) after epileptic fits.

X. Chronic GOUT and ARTERIO-SCLEROSIS lead to an ischæmia of focal areas in the kidneys. The urine is copious, of low specific gravity and contains at times a trace of albumen. The blood pressure is raised, but there is no tendency to uræmia (see § 93).

XI. URINARY CALCULI and CRYSTALS, especially oxalates, may give rise to albuminuria, but other signs are usually present (§ 408).

XII. "LEAKY KIDNEY" or "RESIDUAL ALBUMINURIA" is a name given to a condition of albuminuria which follows a past attack of nephritis, and is due to albumin leaking through the healed scars. It is known by: (i.) the absence of signs of renal failure, and the renal function tests are normal; (ii.) absence of casts; (iii.) normal blood pressure; (iv.) the condition is not associated with progressive renal disease.

XIII. ANÆMIA.—Severe anæmia may be accompanied by a trace of albumen.

XIV. OBSCURE CAUSES, *e.g.*, when albumen appears for unknown reasons, as (1) after burns and other causes of severe shock. (2) In exophthalmic goitre the albuminuria is usually temporary, though it may last for months. It may vary in amount at different times on the same day, which tends to show that it is of vaso-motor origin. The urine in other respects is healthy. (3) Excessive study or other cause of nerve strain has been reported to have occasioned albuminuria. (4) Certain cases of cerebral tumour, and other conditions in which there is increased intracranial pressure, have been attended by albuminuria.

The *Prognosis* of albuminuria in the above groups is that of its cause. Before

giving a prognosis it is important to examine thoroughly and repeatedly the urine, for casts in particular, so as to be satisfied that the kidneys are structurally healthy. Young subjects with functional albuminuria are not necessarily predisposed to kidney troubles, but they are often under par; the albuminuria will disappear as the general condition improves. The prognosis as to life is excellent.

Treatment.—The treatment must be directed to the cause. Rest in bed will do a good deal for the renal complication of cardiac disease. In the *albuminuria of pregnancy* careful investigations should be made, and the amount of urea watched. If (1) there is a clear history of renal disease prior to pregnancy, or (2) puerperal eclampsia has occurred in previous pregnancies, or (3) the renal disease, no matter of what kind it may be, is distinctly *progressive in its nature*, then termination of pregnancy or induction of premature labour should be advised. For the treatment of functional albuminuria general hygienic and dietetic rules must be followed. The administration of calcium lactate and alkalies temporarily stops the albuminuria.

E. *The* ALBUMEN *is* ASSOCIATED WITH BLOOD, *and* CASTS *are scanty or absent*—HÆMATURIA. When the condition is associated with severe abdominal pain, see renal colic, § 408.

§ **406. Hæmaturia.**—When the patient is "passing blood" in the urine, an endeavour should be made to ascertain if the blood comes chiefly at the beginning of micturition, chiefly at the end, or whether it is intimately mixed with the urine and gives to it a "smoky" tint. For the test for blood in the urine and the methods of distinguishing it from hæmoglobinuria, see § 382. The fallacy of menstrual blood must be avoided by using a catheter.

1. *If the blood is bright crimson and comes chiefly* AT THE COMMENCEMENT *of micturition, it is probably of* URETHRAL *or* PROSTATIC *origin.*

In these circumstances, which are mainly of surgical interest, there will probably be a history of injury or gonorrhœa. In congestion or abscess of the prostate there are local pains or tenderness and rectal irritation. Urethral angioma and excessive sexual indulgence in the male and urethral caruncle in the female may lead to hæmaturia.

2. *If the blood comes most freely* AT THE END *of micturition, and especially if in clots, it is probably of* VESICAL *origin.*

The COMMONEST CAUSES of vesical hæmorrhage are:

I. ACUTE CYSTITIS, chiefly at its onset (see § 411). The bleeding is usually slight.

II. CALCULUS, or stone, in the bladder. Here the hæmorrhage is worse after exercise, moderate in amount, and there is pain, which, like the bleeding, is worse at the end of micturition and after exercise or jolting, and is frequently referred to the point of the penis. The ensuing cystitis may complicate the symptoms and render the diagnosis of stone difficult, but its detection by X-ray, the sound or cystoscope is conclusive.

III. TUMOURS of the bladder.—The hæmorrhage here, especially in *papillomata*, is usually great in amount. Shreds of the growth may be passed, and cystitis may develop. In *cancerous* tumours the hæmorrhage is more or less intermittent and resists treatment; there are pain and cachexia, and sometimes the growth may be palpable above the pubes or per rectum. Extension of tumours of neighbouring organs, or even spread of inflammation or congestion, as in appendicitis or dysenteric ulcers, may cause hæmaturia. The cystoscope is the best means we have of recognising the condition of the bladder.

IV. An enlarged PROSTATE may produce hæmaturia either from congestion or from the rupture of enlarged veins near the neck of the bladder.

V. Some of the LESS COMMON CAUSES of vesical hæmaturia are TUBERCULOUS DISEASE of the bladder, VESICAL VARIX, certain constitutional diseases such as SCURVY and PURPURA, and SCHISTOSOMA HÆMATOBIUM.

VI. § **407. Schistosomiasis** (Syn.: *Bilharziasis*).—The endemic hæmaturia of Egypt and South Africa results from the depositions of schistosomal eggs in the bladder by the adult female worm—*Schistosoma hæmatobium*—which lives in the portal system (§ 308, (3)) and pelvic plexuses of veins. Ova occur chiefly in the liver, bladder, lungs, prostate, lower third of the ureter, and in the pelvic viscera of the female; occasionally they are deposited in the pancreas, spleen and colon. Carcinoma of the bladder or penis may occur.



FIG. 107. — EGG of *Schistosoma hæmatobium*, magnified about 120.

Symptoms: Urticarial eruptions, terminal hæmaturia, frequency, perineal, penile and suprapubic pain and aching in the lumbar region. The blood is bright red, occurs at the end of micturition, and is increased by exercise. Cystoscopic examination in the early stages shows round, yellowish-white, pseudotubercles and ulcers; later, papillomata and sandy patches may develop. Schistosomal complement fixation reactions and intradermal tests are generally positive, and eosinophilia may be present, especially in the early stages. In the majority of cases terminal spined eggs are readily demonstrable in the urine. As the disease progresses the clinical picture may be modified by complications. Ureteric involvement may lead to back pressure on the kidney with hydronephrosis and chronic nephritis. Secondary bacterial infection of the genito-urinary tract is common, leading to septic cystitis, pyelonephritis and pyonephrosis; urethral fistulæ and vesical calculi may occur.

Etiology.—After the eggs in the excreta come in contact with water, motile miracidia are set free, enter certain fresh-water snails of the *Bulinus* species and develop in the liver into sporocysts and later cercariæ; these in turn escape into water, penetrate the skin of man during bathing, or invade the mucous membrane of the mouth and the œsophagus during drinking.

Treatment.—Antimony is specific, and the treatment of uncomplicated cases is highly satisfactory. The details of treatment are the same as in schistosomal dysentery (see § 308, (3) (*a*)). The presence of hepatic cirrhosis, splenomegaly, septic cystitis and renal involvement render effective treatment difficult; surgery may be required for complications.

3. *If the blood is* INTIMATELY MIXED *with the urine, causing it to assume a "smoky" tint, it is probably of* RENAL *origin. In these cases also the tests for blood should be carefully applied, and fallacies avoided* (§ 382).

Symptoms and signs pointing to the kidney will usually be detected on examination.

The CAUSES of RENAL HÆMORRHAGE may for convenience be grouped under these headings: (I) Inflammation; (II) Severe congestion; (III) Nephroptosis; (IV) Blood conditions; (V) Renal calculus and crystals; (VI) Drugs; (VII) New growths; (VIII) Essential hæmaturia; (IX) Paroxysmal hæmoglobinuria; (X) Injury.

I. INFLAMMATION: (i.) In acute nephritis the urine also contains casts (§ 397); (ii.) in subacute and chronic nephritis bleeding may occur during an acute exacerbation, or as a consequence of high blood pressure, in the same way that bleeding may occur from the nose (epistaxis) or into the brain; (iii.) Infarction, due to subacute bacterial endocarditis (§ 50);

(iv.) Tuberculous disease of the kidney (§ 412); (v.) Acute pyelitis (§ 412); (vi.) Parasites, *e.g.*, *Schistosoma hæmatobia* (see above). The *Microfilaria sanguinis hominis* usually causes chyluria, but hæmaturia may also occur.

II. SEVERE CONGESTION.—Mild congestion causes albuminuria, but marked congestion causes hæmaturia also. (i.) The commonest cause is right-sided heart failure (§ 55). The scanty, highly-coloured urine contains at first albumen only, but later a large quantity of albumen, accompanied by blood. Particularly in long-standing cases, there is an excess of hyaline casts. (ii.) Sudden congestion occurs after rapid catheterisation of a distended bladder, probably as a result of sudden relief of a bilateral hydronephrosis. Suppression of the urine may also ensue. It can be avoided by emptying the bladder gradually. (iii.) Thrombosis of the renal vein causes acute congestion. It occurs chiefly with streptococcal infections, and rarely in cachectic states—sudden hæmaturia with rapid enlargement of a tender kidney is very suggestive. (iv.) Sudden congestion may occur with a patient who has been bedridden for many months, as with a fractured femur (Wilson), usually on the second day of walking. There is colic, hæmaturia, and pain in the loin, which disappear when the patient returns to bed for a few days.

III. NEPHROPTOSIS, with or without aberrant renal vessels, may produce intermittent attacks of hæmaturia, probably congestive in origin.

IV. BLOOD CONDITIONS, especially scurvy, purpura and malaria.

V. Renal CALCULI and CRYSTALS, particularly oxalates, produce renal colic and hæmaturia (§ 408).

VI. DRUGS, particularly salicylates, phenol and its derivatives, sulphapyridine and sulphathiazole, hexamine, cantharides and turpentine can cause hæmaturia.

VII. NEW GROWTHS of the kidney (carcinoma, sarcoma, hypernephroma and polycystic disease), or of the renal pelvis (papilloma, carcinoma) (§ 424). The hæmaturia is often painless, and either intermittent or continuous.

VIII. ESSENTIAL HÆMATURIA (nephritis dolorosa hæmorrhagica) is a name given to a group of cases in young adults in which either slight or severe unilateral hæmorrhage is accompanied by paroxysmal colic or dull renal pain. The cause is probably a patchy nephritis; bleeding usually ceases dramatically after nephrotomy; nephrectomy should not be performed.

IX. PAROXYSMAL HÆMOGLOBINURIA is not, strictly speaking, hæmaturia. Free hæmoglobin is plentiful in the urine (§ 409), but blood discs are absent.

X. **Injury of the Kidney,** laceration or rupture, is usually caused by a fall on the back of the loin, or in " buffer accidents " on the railway, or in street accidents. There may be no bruising or external signs, but a laceration of the kidney may be inferred from (1) the history of such an accident; (2) a tense swelling (due to extravasated blood) with increased area of dulness in the region of the kidney; and (3) copious hæmaturia. In a few cases there is no hæmaturia, and the other two evidences have to be relied on. Immediate operation is sometimes necessary, the collapse being treated by blood transfusion or saline injections.

§ 408. Renal Calculus and Renal Colic.—Calculi may form either in the pelvis of the kidney (Fig. 103) or, more rarely, in its substance. Perhaps the commonest form, dark brown in colour, consists of *calcium oxalate*,

and gives rise to more acute symptoms, for each bristles with sharp-pointed crystals which cause bleeding and colour the calculus. Another form consists of *uric acid* and urates mixed in varying proportions (§§ 388, 393). These form light brown stones, round or branching, and are the commonest stones in gouty subjects, and those whose highly acid urine habitually deposits urates. These two stones occur in acid urine. Calculi are often multiple. Compound stones consist of an oxalate or organic nucleus, or alternate layers. Phosphate stones are rare and usually occur where infection is present, in an alkaline urine. Cystine and xanthin are rarely met with renal calculi. Various *events* may happen. (1) A large calculus may remain in the renal pelvis, giving rise to chronic pyelitis (§ 412) for years (Fig. 103); or (2) by its movement produce acute symptoms, RENAL COLIC. (3) It may obstruct the ureter and lead to hydro- or pyo-nephrosis (§ 424). (4) If the other kidney is not healthy sudden blocking may lead to obstructive suppression (§ 421). (5) It may pass into the bladder and result in cystitis. (6) Small stones may be voided through the urethra as "gravel." (7) In rare cases small calculi become encysted and quiescent. (8) Hypertension may develop (§§ 87, 403). The clinical history of renal calculus consists of (*a*) *attacks of renal colic*, separated by (*b*) *intervals* in which the symptoms are those of calculous pyelitis.

The *Symptoms of Renal Colic* consist of severe paroxysms of lancinating pain, starting in one loin, shooting down to the front of the thigh or testicle or vulva on that side. This is associated with retraction of the testicle and with frequency of micturition, and is attended by vomiting, shivering, sweating, pallor, and a certain amount of collapse. These symptoms are in most cases followed by hæmaturia, the urine containing blood and pus cells, but usually no casts. Crystals may be present, and reveal the nature of the stone. Most blood and pain occur with an oxalate calculus.

The *diagnosis* of renal from other forms of colic is given in Table XIV, § 246. Cystoscopic examination may reveal blood issuing from one ureteral orifice or even an impacted calculus. X-ray examination is of assistance except in the case of uric acid and cystine stones. All the symptoms of renal colic may arise simply from the irritation of *fine crystals*. They may also be produced without alteration in the urine by *movable kidney*; or by the passage of *clots* of blood or *caseous material* down the ureter. *Malignant* disease of the kidney may be mistaken for calculus, but in that case the blood is more copious and more constant, and the pain is less severe, but more continuous.

Treatment.—(1) Of the colic and (2) during the intervals. 1. The treatment of an attack of *renal colic* consists mainly in the relief of the symptoms—pain, vomiting, and collapse. Usually nothing avails except injections of morphia, papaverine, ephedrine, trasentin or atropin. Locally, hot applications relieve. Effervescing citrate of potassium with spiritus ammoniæ aromatici may be administered with advantage. After the painful attacks the patient must rest to allow the inflammation to sub-

side. 2. The treatment in the intervals resolves itself into the removal of the stone, and treatment directed to the pyelitis. The urine in all cases should be kept diluted by drinking plenty of fluid. Dietetic treatment is of great use in some cases. If oxalates are being passed, any dyspepsia should be carefully treated ; for diet, see § 297. XV ; if the urine is kept strongly acid with sodium acid phosphate or with ammonium chloride, the crystals are kept in solution. In uric acid cases a purin free diet is given. The alkaline waters are very useful here, such as those of Vichy, Ems, and Contrexéville. In uric acid calculus large doses of alkaline salts are certainly useful, especially the citrate and the acid tartrate of potassium. Begin with potassium citrate gr. 50 in 4 fl. oz. of water every four hours until the urine is alkaline, and then give an effervescing drink, consisting of sodium bicarbonate gr. 60, and citric acid gr. 40 in 4 ounces of water, t.i.d. This treatment should not be continued if the urine is or has become ammoniacal. For pyelitis, see § 412. Operative treatment is often called for, although small stones are often passed spontaneously. Before operation the function of the other kidney must be investigated.

Hyperparathyroidism due to an adenoma can occur with minimal bone changes : any patient with a renal calculus (especially if multiple) and a serum calcium over 12 mgm. per cent. should be suspect.

§ **409.** In **Paroxysmal hæmoglobinuria** porter-coloured urine is passed at intervals. An attack commences abruptly with (1) a rigor and temperature to 104°, nausea, headache and malaise. (2) Abdominal cramp, aching pains in the back and legs : severe shock may follow. (3) An hour or so later the patient passes dark, highly albuminous urine, showing the spectroscopic bands of methæmoglobin, oxyhæmoglobin and hæmatin, containing no red cells, but albumen, red cell casts and amorphous hæmosiderin may be present. Anuria is a dangerous sequela. (4) In severe cases, a hæmolytic anæmia is present which may endanger life. Thrombosis and infarcts are common. Free hæmoglobin can often be detected in the plasma as well as in the urine. Each attack lasts a few hours, passes off suddenly only to recur later.

The types are—(1) In 90 per cent. of the cases (Roberts) the attacks are connected with chill to the surface. In this type attacks are induced by immersing a hand in ice-cold water for 10–20 minutes. A hæmolysin is present in the serum which unites with the red cells when the temperature is lowered and lyses them when the temperature rises again (Donath-Landsteiner reaction). The cause is usually syphilitic as in most cases stigmata of acquired or congenital syphilis are present, the Wassermann reaction is positive, and the disease is cured by antisyphilitic measures.

(2) In the second decade hæmoglobinuria may follow severe exertion, and especially running on a metalled road. Hæmoglobinæmia is usually absent, and it has been suggested the hæmolysis occurs locally in the renal vessels (Witts). It is often accompanied by lordosis, and treatment to overcome this helps. Patients are spontaneously cured in a year or so.

(3) Recurrent hæmoglobinuria, usually occurring at night with a hæmolytic anæmia (Marchiafava-Micheli syndrome), is often associated with splenomegaly, some bronzing of the skin and a constant reticulocytosis. Sooner or later the severe anæmia and hæmolysis endanger life, but splenectomy and transfusions are unavailing. The cause is associated with an altered pH in the blood at night. Death usually occurs in three to five years.

(4) Massive toxæmia with Cl. Welchii or with the Bartonella of Oroya fever, and the hyperacute form of Lederer's anæmia, are occasional causes.

(5) Favism following ingestion of the sensitised portion of the bean Vicia fava.

(6) In paralytic hæmoglobinuria the pigment is myohæmoglobin and is associated with weakness and paralysis of skeletal muscles.

The *Diagnosis* depends on the precipitating cause. Only in the form due to exposure to cold is syphilis a factor.

The *Treatment* consists of rest in bed during the attacks, with warmth.

HÆMOGLOBINURIA AND METHÆMOGLOBINURIA occasionally accompany severe burns and acute infective diseases, especially malaria. It may be produced by toxic doses of chlorate of potassium, nitrites, pyrogallic acid, arseniuretted hydrogen, and quinine in those who have had malaria. Blackwater fever, see § 511. Hæmoglobinuria also occurs after incompatible blood transfusions (§ 537).

EPIDEMIC HÆMOGLOBINURIA is seen in the new-born, with jaundice and nervous symptoms.

F. *The patient complains of* LASSITUDE *and ill-health, which may be associated with fever; the urine is found to contain* PUS (§ 385)—*i.e., there is* PYURIA. *With few exceptions, when the pus comes from the* BLADDER *the urine is* ALKALINE, *and the pus remains diffused through the urine; but when it comes from the* KIDNEYS *or any other part of the urinary passages, the urine is* ACID, *and the pus settles at the bottom. Pus cells are often accompanied by a trace of albumen in the urine.*

§ **410. Pyuria.**—If we except the rupture of an abscess into the urinary passages, there are three sources of pus in the urine:

1. From the **Urethra** (*e.g.*, gonorrhœal or B. coli infection).
2. From the **Bladder** (cystitis).
3. From the **Kidney** (pyelitis).—The chief forms are due to B. COLI INFECTION, or to ASCENDING, CALCULOUS or TUBERCULOUS PYELITIS.

Abscesses bursting into the Urinary Tract.—The abscesses most liable to burst into the urinary tract are: abscess due to diverticulitis; prostatic abscess (below); perineal abscess; pelvic cellulitis; psoas abscess; perinephric abscess; and abscess of the liver; and there are also many other sources. (i.) The urine is usually acid; (ii.) the pus is in large quantity and settles at the bottom; (iii.) there is a clinical history of abscess prior to the appearance of pus in the urine; and (iv.) localising signs of the abscess may be present.

It is believed by some observers that persons in health may pass a few leucocytes, but it is extremely probable that these are always derived from the generative organs (male or female), and that the occurrence of *any* pus cells in a properly collected catheter specimen is always pathological: a number of leucocytes are present in acute and subacute nephritis. Special precautions may have to be taken to exclude pus mixing with the urine as it is passed (false pyuria). When the presence of pus is suspected, the reaction should be tested immediately after it is passed, before decomposition makes the urine ammoniacal.

1. *The pus comes chiefly at the* BEGINNING OF MICTURITION, *and the urine is* ACID; there is PAIN IN THE URETHRA during micturition. The pus comes from the URETHRA, and is usually caused by one of three conditions:

I. URETHRITIS.—There is pain, swelling, and redness of the meatus, scalding during micturition, and discharge of pus cells (often with gonococci) apart from micturition.

II. PROSTATIC ABSCESS is known by: (1) pain in the perineum, which is worse at the end of micturition; (2) the finger in the rectum detects a tender, fluctuating swelling; (3) the symptoms closely resemble those of vesical calculus with concurrent cystitis. It may be distinguished from this, however, by: (i.) a history of gonorrhœa, which is the chief cause of prostatic abscess; (ii.) the signs on examination per rectum; and (iii.) a discharge occurring in the intervals between micturition.

III. PERINEAL ABSCESS is detected by the local signs.

2. *The* **pus** *comes chiefly at the* END OF MICTURITION, *or is intimately mixed with the urine. There is* SUPRAPUBIC PAIN *or* DISCOMFORT *and frequency of micturition.*[1] The pus comes from the BLADDER, and is indicative of CYSTITIS.

§ **411. Cystitis,** or inflammation of the bladder, occurs in two well-recognised forms—acute and chronic.

(*a*) ACUTE CYSTITIS.—(1) In this condition the pus is in small amount, and in severe cases there may be considerable hæmaturia at the onset. At first the urine is acid, but it soon becomes alkaline, and ropy with pus and mucus. (2) There are pain and tenderness in the hypogastrium. (3) Micturition is frequent and painful ("scalding"). There is a constant desire to pass water immediately after micturition (strangury); this relieves the pain for a short time, unless the cystitis is due to stone in the bladder, when the pain is severe *after* micturition, because the inflamed walls of the emptied bladder come into contact with the stone. (4) There is generally marked constitutional disturbance, with pyrexia.

(*b*) In CHRONIC CYSTITIS (which may supervene upon the acute form, or may be chronic from the onset), there is (1) a larger amount of pus. (2) The urine is alkaline directly it is passed and contains a large amount of ropy mucus.[1] (3) The pain and other symptoms are less severe than in acute cystitis.

Etiology.—Infection of the bladder is rarely primary in origin. Predisposing causes are: (i.) the presence of residual urine, the bladder never being completely emptied. This occurs with prostatic enlargement, urethral stricture, atony of the bladder in old age, and various nervous disorders producing paralysis and retention (§ 420). (ii.) Stone or foreign body; (iii.) papilloma, carcinoma and other tumours; (iv.) diverticulum of the bladder; (v.) following surgical operations on the bladder or other pelvic organs. Infection spreads to the bladder: (i) in the stream of urine from the renal pelvis, as in B. coli and tuberculous pyelo-nephritis; (ii.) from adjacent organs, especially cervicitis, diverticulitis or growths of the colon and rectum (forming a fistula), pelvic cellulitis or pelvic peritonitis; (iii.) via the urethra, as after the passage of infected instruments or foreign bodies introduced by the patient, by extension of urethritis, and possibly via the wide urethra of women, especially when there is leucorrhœa or infection of Bartholin's glands or Skene's ducts.

[1] The urine may be acid—(i.) at the onset of acute cystitis; (ii.) in the stage of recovery from chronic cystitis; (iii.) in the early stage of tubercle and new growths of the bladder; (iv.) in cystitis due to Bacillus *coli communis*. In all other conditions in which the urine contains pus derived from the bladder the reaction is alkaline.

Almost any variety of organism may be found, but the most common are (*a*) in acid urine B. coli, tubercle bacilli and gonococci, (*b*) in neutral urine B. coli and streptococci, (*c*) in alkaline urine staphylococci and B. proteus. For schistosomiasis, see § 407.

Differentiations.—(1) Cystitis due to VESICAL CALCULUS.—In addition to the symptoms of simple cystitis, there are (i.) very severe pain at the end of micturition lasting for some time after, shooting down the urethra; pain is often much worse after jolting, as on a bus ride. (ii.) Hæmaturia is common, though in some cases it may be so slight that it is detected only by the microscope; (iii.) sometimes a preceding history of renal colic (§ 408); (iv.) the stone may be detected by the sound, the cystoscope or by radiography.

(2) Cystitis due to NEW GROWTH IN THE BLADDER, or ULCERATION, is characterised by (i.) continuous suprapubic pain, with paroxysms of lancinating pain, quite independent of micturition and movement; (ii.) copious hæmorrhage at intervals; (iii.) the urine may contain cancer cells or tubercle bacilli; a tumour may be felt per rectum or through the abdominal wall. (iv.) Cystoscopic examination or radiography may settle the diagnosis. The cancerous ulcer is often covered by a fine deposit of calcium phosphate which gives a characteristic X-ray appearance.

(3) ABACTERIAL PYURIA causes the symptoms and signs of cystitis and urethritis, but organisms cannot be cultured from the urine and tubercle bacilli cannot be found. The amount of pus from the bladder and urethra may be considerable and cystoscopy reveals generalised cystitis. *Treatment:* the disease fails to respond to the usual urinary antiseptics but disappears rapidly after 1 or 2 small doses of neoarsphenamine. Relapse may occur unless septic foci are treated.

Prognosis.—Cystitis is not dangerous to life unless the inflammation spreads upwards from the bladder to the kidneys and produces pyelonephritis; but, on the other hand, it is a very troublesome, painful complaint, and has a special liability to recur. When the cause is not removable—*e.g.*, in cystitis due to tumours of the bladder—the prognosis is very grave. When it is due to retention of urine, and when it is due to gonorrhœa, it tends to cause ascending pyelitis and pyelo-nephritis. When there is pre-existing hydronephrosis (§ 424), and acute cystitis develops, the inflammation is almost certain to extend upwards to the kidney, and so lead to pyonephrosis.

Treatment.—The cause must be sought for, and, if possible, removed. (*a*) Otherwise, in the *acute* form absolute rest in bed with milk diet is necessary. Copious libations of water, barley-water, and other bland fluids are called for. The drug chosen will depend on the infecting micro-organism: with coli infections, a sulphonamide drug with alkalies, streptomycin or mandelic acid preparations should be used as for pyelitis (§ 412); but in coccal infections penicillin or hexamine and sodium acid phosphate should be used—otherwise boric acid (gr. 10) and salol (gr. 5). The sulphonamide group of drugs has been found to have a lethal effect on gonococci, and on many strains of B. coli, B. proteus vulgaris, and occasionally staphylococci. B. proteus and Ps. pyocyaneus infections are most obstinate and often respond to chloromycetin or to lavage with acriflavine (1 in 8,000). Hyoscyamus, hot sitz-baths and morphia suppositories relieve pain. (*b*) For the *chronic* and *subacute* forms, apart from the drugs just

mentioned, it may be necessary to wash out the bladder with warm water and boric acid (gr. 10–20 to 1 fl. oz.), or with mercury oxycyanide ($\frac{1}{4000}$–$\frac{1}{8000}$), followed by normal saline to prevent mercurialism. Cystopurin, hexyl-resorcinol and pyridium are also good. *Prophylactically*, penicillin injections are very valuable in those with a paralysed bladder, *e.g.*, in paraplegia.

3. *The* **pus** *is associated with a urine which is* ACID *when freshly passed* **(acid pyuria)**, *the pus cells are at first disseminated through the urine, but in a short time they settle down as a* SEDIMENT, *and there is* PAIN, *perhaps* SWELLING *of the kidney, and* PYREXIA ; the pus comes from the kidney—the disease is PYELITIS.[1]

§ **412. Pyelitis,** or inflammation of the pelvis of the kidney, is indicated by the symptoms just mentioned. The urine, which is acid unless there be concurrent cystitis, contains, in addition to pus cells (Fig. 95), epithelial cells from the renal mucosa and often red blood cells; but, unless the renal parenchyma is involved, no casts and no albumen in excess of the quantity which would be accounted for by the pus are found, nor is there any dropsy. There is increased frequency of micturition. Renal pain and tenderness are nearly always present, but they vary widely in degree and character in the three varieties about to be mentioned. *The kidney should always be carefully examined* (§ 394), because, in addition to the renal congestion, all forms of pyelitis are liable to result in partial or complete obstruction of the infundibula, and the gradual supervention of pyonephrosis. A few pus cells in the urine may be found in acute nephritis, with typhoid and other fevers, and toxic doses of cantharides or turpentine. Apart from these there are four well-marked varieties of acid pyuria.

(I) PRIMARY INFECTIVE PYELITIS.—This is the commonest group, and in the majority of cases the kidney is primarily infected from the blood stream by the bacillus coli. The disease occurs chiefly in females, either children or adults, and especially during pregnancy. The right kidney is most commonly involved, but both kidneys or only the left may be affected. The disease may be acute or chronic. (*a*) ACUTE PYELITIS. *Symptoms:* (i.) *Constitutional:* sudden onset, with headache, languor, anorexia, shivering, sweating, dry furred tongue and pyrexia of swinging type, up to 103 or 104° F. In adults, one or more rigors, and in children convulsions may occur. (ii.) *Urinary:* The first symptoms are often due to bladder irritation—frequency of micturition, perhaps every 15 to 20 minutes, sometimes associated with strangury. Later, a dull ache in the loin is felt, and local tenderness and rigidity develop. Sometimes acute abdominal pain may simulate appendicitis. The urine is concentrated, and contains a trace of albumen, pus cells, bacilli, occasionally

[1] In obscure or resistant cases it may be necessary to collect a specimen of urine from each kidney by ureteric catheterisation. This will confirm that pus cells and organisms are derived from the *kidney*, and will decide whether from one or both sides. This procedure should never be undertaken if the bladder is extensively septic, lest ascending pyelo-nephritis be induced.

a considerable amount of blood. In the earlier stages the amount of pus and bacilli may be microscopic, but later the urine is uniformly turbid, gives a shimmering appearance when rotated in a glass, and has an unmistakable fishy odour, due to the presence of sulphides. (*b*) Chronic Pyelitis or Bacilluria. *Symptoms:* (i.) *Constitutional:* general ill-health, headache, periodic low-grade pyrexia. (ii.) *Urinary:* frequency of micturition, enuresis in children, pain in the loins or hypogastrium. The urine shows a trace of albumen, pus, bacilli and occasional hæmaturia, as in the acute form.

Etiology.—(i.) The *Bacillus coli* is the common infecting agent and may arrive (*a*) from the blood stream, (*b*) from the colon, and (*c*) from the pelvic organs by the lymphatics. (ii.) When other organisms are present, such as staphylococci, streptococci, *B. pyocyaneus*, *B. proteus* or *B. subtilis*, the infection is often secondary to some other disease of (*a*) the kidneys, such as hydronephrosis, tuberculosis, or to some congenital abnormality, such as double ureters: or (*b*) disease of the bladder, prostate, cervix uteri, Bartholin's glands or Skene's crypts.

II. Ascending Pyelitis or Pyelo-nephritis arises from: (*a*) *obstruction of the urinary passages* below the kidney. The resulting retention and decomposition of the urine causes infection to arise, which may go on to pyo-nephrosis. (*b*) *Extension of cystitis* without obstruction, and thus the numerous causes of the latter disease (§ 411) are brought into operation. *Symptoms:* (i.) A high swinging temperature, often with repeated rigors. (ii.) Pain, tenderness, rigidity, and often a considerable enlargement of the kidney may be felt in the loin. (iii.) There may be a history of the cause, *e.g.*, enlarged prostate, renal calculus. (iv.) Often both kidneys are involved, with gradual diminution of the urinary output and symptoms of uræmia. *Treatment:* Surgical aid should be sought early. If time permits, special investigations as to the cause and the functional condition of the kidneys should be undertaken.

III. Calculous Pyelitis is due to the irritation and obstruction set up by the presence of a stone. The *Differential Symptoms* are: (i.) A history of renal colic (§ 408) is often obtainable. (ii.) Pain on the diseased side, which varies with exercise; and (iii.) hæmaturia, also varying with exercise. (iv.) The quantity of pus often varies from day to day, and the patient may feel easier after its discharge, as the retained pus causes pain, and sometimes swelling. (v.) Attacks of intermittent pyrexia and sometimes rigors. (vi.) Crystals in the urine aid the diagnosis considerably.

IV. Tuberculous Pyelo-nephritis.—Tuberculous disease of the kidney may be primary or secondary to tubercle elsewhere, and is often associated with tuberculous infection of the genital system. Sometimes both kidneys are diseased. This condition may be difficult to diagnose from Calculous Pyelitis. *Differential Symptoms:* (i.) Increased frequency of micturition, and perhaps strangury, is the commonest early symptom; (ii.) hæmaturia occurs in 75 per cent. of cases (Fullerton); (iii.) dull pain in the loins, liable to colicy exacerbations from the passage of caseous masses; (iv.) *pyrexia of a regularly intermitting type;* (v.) the urine is acid, contains some albumen, pus and often red blood cells. Tubercle bacilli may be demonstrated in the deposit of a 24-hours' specimen, by culture, or by guinea-pig inoculations; other organisms are absent unless secondary infection has occurred. A sterile pyuria

is often tuberculous or due to abacterial pyuria; (vi.) the cystoscope may show the presence of swelling or ulceration at the mouth of one ureter. The tuberculous focus in the kidney may or may not be constantly in connection with the ureter (the "open" and the "closed" types). In the latter, many specimens of urine may have to be searched before tubercle bacilli are found.

Prognosis.—With modern methods of treatment, uncomplicated coli pyelitis usually clears up within 3–4 weeks, and provided the urine has been rendered sterile, as shown by examination of catheter specimens, relapse is unlikely. The course of ascending pyelitis depends very much upon the cause, the possibility of its removal, the age of the patient and the general condition: it used to be the common mode of death after fracture of the spine or transverse myelitis. Calculous pyelitis may last for years, but after surgical removal it may be possible to sterilise the urine with modern drugs. In the tuberculous form the prognosis depends on whether one or both kidneys are involved, the possibility of successful surgical removal, and the presence of lesions elsewhere. Pyelo-nephritis and pyonephrosis (§ 424) may follow all the chronic forms of pyelitis.

Treatment.—In all forms of pyelitis fluid diet and warm drinks, rest in bed and warmth, are essential; cupping of the loins is sometimes useful. (1.) The most common form is that due to bacillus coli infection. When there is fever, the best drugs are equal parts of potassium citrate and sodium bicarbonate (grs. 30 of each). This dose must be given four, three or two hourly (even at night) until every specimen of urine is alkaline to litmus paper. Sulphanilamide or one of the other sulphonamides (G. 1 six-hourly) can be combined with these doses of alkali, and acts best in a strongly alkaline urine.[1] At the same time copious drinks of fluid must be given (4 to 6 pints daily); with this the temperature should settle in 3 to 4 days and the urine be sterile in 7 to 10 days. If alkalies alone are used once the fever and vomiting have been controlled, it is preferable to make the urine strongly acid. For this purpose hexamine (gr. 20) before meals, and sodium acid phosphate (gr. 30) after meals, were formerly used and are still most effective with those forms of B. coli which are "late lactose fermenters." Apart from these older methods, streptomycin is giving excellent results. The ketogenic diet has been replaced by mandelic acid preparations. To obtain the maximum bactericidal effect fluid intake must be limited to 2 pints daily and the urinary acidity maintained below pH 5·5 (*i.e.*, a red colour when tested with methyl red). Sodium mandelate (G. 3 t.i.d.) is combined with sodium acid phosphate or ammonium chloride (G. 1–3 t.i.d.); these are better given as ammonium mandelate (G. 3 t.i.d. or q.i.d.). Calcium mandelate in similar doses is equally efficacious and less nauseating. With these preparations the urine should be sterile in 2–3 weeks. Whichever method is used, it must be continued until two successive specimens of urine at

[1] In view of the very variable susceptibility of different strains to the various sulphonamides, in a resistant case it is wise to perform laboratory tests to determine which sulphonamide is best.

an interval of 1–2 days are sterile. *General measures.*—Diet is important. Food of high protein content should be avoided; skimmed or ordinary milk, raw salads and fruits and wholemeal bread taken freely. The bowels must be regulated by mild aperients, such as paraffin, petroleum agar, senna, etc., so that the stools are neither constipated nor loose. Colon irrigation is sometimes advisable. *B. acidophilus* and intestinal antiseptics aid. (2.) When the infecting pyogenic organism is other than *B. coli* the urine must be kept acid with sodium acid phosphate, and antiseptics used, such as hexamine, hexyl-resorcinol or pyridium. Preparations of mandelic acid sometimes succeed in cases other than those due to *B. coli*. In staphylococcal infections penicillin injected is valuable: boracic acid (gr. 10) and salol (gr. 10) are sometimes effective. (3.) Many cases call for nephrectomy or other surgical measures. Before operation it is necessary to determine which kidney is diseased and the state of activity of the healthy kidney. This is seen by the cystoscope, the ureteral catheter and sometimes by X-ray. In cases of *calculous pyelitis*, large doses of potassium citrate and bicarbonate may be employed for uric acid calculi; for oxalates, see Oxaluria (§ 423); and nephrolithotomy is needed in nearly all cases. (4.) In cases of *tuberculous pyelitis*, build up the general health. Excision of the kidney is to be advised if (i.) the other kidney is shown to be healthy; and (ii.) there is no tuberculous disease elsewhere in the urinary tract, lungs or intestines. Heliotherapy, properly carried out, is advisable in some cases, and a course of tuberculin has given good results.

A **diminution in the specific gravity,** *when marked and continuous, even in the absence of albumen, is suggestive of* CHRONIC INTERSTITIAL NEPHRITIS, *or more rarely* DIABETES INSIPIDUS. **A marked increase** *in the specific gravity is suggestive of* DIABETES MELLITUS.

§ **413.** The other **causes of altered specific gravity** are relatively less important, because they are identified mainly by other means. Nevertheless, the specific gravity of the urine is an extremely important feature, because, in the absence of sugar, it is a MEASURE OF THE UREA and SODIUM CHLORIDE EXCRETION, the specific gravity being higher in direct proportion to the amounts contained in a given sample of urine. Therefore, it is a very fair measure of the power of concentration of the two kidneys taken together (and see §. 376). For this purpose an early morning specimen is essential, to avoid the effect of food and drink consumed during the day.

The specific gravity is DIMINISHED in—

1. Increased intake of fluid.
2. When the kidney reserve is called upon (see introduction to this section), as in Chronic Interstitial Nephritis, Secondary Contracted Kidney, etc.
3. Polyuria, and all the diseases mentioned below under that heading, excepting Diabetes Mellitus.
4. Myxœdema and other conditions with lowered nitrogenous metabolism.

The specific gravity is INCREASED in—

1. Diabetes Mellitus (owing to the sugar).
2. Some renal diseases where the quantity of water is considerably diminished, such as Acute Nephritis, Subacute Parenchymatous Nephritis, or the Cardiac Kidney.
3. Febrile and other conditions where the nitrogenous disintegration is excessive.
4. Whenever the urine becomes concentrated by profuse sweating, vomiting, diarrhœa, or diminished intake of fluid.

An **increase** (POLYURIA), *or* **diminution** (OLIGURIA), *in the* **quantity of urine** *is complained of by the patient in several important diseases.*

§ **414.** In **Polyuria** it is necessary to measure the total diurnal quantity, since patients are very apt to mistake increased frequency for increased quantity, and *vice versa.* It must be remembered that *in old age*, there is normally some increase in the diurnal excretion due to loss of concentrating power in the renal tubules.

Otherwise there is INCREASED QUANTITY of urine secreted in—

1. *Diabetes mellitus,* which is known by the high specific gravity of the urine and persistent glycosuria.
2. *Diabetes insipidus*—low specific gravity and malaise, but no sugar.
3. *Chronic interstitial nephritis,* which is known by the persistent low specific gravity of the urine, slight albuminuria, etc. (§ 401).
4. *Amyloid kidney,* which is known by the low specific gravity of the urine and great albuminuria (§ 404).
5. *Dietl's crisis* is known by a dull pain in the loin which becomes more severe, associated with an enlarged tender, and often mobile, kidney. As the pain subsides, polyuria occurs for a few hours, with decreasing tenderness and swelling of the affected kidney: recurrences are common. The condition is not due to hydronephrosis but to temporary engorgement of a mobile kidney which becomes twisted on its pedicle: the polyuria is a reaction to the establishment of a normal blood flow as the attack subsides.
6. *Convalescence* after fevers.
7. *Temporary polyuria* occurs in hysteria, nervous excitement, Dietl's crises, alcoholism, following an attack of paroxysmal tachycardia or of asthma, and any condition giving rise to a reactionary or paralytic condition of the abdominal sympathetic. Cerebral tumours may be accompanied by polyuria.
8. During the administration of *diuretics.*
9. During the *absorption of exudations,* such as general anasarca.

There is DIMINISHED QUANTITY of urine in—

1. Acute Nephritis.
2. Subacute Nephritis.
3. The final stage of Chronic Interstitial Nephritis and of Secondary Contracted Kidney.
4. The Cardiac Kidney and some other Renal Congestions.
5. Febrile states.
6. Whenever there is profuse vomiting, diarrhœa, or perspiration, or when little fluid is taken.

The patient complains of **polyuria** ; *the urine is of* HIGH SPECIFIC GRAVITY, *and* CONSTANTLY *contains* GLUCOSE **(glycosuria)** ; *there are also fatigue, thirst, and, in spite of a voracious appetite, gradual loss of flesh.* The disease is DIABETES MELLITUS. (See § 381 for Fallacies.)

§ 415. Temporary Glycosuria may arise in many conditions in which the carbohydrate metabolism is deranged; often it is of little or no consequence. (1) There may be a temporary diminution of sugar tolerance, particularly with septic infections (boils, etc.) in the elderly. (2) Chronic alcoholism. (3) Graves' disease. (4) Pregnancy and suckling (lactosuria). (5) Conditions, such as meningitis or tumour affecting the brain, especially the pituitary or the fourth ventricle. (6) Dietetic errors, as after a heavy meal, especially in the obese. (7) During the paroxysms of ague and collapse of cholera. (8) Chronic nephritis and high blood pressure. (9) After acute fevers, such as influenza or diphtheria. (10) At times of sudden emotion or physical stress (*e.g.*, asphyxial conditions), due to excess of adrenalin in the blood. (11) After epileptic fits.

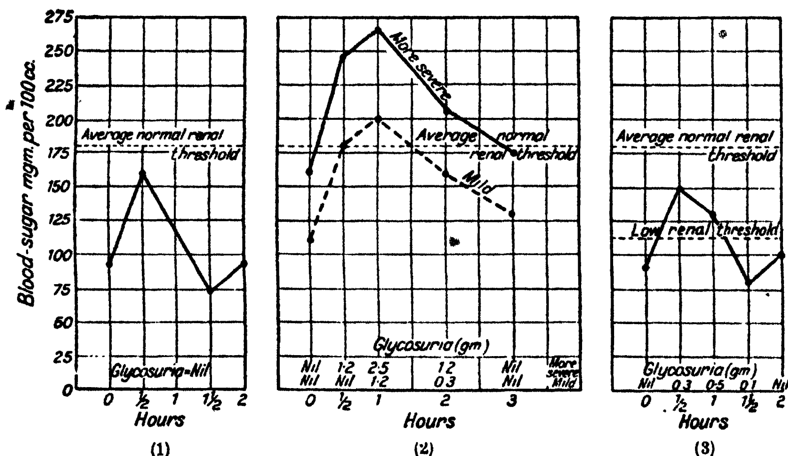


FIG. 108.—SUGAR TOLERANCE CURVES after 50 grammes glucose, with corresponding urinary sugars. (1) Normal. (2) Curves of mild and more severe diabetics. (3) Renal glycosuria showing lowered renal threshold.

(12) **Lag glycosuria** is a condition where the blood sugar rises rapidly after a meal to a value above the renal threshold; the fasting value is normal and the blood sugar returns to normal at the usual rate. It is believed to be due to a delay in the action of insulin. Its presence can only be satisfactorily determined by a sugar tolerance curve, and it has no clinical significance.

(13) **Renal glycosuria** (Diabetes innocens, renal diabetes). When a small quantity of sugar is excreted in the urine, and yet the blood sugar is not above normal (§ 416), the condition is one of renal glycosuria. The threshold, or point at which sugar is excreted by the kidney, is lowered. Renal glycosuria may be found accidentally whilst the urine is being examined. The sugar excretion in this condition is not much affected by increasing the carbohydrate in the diet; in the true diabetic the contrary is true. A sugar tolerance test and a study of the blood sugar curve is required before diagnosing the glycosuria as renal (Fig. 108 (3)). No treatment is required for this condition.

§ 416. Diabetes Mellitus is a constitutional disease, characterised by the passage of large quantities of urine containing glucose.

Symptoms.—The patient may first complain of the symptoms of the disease itself, or of one of its complications (*e.g.*, cataract, gangrene). The primary symptoms are: (i.) The urine is abundant (polyuria), and may amount to 6–20 pints a day; clear, pale, but of high specific gravity,

1030–1050. The specific gravity is always higher than would be expected from the concentration as judged by the amount of pigment present. Sugar may be in amounts varying from 1 to 9 per cent., is often accompanied by ketone bodies, and a trace of albumen may be present. If the urine drops on the boot, a crystalline deposit may be noticed by the patient. (ii.) Excessive thirst (polydipsia) and a dry tongue, which may become raw and beefy. (iii.) Loss of weight may be extreme, and is a gauge of the severity of the condition. (iv.) The appetite is normal or excessive (especially in relation to the weight), unless ketosis or other complications are present, when it usually fails. (v.) General symptoms such as lassitude, progressive weakness and ready fatigue. (vi.) The skin may lose its elasticity and become dry : it often acquires a yellow tint, especially on the hands and face, by which the disease may be suspected. This is due to an excess of a yellow pigment (carotin) circulating in the blood and staining the tissues. (vii.) The blood sugar is above the normal. This may be determined by estimating the fasting value (normally ·07–·10 per cent.) or by a sugar tolerance estimation. In this test, after determining the fasting value, 50 grammes of glucose are administered and the blood sugar value determined each ½ hour for 1½–2 hours. Typical curves are shown (Fig. 108).

Varieties. There are two well-marked varieties : (*a*) A mild form met with in corpulent middle-aged people, where the symptoms are moderate, and dietetic restriction removes the sugar from the urine. (*b*) The severe variety assumes *acute* and *chronic* forms. The acute form usually occurs in children or young adults, and occasionally after head injuries. The chronic form is met with in older people, and is attributed sometimes to mental worry. It also occurs with other causes of temporary glycosuria (§ 415) which become chronic.

Etiology. (1) The usual cause is insufficient insulin production by the β cells of the islets of Langerhans. This may be due to (*a*) an inherited tendency. The disease often runs in families ; in successive generations it tends to occur at an earlier age, with corresponding increase in severity of the disease. (*b*) Infections : A *generalised infection* (i.) calls for a greater output of insulin, which may not be forthcoming, and (ii.) may damage the pancreatic cells. With *acute infections* (boils, carbuncles, pneumonia) the disease may first manifest itself, the condition being temporary or permanent. (*c*) Progressive fibrosis of the pancreas occurs in hæmochromatosis and sometimes in tertiary syphilis. (*d*) A gradual obliteration of blood supply is met in arterio-sclerosis of the cœliac axis and pancreatic arteries. (2) Overaction of the thyroid gland, especially primary thyrotoxicosis, causes a rise in blood sugar level which the pancreas tries to correct. When thyrotoxicosis and diabetes occur together, wasting is rapid and often extreme. In some cases of thyrotoxicosis the blood sugar level is not raised, but glycosuria occurs due to a lowering of the renal threshold (renal diabetes). (3) Oversecretion of the pituitary, as in tumour, basophilism, acromegaly, etc., may produce glycosuria either

temporarily or permanently. (4) Temporary glycosuria from overaction of the suprarenals occurs in times of sudden stress and emotion, as at a medical examination or with athletic sports.

Complications are numerous : (1) *Ketosis* and *coma* are due to defective fat metabolism. The excessive fat utilisation is shown by the excess of fat in the blood (lipæmia) : in the absence of sufficient glucose utilisation, the end products of fat metabolism cannot be converted to CO_2 and water, and accumulate in the blood as β oxybutyric and aceto-acetic acids. The former is comparatively harmless but the latter stimulates respiration and depresses the brain, producing drowsiness and finally coma. In the more usual form of diabetic coma these ketone acids are secreted by the lungs and kidneys, and by losing CO_2 are partly converted to acetone, giving a sweet-smelling breath and the ferric chloride and Rothera's tests in the urine (§ 384). A rarer and more fatal variety is that in which the kidneys are unable to secrete these ketone bodies (anuric form), the urine being scanty or absent, containing albumen and abundant casts, and with a corresponding rise in blood urea. The symptoms of ketosis are (i.) in the *earlier stages*, loss of appetite, abdominal pain, nausea and vomiting: drowsiness is usually present but occasionally may be replaced by undue restlessness, irritability and giddiness. In the *later stages* coma develops. This is accompanied by slow deep breathing (" air hunger "), a sweet-smelling breath, diminution in the urinary volume which may be extreme in the anuric form, and usually the presence of acetone and aceto-acetic acid in the urine, with a lowered intraocular tension of the eyeballs. (2) *Infections*, especially staphylococcal and tuberculous, are liable to arise. The former may give rise to skin infections—pruritus vulvæ, boils, carbuncles and deep-seated abscesses ; the latter commonly causes pulmonary tuberculosis. It is essential to examine the urine in all cases of pruritus vulvæ, boils and carbuncles, and of acute infections, especially in elderly subjects with pneumonia, who are not responding satisfactorily to treatment. (3) Ocular changes : cataract, retinitis, optic neuritis and atrophy, and blurred vision due to rapidly developing short or long sight. (4) Cardiovascular changes, especially arterio-sclerosis of the larger and medium sized vessels, often associated with hyperpiesia. Gangrene readily supervenes in the toes and feet, usually of the dry variety ; secondary infections may produce a moist gangrene. (5) Polyneuritis (§ 794. X) is common, and cerebral changes (depression or restlessness, mania and melancholia) make satisfactory treatment difficult. (6) Pregnancy markedly increases the need for insulin : the fœtus often dies in utero in the last month of pregnancy. The size of the fœtus and of the newly born is markedly above normal.

Diagnosis.—In any of the conditions mentioned under *Complications* the urine should be examined. This is the key to the diagnosis. In *diabetes insipidus, granular kidney, amyloid kidney*, and sometimes in *hysteria* the quantity of urine is excessive, but in none of these conditions is sugar present. Two golden rules will enable us to identify a case of

diabetes which otherwise might be overlooked: Always examine the urine of a patient suffering from (1) boils or eczema of the genitals and (2) apparently causeless wasting. Other causes of glycosuria are discussed in § 415.

Prognosis.—1. The glycosuria which is met with chiefly in corpulent persons and others over thirty-five—the so-called "alimentary" glycosuria—has no thirst or other symptoms, but may be true diabetes. Generally with suitable diet and weight reduction the sugar disappears, and the condition warrants an excellent prognosis. 2. In the severer forms the prognosis chiefly turns upon the age of the patient. Before the discovery of insulin if the disease were established in a young adult, life rarely lasted more than two years. Since the discovery of insulin the outlook has much improved. The prognosis is now largely dependent on the patient's careful fulfilment of directions with regard to diet and insulin. The presence of *complications* does not materially add to the gravity of the situation except when phthisis, severe septic infections and gangrene are present. Operative risks, especially under general anæsthesia, are markedly increased when diabetes is present. Death may ensue in three ways: (i.) By complications—a third of the cases die of phthisis; (ii.) asthenia; and (iii.) with coma, a contributory cause of which is often a septic focus such as tonsillitis or otitis media.

The *Treatment* of diabetes has been revolutionised by the introduction of insulin, and the previous methods of starvation have been completely superseded. Insulin is injected to supplement the patient's own insulin. The principles of treatment are: (i.) sufficient calories must be given to maintain normal nutrition; (ii.) the diet taken must prevent ketosis; (iii.) as much variety as possible should be allowed; (iv.) the blood sugar should be maintained within normal limits.

To calculate the calories necessary for a patient, we must remember that elderly obese diabetics should be given the number of calories equivalent to their weight before obesity set in: many of these become sugar free by simple restriction of bread, sugar and other carbohydrates, and their sugar tolerance may return to normal as they lose weight (Embleton). In children, extra calories have to be allowed for growth. In adults, the values are: (*a*) For sedentary workers, 25 calories, (*b*) for those doing moderate muscular work, 35–40 calories, and (*c*) for heavy manual workers, 50–60 calories per kilo body weight. In pyrexial conditions, and especially in phthisis, the diet must be more liberal, but subjects with uncomplicated diabetes must not be allowed to become overweight. Having fixed the daily allowance of calories, the amount to be given as protein is usually next determined at about ⅔ gram per kilo body weight. The remaining calories have to be distributed between fat and carbohydrate.[1] The present tendency is to give more carbohydrate than formerly; 100–150 grams a day are commonly given. Some physicians are giving amounts as high as 200–250 grams daily. The remaining calories are given as fat. The advantages of a high carbohydrate low fat diet, as in Rabinowitch's scheme (fat not above 50–55 gms., carbohydrate 200–250 gms.) are (i.) it is more palatable and more closely

[1] Many of the advertised starch-free breads are by no means what they claim to be; the careful physician should examine them for starch with the iodine test, and for sugar by boiling them with dilute sulphuric acid, neutralising with caustic potash and adding Fehling's solution. Soluble saccharin B.P. is taken in place of sugar.

resembles the normal diet, (ii.) it is cheaper, (iii.) the sugar tolerance increases in proportion to the carbohydrate value of the diet, (iv.) insulin requirements are not greater than on a high fat low carbohydrate diet, (v.) the patient feels better, (vi.) ketosis and complications such as arterio-sclerosis and infective disorders are less common, (vii.) the heart muscle keeps in better condition. There are several convenient methods of giving as much variety as possible. Lawrence's "Line ration" scheme is easy to follow and is arranged to save trouble in calculating. A "black line" contains 10 G. of carbohydrate (41 calories) and a "red line" contains 7½ G. of protein and 15 G. of fat (114 calories). The carbohydrate of one line may be replaced by the corresponding number in another line. A slight modification of this scheme will allow a higher carbohydrate diet. A convenient list of diets is given in § 297. IX.

INSULIN B.P., better known as soluble insulin (S.I.), must be given when the patient cannot be maintained sugar free on the correct diet. It should be started immediately the patient is seen if the amount of ketosis in the urine is sufficient to give a positive ferric chloride test. During stabilisation the urine must be tested four-hourly, and the insulin administered once, twice or three times daily according to the severity of the case, 15–20 minutes before the principal meals. The dose should be increased by 5 units daily until the urine is sugar free, and then it is wise to perform blood sugar estimations to make sure the values are within normal limits. For severe cases, double and quadruple strengths of insulin are available.

Later preparations of insulin are: (*a*) protamine insulin with zinc (P.Z.I.) in which insulin is combined in an insoluble form, and is slowly liberated in the body. Its action starts some 10 hours after injection and is maintained up to 24 hours or more, depending on the dose. P.Z. insulin is much weaker in its actions than is soluble insulin, and cannot be used in diabetic coma. It is useful in mild diabetics, when it is given in a single morning dose: more commonly it is used with soluble insulin and given as a single injection before breakfast: the soluble insulin is withdrawn into the syringe first, and the P.Z. insulin added, this order being necessary to prevent P.Z. insulin being introduced into the soluble insulin bottle. The combination enables the soluble insulin to act soon after it is given, and the P.Z. insulin continues the action for the remainder of the 24 hours. In any case, with P.Z. insulin 10–20 G. of carbohydrate must be given at bedtime, to prevent hypoglycæmic reactions during sleep, for these are insidious in onset. (*b*) More recently globin insulin with zinc has been introduced: the action commences 2 hours after injection and continues for 16 hours or more, so that a pre-breakfast dose is liable to give hypoglycæmia in the late afternoon. After stabilisation with any of these preparations the dose of insulin may have to be varied from time to time as the disease gets more or less severe; and the patient should not only be instructed how to ward off hypoglycæmic reactions but also how to give his own insulin and test the urine regularly for sugar and acetone.

Ketosis is usually effectively controlled by the combination of insulin with increased carbohydrate and diminished fat in the diet. In pyrexial disorders, there is an increased need of insulin, whereas in pregnancy the dose must be immediately reduced by one half directly after childbirth.

§ 417. Treatment of Diabetic Coma. The main indications are to combat the ketosis and the dehydration which so often accompanies it. Immediately, 50 units of insulin followed by 1–2 pints of normal saline, or saline with added sodium bicarbonate, must be administered (intravenously, if possible). If *consciousness returns,* 500 G. of glucose should be dissolved in 2,500 c.c. of half-normal saline, and 100 c.c. administered by mouth each hour, with 10 units of insulin hourly, until the blood sugar level falls to 0·30 per cent. If the *patient is still unconscious,* 50 G. of glucose in

1–2 pints of normal saline are given (preferably) into a vein or by a duodenal tube, with 30–50 units of insulin subcutaneously each 4 hours until consciousness returns. Then the intravenous medication may be replaced by rectal glucose or the half normal saline and glucose by mouth, until with the control of the ketosis, milk, Benger's food, etc., may be commenced. In the "*anuric*" *variety* still larger doses of intravenous saline (3–4 pints in the first hour) with 50–100 units of insulin must be used : subsequently further intravenous dextrose saline (1–2 pints) with saline per rectum must be combined with 4-hourly insulin until the urine is passed in adequate quantities. When an infection has precipitated coma, often there is no pyrexia, but leucocytosis gives valuable confirmation and penicillin is often necessary. The doses of insulin may then have to be very large, even 500–600 units in 24 hours, but regular blood sugar analyses are essential when using such doses. The circulatory collapse must be met with warmth, the infusion of blood plasma and stimulants such as nikethamide (coramine) or adrenalin.

Estimation of the Blood Sugar. Folin-Wu Method.—Blood is obtained from a finger prick, and 0·2 c.c. is measured accurately into 1·6 c.c. of sodium tungstate solution [1] in a centrifuge tube. Then 0·2 c.c. of 2/3N sulphuric acid is added, and the whole is shaken. By this means the blood is diluted ten times, and the protein coagulated. The protein precipitate is centrifuged off, or allowed to settle, and 0·75 c.c. of the supernatant fluid is pipetted into the special hard glass boiling-tube. From two standard solutions containing respectively 0·01 per cent. and 0·02 per cent. glucose, 0·75 c.c. of each are placed in similar hard glass tubes. To each of these three, 0·75 c.c. of the copper solution is added, and the solutions shaken together. The tubes are boiled for exactly six minutes in a boiling water-bath, and after cooling in a cold water-bath for three to five minutes, 0·75 c.c. of the sodium molybdate solution is added, and each tube has distilled water added to the 9 c.c. mark. The relative depths of the colour of the blue solutions are compared in a colorimeter or in Nessler tubes, the amount of sugar present in each being proportional to the depth of the colour. Suppose a depth of 50 mms. of the unknown sugar solution matches a depth of 40 mms. of the 0·02 per cent. standard sugar solution, the unknown solution contains $\frac{40}{50} \times \cdot 02$ per cent. sugar. Allowing for the dilution of the blood $\times$ 10, the blood sugar value is $\frac{40}{50} \times 0{\cdot}02 \times 10$ per cent. $= 0{\cdot}16$ per cent.

The normal fasting blood sugar is 0·08 to 0·10 per cent. After a meal it may rise to 0·17 per cent. Values above 0·20 per cent. are abnormally high. The blood sugar becomes too low after an overdose of insulin, and may fall to 0·03 to 0·05 per cent.

§ 418. Hypoglycæmia and Hypoglycæmic Coma. Since overdosage by insulin causes too great a fall in the blood sugar (hypoglycæmia), the symptoms of this condition must be carefully watched for. There may be sweating, weakness, tremors, palpitation, inco-ordination of movements or of speech, "sinking feelings," numbness of lips and occasionally diplopia (§ 845. VIII). Such results may be warded off by a hot drink of milk or a tomato ; if these fail a little sugar in water is rapidly effective. In severe cases, these early symptoms are followed by unconsciousness and epileptiform convulsions, which may be fatal. The diagnosis is made by the symptoms and by blood sugar estimation ; symptoms occur when this is below 0·07 per cent. If mild symptoms of hypoglycæmia occur at the

[1] The solutions are bought from British Drug Houses, London.

same time on succeeding days, reduce the dose of insulin. Coma is usually due to carelessness on the part of the patient, as when the usual dose of insulin is taken without being followed by a meal. In the severe cases, the blood sugar must be raised by injection of adrenalin (♏ 10–15) or pituitrin (c.c. ½–1); or administration of glucose by a stomach tube or intravenously. Cases are recorded in whom symptoms of hypoglycæmia occurred spontaneously, just before a meal; adenoma of the pancreas was found and removed, with abatement of the symptoms.

The patient complains of **polyuria** *and many of the other symptoms of Diabetes Mellitus, but the* SPECIFIC GRAVITY OF THE URINE IS LOW, *and there is* NO SUGAR. The disease is DIABETES INSIPIDUS.

§ **419. Diabetes Insipidus** is characterised by great and persistent increase in the quantity of the urine, without glycosuria and albuminuria, attended by great thirst and emaciation. It is due to deficiency of the anti-diuretic principles of the posterior lobe of the pituitary, and may come on spontaneously or after head injuries.

Symptoms.—(1) The amount of urine may be very great, from 10 to 20 pints per day. It is pale in colour, so that it may resemble clear water. The specific gravity averages 1002 to 1005. The diurnal amount of solid constituents is as a rule not very much increased, and no other abnormality may be present. Occasionally traces of albumen and sugar appear towards the end. (2) In the mild form of the disease polyuria and thirst are the only symptoms; but in the severer variety nearly all the symptoms mentioned under Diabetes Mellitus are also present—dry skin, emaciation, large appetite, and alternating constipation and diarrhœa. Indeed, it is distinguished from that condition only by the absence of glycosuria. Intercurrent attacks of pyrexia have been observed. (3) Obscure nervous symptoms are common in this disease—irritability of temper, disturbed sleep, occipital headache, neuralgic pains in the lumbar region, diminished reflexes, and muscular twitchings.

Diagnosis.—The disease is apt in its early stages to be mistaken for *chronic interstitial nephritis*, but the greater age of the patient, the presence of traces of albumen, and of cardio-vascular symptoms, and the absence of thirst and voracious appetite distinguish the latter condition. With *amyloid kidney* there is albumen, and with both *hydronephrosis* and *polycystic kidney* a tumour is generally palpable in the region of the kidney. In *Diabetes Mellitus* there is glycosuria.

Causes.—More men than women are affected. Childhood and early middle age are the favourite ages. Causal factors are: injury to the posterior pituitary body, as by a primary tumour or secondary metastases, syphilis, meningitis, trauma, or xanthoma in Hand-Schüller-Christian disease.

Prognosis.—The milder varieties may last for many years, and exist rather as an inconvenience than as a malady. In the severer forms, especially those due to intracranial tumours, the course may be rapid. When setting in acutely after head injury (which may be attended by some glycosuria at first) recovery may ensue after a year or so. In general terms, acute cases are more hopeful than those which start insidiously. Death takes place from exhaustion, drowsiness passing into coma, with or without convulsions, or from complications such as phthisis or pneumonia.

Treatment.—Substances which increase diuresis, such as tea, coffee, alcohol and salt, should be avoided, but the amount of fluid taken should not be reduced below that which the patient can comfortably manage. The active principle in the pituitary is supplied by giving injections of posterior pituitary extract, or better still pitressin tannate in oil, both of which contain the antidiuretic factor. In some cases the missing factor can be given by painting pituitary extract on the nasal mucosa or by inhaling piton snuff. However these are given, the extracts lose their efficacy after a time. Thyroid administration sometimes helps. Anti-syphilitic treatment is given when there is a positive Wassermann. Lumbar puncture has been helpful in some cases.

The patient complains that he **cannot pass water,** *and a* DISTENDED BLADDER *can be made out by palpation and percussion above the pubes, or by the passage of a catheter.* The condition is RETENTION OF URINE.

§ **420.** The Causes of **Retention of Urine** come mainly within the province of the surgeon. Those of *sudden* onset are often due to urethral spasm or congestion; those of *gradual* onset are more numerous. The age and sex of the patient may aid us. Thus, in *childhood* we may suspect impacted calculus or foreign body, a congenital valve of the urethra, phimosis, or a ligature round the penis; in *women*, tumours pressing on the neck of the bladder (*e.g.*, fibroid or retroverted enlarged uterus), hysteria, or reflex irritation after parturition; in young or middle-aged *adults*, urethral stricture, gonorrhœa, with congested mucous membrane, spasm after exposure to cold or a drinking bout, or tabes dorsalis; in *old men*, prostatic enlargement, or atony of the bladder. At all ages there may be a calculus or tumour blocking the neck of the bladder, paralysis of the bladder from diseased or injured spinal cord or brain, or reflex spasm after operations about the perineum. Hydronephrosis commonly results.

The *Treatment* is mainly surgical. Before undertaking any operation the blood urea (§ 389) should be estimated. If this is high, over 75 mgm. to 100 c.c. of blood, there is interference with the kidney function and operation is dangerous to life; drainage of the bladder improves the condition and operation may be safe later on. In cases of spasm a hot bath or hot fomentations to the abdomen give relief. Hysterical and other nervous affections are referred to elsewhere. Atony and simple vesical paralysis may be treated by an injection of carbacholum B.P. (doryl) or a mixture containing nux vomica and belladonna, or by the constant current, one pole being placed on the perineum and the other just above the pubis.

The patient complains that he has **not passed any water** *for some time, but there are* NO EVIDENCES *of a* DISTENDED BLADDER, *and on passing a catheter it is found to be empty, or nearly so.* The condition is SUPPRESSION OF URINE.

§ **421. Suppression of Urine** (Syn., Anuria) is a very grave condition. A catheter should always be passed before the diagnosis of suppression is made. There are two kinds: I. OBSTRUCTIVE suppression, which is due to some obstruction to the flow of urine through the ureters; and II. NON-OBSTRUCTIVE suppression, which is due to the non-secretion of urine by the kidneys. The latter form is sometimes spoken of as true suppression.

I. OBSTRUCTIVE SUPPRESSION is due to blocking of both ureters (the kidneys being healthy) by (i.) renal calculi; (ii.) a renal calculus blocking one ureter may cause reflex suppression in the other kidney; (iii.) blocking of both ureters by solphonamide crystals (especially after sulphathiazole and sulphadiazine); (iv.) tumour at the base of the bladder; (v.) congenital malformation of the ureters. When only one ureter is completely blocked, the urine that passes is clear, of low specific gravity, and non-albuminous; but, provided the other kidney is healthy, there is no renal inadequacy, the healthy kidney undergoing compensatory hypertrophy (see also Hydronephrosis, § 424). When both ureters are blocked, a condition known as "*latent uræmia*" arises. The *Symptoms* are: the patient passes no urine for about a week, and may complain of nothing except slight drowsiness, but after eight or ten days he becomes restless, with contracted pupils, subnormal temperature, dry brown tongue, and muscular twitchings. In other cases vomiting may be so severe as to suggest the presence of intestinal obstruction. Death is usually sudden, after ten to fourteen days, the mind remaining clear to the end.

II. The causes of NON-OBSTRUCTIVE SUPPRESSION are: (i.) Acute nephritis, or the terminal stage of chronic nephritis (ten to twenty hours before death); (ii.) the anuric form of diabetic coma (§ 416); (iii.) collapse and shock (of which suppression is

one of the symptoms)—*e.g.*, after abdominal operations or injuries, severe burns, severe diarrhœa and vomiting, fevers, local inflammations or any cause of sudden fall of blood pressure; (iv.) acute poisoning with phenol, lead, phosphorus, turpentine, or with certain sulphonamide drugs; (v.) embolism or thrombosis of both renal arteries (very rare); (vi.) incompatible blood transfusions (§ 537); (vii.) after passage of a catheter, cystoscopy, pyelography or other instrumentation; (viii.) crush injuries. Whichever of these causes is in operation, the *Symptoms* are: (1) any urine passed is highly-coloured and concentrated (high specific gravity), and may contain albumen and casts (indicating that the suppression is due to renal disease); (2) there may be urgent vomiting, diarrhœa, and sweating. The other symptoms are those of acute uræmia (§ 372) and those of the cause.

CRUSH INJURIES follow severe crushing of a limb under débris. The urinary output due to the initial shock falls further, with marked albuminuria and dark brown granular casts. Complete suppression often follows, with incessant vomiting and thirst; death frequently occurs on the 7th–8th days. *The cause* may be due to a reflex from the injured limb causing the blood flow in the kidneys to by-pass the glomeruli ("renal shunt").

Prognosis of Suppression.—Suppression is a very serious condition, though the gravity depends somewhat upon the cause. Of the *obstructive* forms, calculus blocking one ureter, the kidney of the opposite side being healthy, is perhaps the most favourable. If the obstruction affects both ureters and is not removed, death will occur in about eleven days after the obstruction began. In the *non-obstructive* forms death or partial recovery takes place in a few days.

Treatment.—Hot air baths, hot packs, and other diaphoretics promote the action of the skin, and so relieve toxæmia. In acute *non-obstructive suppression,* fluids must be given freely by mouth: or as 5 per cent. dextrose into a vein. To the dextrose may be added ½–1 pint of sodium sulphate (4 per cent.) or a 50 per cent. solution of sucrose (1 c.c. per lb. body weight). Alkalies are of value when the blood alkali reserve is low and especially for cases following sulphonamides or blood transfusion. Free purgation promotes the excretion by another channel; cupping, wet or dry, over the loins relieves the local congestion. Good results have been obtained from blocking the sympathetic vaso-constrictor fibres to the kidneys with either a spinal anæsthetic, or a bilateral paravertebral block with procaine. Decapsulation of the kidneys may relieve, for the kidneys are often in a state of "cloudy swelling," and when given space to expand recover their function. When a sulphonamide drug is causal, lavage through a ureteric catheter by 2·5 per cent. sodium bicarbonate will remove crystals from the ureters: otherwise bilateral nephrostomy may be required.

For the treatment of *obstructive suppression* a surgeon should be called at once.

The patient complains that his urine **dribbles away constantly,** *and on percussing over the pubes or passing a catheter, his bladder is found to be empty. He has* TRUE INCONTINENCE. *If there is* INCREASED FREQUENCY *he has a frequent call to urination, and cannot always hold his water.*

§ 422. **Incontinence of Urine** may be either TRUE INCONTINENCE or INCREASED FREQUENCY.

(*a*) TRUE INCONTINENCE, when the urine dribbles away involuntarily as fast as it is formed, must not be confused with *overflow* or *false incontinence,* which is due to the overflow of a distended bladder in *retention.* The latter is recognised by the signs of a full bladder and by the relief afforded by the passage of a catheter. In true incontinence, which is relatively a rarer condition, the *Cause* is generally quite apparent, such as vesico-vaginal fistula, paralysis and dilatation of the sphincter after

the operation of lithotrity, or the paralysis of the sphincter associated with various cerebro-spinal affections (§ 690).

(*b*) INCREASED FREQUENCY OF MICTURITION is a very common complaint. The patient can hold his water, but the calls to urinate are too frequent, and sometimes so urgent that a few drops dribble away before arrangements can be made. "Stress incontinence" indicates that any sudden strain, *e.g.*, emotion, laughing, crying, coughing, will cause dribbling. The normal time during which the urine can be retained varies in different individuals, and also according to the amount of fluid taken; but four or five hours is a fair average. It is longer in the female than the male; some women can retain the urine for ten or twelve hours. The habit is injurious, and is said to lead to abnormal flexions of the uterus.

Increased frequency is due to many *Causes*. The first point to determine is whether there is any marked increase in the diurnal quantity, as in diabetes mellitus, diabetes insipidus or chronic nephritis, because any of the causes of polyuria (§ 414) may be a cause of increased frequency of micturition. In young adults diabetes is the commonest, but in advancing years chronic nephritis and enlarged prostate are by far the most common causes. Our attention is often first drawn to the latter condition because the patient develops a habit of rising at night to pass water. It is not always easy to decide whether the quantity is increased or not, as the patient is apt to think that, because he passes water too often, he passes too much: it may be necessary to measure the diurnal volume of urine. There remains three groups of causes of increased frequency to consider: 1. Some cause of *local irritation* is undoubtedly the most frequent. The *urine* may be too acid. *Bacilluria* may for long cause no symptom except increased frequency of micturition; this is a common symptom in coli bacilluria. The *bladder* may be irritable, owing to an enlarged prostate (the usual cause of abnormal frequency in old age), chronic cystitis, ulceration, tumour, stone, oxaluria, or pressure upon the viscus by a displaced or enlarged uterus. Or the irritation may be in the *kidneys* from the presence of stone, tubercle, or other cause of pyelitis (§ 412). Or the irritation may be *reflex*, from disease in the vicinity of the bladder, worms, phimosis, fissure, piles, prolapse or polypus of the rectum, vascular urethral caruncle (a cause frequently overlooked in women), pelvic inflammation, or varicocele. 2. *Constitutional* causes are occasionally associated with this condition, such as hysteria, sexual excesses and possibly adenoid vegetations in the pharynx. 3. The *sphincter* may be incompetent, especially with cystocele. And see § 456. *A congenital* want of development of the sphincter is sometimes present. True congenital cases are rare, and defective action of the sphincter is more frequently due, especially in women and children, to some of the reflex causes above mentioned, the habit persisting after the cause has been removed.

NOCTURNAL INCONTINENCE (enuresis) in children is a troublesome and frequent condition; if untreated it may persist into adult life.

Usually the child has gained proper control of the urine by the age of 2–2½ years, and nocturnal incontinence shows itself later. When complete continence has never been attained, lesions such as spina bifida, a congenital valve in or imperfect development of the urethra must be looked for. In all cases it is important to exclude lesions such as stone in the bladder, cystitis or pyelitis, renal tuberculosis or polyuria with chronic nephritis. Reflex causes such as threadworms, a local vulvitis, and, according to some, phimosis or nasopharyngeal adenoids may be contributory. Having excluded organic diseases, the children having nocturnal incontinence come usually under three types: (i.) In the largest group the condition is the result of an anxiety neurosis: such children are intensely worried about their trouble, are made worse by punishment or the jibes of their brothers and sisters, and so long as their parents continue to regard the condition as a fault, it remains incurable. (ii.) In a small proportion, carelessness and laziness of habit is causal. These children are usually obese and mentally sluggish. (iii.) In a few, mental deficiency is present, making training in their earlier years impossible. In such, diurnal incontinence of urine, and often of fæces, results.

Both *Prognosis* and *Treatment* turn almost entirely upon the cause, and are hopeful in proportion as this is removable. The power of retention of the urine is a habit which can be cultivated in early life, and the relative frequency in different individuals varies with habits engendered in infancy and childhood. Local lesions and reflex causes must be removed when possible. Where there is an anxiety state, it is well to explain the condition to the child, in kindly fashion; stop punishments and scoldings, and adopt a confident attitude that the condition will ultimately be curable. Fluids towards the end of the day should be strictly limited, and the bladder emptied at bedtime. A simple expedient is to let the child keep a calendar which he marks himself, crossing out the nights on which enuresis has occurred; a suitable reward for gradual improvement often works wonders. Most drugs probably act by suggestion, but bromides, belladonna and ephedrine are helpful. Operative measures are not necessary or justified unless organic disease is present. In the sluggish, lazy child, thyroid is useful.

§ **423.** *The urine presents a* **cloudiness,** *due to some* CRYSTALLINE *or* OTHER DEPOSIT; it may be URATES, URIC ACID, PHOSPHATES, OXALATES, or FAT, unless it be pus (§ 410), blood (§ 406), or bacteria (§ 392).

With excess of URATES *the urine,* CLEAR *when first passed, becomes cloudy, with a pinkish* AMORPHOUS DEPOSIT *when it gets cold; the deposit dissolving again when heated in a tube.* This condition is still believed by many to be due to functional derangement of the liver. Various other conditions with which excess of urates and uric acid in the urine may be associated, as a more or less subordinate symptom, have already been referred to in § 393.

The clinical significance of uric acid and urates is still a subject of debate. The deposit may be physiological when occurring after a heavy meal or undue exercise.

In *Multiple Myeloma* the urine may be cloudy on standing or even passing, due to the presence of the Bence-Jones protein (§§ 386, 598. X).

Phosphaturia *is usually indicated by cloudiness in a neutral or alkaline urine* (§§ 388 and 393). It signifies decreased acidity of the urine rather than increased excretion of phosphates. (1) Phosphates frequently occur in the urine in such quantity as to cause a turbidity even when *first passed.* They appear especially towards the end of micturition and may alarm the patient unnecessarily. Phosphates may be especially abundant in the "alkaline tide" of the early morning or after dinner, and may cause an iridescent "scum" on the surface of the water. There may be no symptoms, even when phosphates are passed in large quantities; but more frequently phosphaturia is accompanied by chronic dyspepsia. Phosphaturia may occur with any cause of (1) wasting or with (2) depression and anxiety, when it is probably due to defective acid formation and lowered metabolism. Phosphates in *excess* occur with (3) hyperchlorhydria, (4) wasting disease and (5) after a diet rich in fruit and vegetable. Phosphates are *diminished* in pregnancy and in convalescence after fevers. A deposit of triple phosphates in freshly passed urine indicates decomposition in the bladder.

The *treatment* is based on the cause. Usually the condition responds to measures designed to keep the urine acid, as with ammonium chloride or with sodium acid phosphate, combined with rest or wise regulation of work and worry. As there is evidence that disorder of the calcium metabolism affects the phosphates, success often follows a diet poor in calcium. Therefore milk, eggs, fish and fruit are cut out and potato and other foods poor in calcium content are given freely.

Oxaluria *is generally indicated by a "powdered wig" deposit on the top of the mucus which settles at the bottom* (§ 393). Transient oxaluria has no clinical significance except as indicating the *nature* of a stone, which has revealed its *presence* by other symptoms. It is also found after a diet of rhubarb, sorrel, spinach, tea and coffee, or cocoa. But oxaluria is also connected with other clinical conditions. (1) Cases have been recorded where rapid emaciation and pains in the loins and back were attended by an excess of oxalates in the urine. (2) Pancreatic disease: they are said to be abundant in the early stages of chronic pancreatitis. (3) Other observers have connected certain nervous symptoms, such as mental depression; it is probable that these symptoms are connected with the concurrent dyspepsia and pains. (4) Oxaluria is associated with abnormal fermentation of sugar in the intestine. Urates are generally precipitated in the urine at the same time as the oxalates. (5) Oxalates are found in large excess in paroxysmal hæmoglobinuria (§ 409) and their presence may cause hæmaturia and albuminuria.

Treatment consists in avoiding foods which contain oxalates and those which allow excessive carbohydrate fermentation in the intestine. See Diet (§ 297. XV). The formation of crystals is prevented by the ingestion of magnesia. Calculi of oxalates are reduced by rendering the urine strongly acid with acid sodium phosphate or ammonium chloride.

Fat may occur in the urine in subacute parenchymatous nephritis attended by much fatty degeneration of the epithelium, and after fractures of the bones. It is found in great abundance in **Chyluria.** The presence of chyle in the urine gives a milky white appearance and the power of coagulating. In the tropics chyluria is due to the filaria sanguinis hominis producing obstruction of the thoracic duct; in this country enlarged glands or new growths are the principal causes. The back pressure on the lymphatic vessels of the kidneys and bladder causes some of them to rupture into the urinary tract. The urine passed at night is the more completely white; that passed by day may be mixed with blood. Embryos are to be found in the urine with a few red and white blood-cells, albumen, fat, and shreds of fibrin. Chyluria may follow trauma, and may accompany leukæmia in rare cases.

Prognosis.—The patient may live twenty years with but little impairment of health. In other cases, however, great debility and mental depression may be present.

Treatment.—Prevent the disease by boiling the drinking-water. To meet the loss of weight give plenty of nourishing food.

In *Pseudo-Chyluria* the milky appearance of the urine is due to the presence of the same material that occurs in pseudo-chylous ascites.

§ 424. **Renal Tumours** may be of six kinds: (I.) HYDRONEPHROSIS; (II.) PYONEPHROSIS; (III.) PERINEPHRIC ABSCESS; (IV.) MALIGNANT DISEASE; (V.) POLYCYSTIC DISEASE; and (VI.) MOVABLE KIDNEY. The last-named is described under Abdominal Pain (§ 253), which is the symptom for which advice is sought. Extravasation of blood after injury to the kidney may simulate a tumour.

The *Physical Signs* common to all tumours of the kidney, and their diagnosis from other ABDOMINAL TUMOURS are given in §§ 263 and 394.

I. **Hydronephrosis** is a term indicating a cystic tumour of the kidney, caused by the gradual or intermittent obstruction of the urinary passages, and the consequent dilatation of the pelvis of the kidney. It is always present with normal pregnancy.

The *Symptoms* by which this tumour is recognised are: (i.) Intermittent attacks of renal pain, often with vomiting. (2) If large, a renal tumour develops. (3) Local pressure symptoms may arise, causing pain or disturbance of function of the neighbouring organs. (4) Constitutional and general symptoms are absent, unless the stagnant urine becomes infected. (5) It may be discovered on investigating for the cause of pyelitis, the condition having been unrecognised previously.

Etiology.—The causes of obstruction to the outflow of the urine may be (i) *congenital* (narrowed ureters, aberrant renal vessels, a valve in the urethra); (ii.) *acquired* causes, which may occur (*a*) in the *urethra*, such as stricture or enlarged prostate; (*b*) in the *ureter*, such as occur from stone or blood-clot; pressure by pelvic or other tumours; contraction after operation, injury, or disease of the ureter; kinking, as in movable kidney (often associated with aberrant renal vessels). These acquired causes give rise to a *gradual obstruction* (Fig. 105), and when the obstruction is intermittent the tumour may become very large, when it is liable to be mistaken for an ovarian cyst, or even for ascites. In such cases a trocar introduced at operation will reveal fluid free of the albumen which is always present in an ascitic fluid (Table LX). *Complete obstruction* of a ureter causes atrophy of the kidney, not hydronephrosis.

Prognosis.—If the condition is unilateral and intermittent it may cause little trouble, and may disappear after a duration of years. On the other hand, a double hydronephrosis is very serious, as it leads to uræmia. A surgeon should be consulted early. The complications are rupture into the peritoneum or pleura; suppuration in the pelvis of the kidney (pyonephrosis); or uræmia, due to atrophy of the substance of both kidneys.

Treatment.—In all cases the cause must be ascertained and, if possible, treated. Osler recommended the use of a pad to retain the organ in place. Surgical treatment is usually advisable.

II. **Pyonephrosis** is a cystic tumour of the kidney due to distension of the pelvis and calyces by fluid containing pus. It is consequent on obstruction to the free outlet of the urine in septic cases of pyelitis, or sepsis supervening on hydronephrosis.

The *Symptoms* are: (1) The tumour is tender to palpation; (2) symptoms of pyelitis are present—pyuria, intermittent pyrexia, sometimes rigors, a toxic appearance, and dull pain in the loin; (3) at intervals, when the obstruction is removed or diminished, the tumour may subside, coincident with the passage of a large quantity of pus in the urine.

The *Causes* are: (1) *pyelitis* (§ 412), with blocking, partial or complete, of the ureter; or (2) *hydronephrosis* (*vide* Causes of this above) becoming septic—*e.g.*, from extension upwards of cystitis.

Diagnosis.—(1) From *hydronephrosis*, which has no tenderness or fever; (2) from *perinephric abscess*, which has greater tenderness in the loin and a more superficial swelling, with local signs of abscess sooner or later.

Prognosis.—The condition is serious. A tuberculous pyonephrosis may undergo cure by fibrosis. The structure of the kidney is largely destroyed, and in bilateral cases, uræmia will result. A fatal issue is rapidly brought about by the tumour bursting into the abdomen or chest.

Treatment is surgical, and nephrectomy is usually indicated. Lavage through a ureteric catheter may temporarily relieve.

III. Perinephric Abscess is fairly common. It may arise by (i.) a blood-stream infection often associated with boils; (ii.) extension from kidney disease (pyelitis, pyonephrosis or tuberculosis); (iii.) extension from a perityphlitic abscess; (iv.) extension from other organs—*e.g.*, abscess of the liver, empyema or spinal caries; (v.) after an injury. The *Symptoms* are: (1) dull, aching pain in the loin, sometimes radiating down the leg; (2) deep-seated resistance of the erector spinæ, tenderness on pressure in the post-renal angle, or in the hypochondrium in front; (3) the temperature is continuous, or pyæmic in acute cases with sudden onset, or intermittent in insidious cases; (4) the leg on the same side is kept flexed and the patient stoops when walking; (5) swelling, with œdema of the skin, which appears late in the disorder, is felt between the iliac crest and the last rib, and it may be fluctuant; (6) the urine may or may not be altered according to the cause, but traces of albumen are common; (7) marked leucocytosis; (8) collapse of the base of the lung and sometimes a small pleural effusion. The *Diagnosis* is difficult in the early stage when pain alone is present, when it may readily be mistaken for *lumbago, appendicitis* or *spinal disease*, but there is no fever in the first of these. Later it may be mistaken for a *renal tumour*, but in a simple tumour fever is absent, and the leg would not be held constantly flexed; the aspirating needle may be used. In *pyonephrosis* there is not such acute pain or tenderness. *Prognosis.*—The abscess tends to open or to burrow its way in various directions, into the alimentary or urinary canals, peritoneum, or pleura. It may point in the lumbar region or various other directions, and burrow for a considerable distance. *Treatment.*—In the early stages, before the diagnosis can be certain, give penicillin, hot fomentations and opium for the pain; as soon as pus is recognised operative procedure is necessary.

IV. Malignant Disease starting in the Kidney is a rare condition. It affects children under nine (in whom *sarcoma* chiefly occurs), and adults over forty (in whom usually it is *carcinoma*), there being a remarkable immunity between these age periods.[1] Renal sarcoma is the commonest abdominal growth in children (Wilms' tumour). It is met in the first five years of life and is believed often to start before birth. After a period of immunity, malignant disease is found again in people between fifty and sixty. *Hypernephroma* is the commonest form of carcinoma in adults. It may lie latent for years and then assume great malignancy: metastases occur in the opposite kidney, grow along the renal veins and produce early deposits in bones.

The *Symptoms* are: (1) The tumour is rapidly growing, usually of firm consistence, but if of very rapid growth it may appear fluctuating; (2) hæmaturia, frequent, inter-

[1] The solid tumours affecting the kidney consist of (*A*) *Connective tissue type*:—I. Simple or benign growths (fibroma, lipoma, angioma); II. Sarcoma, which is by far the commonest . (*B*) Growths of an *epithelialt ype*:—I. Adenomatous growths (simple adenoma, trabecular, and papilliform cystomata). II. True Carcinoma:—(1) glandular type; (2) malignant papilloma. (*C*) Hypernephroma.

mittent, and of moderate amount; (3) progressive emaciation; (4) the pain is variable, sometimes it is very severe, owing to pressure upon or infiltration of the neighbouring organs. Sometimes pain is entirely absent, and the tumour may have attained a very large size before any symptoms occur; (4) in left-sided hypernephroma left varicocele occurs, and is a valuable early diagnostic sign. (5) In hypernephroma, a spontaneous fracture of bone or an unexplained pyrexia may be the first symptom.

Diagnosis.—When a tumour occurs in a movable kidney it is apt to be mistaken for *ovarian tumour* or *fibroid*, and vaginal examination is necessary (see § 263 for diagnostic points). *Tuberculous* kidney in a child may present difficulty, but the pain is less, and pyuria is present rather than hæmaturia. *Pyonephrosis* is accompanied by fever, the swelling is fluctuant, and there is a history of pyuria. *Retroperitoneal* and *renal sarcoma* are the chief causes of enormous abdominal tumours in children. The diagnosis of malignant tumours is not usually difficult.

The *Prognosis* is very grave. If untreated, death occurs in six to twelve months after detection of the growth, the cancer of adults being of somewhat slower growth.

Treatment is usually too late; early excision gives the only chance of life.

V. Polycystic Disease of the Kidneys is a rare condition, usually of congenital origin and often familial, in which both kidneys contain cysts of varying size and number.

Symptoms.—(1) There is complaint of a dull dragging pain in one or both loins. (2) With this there is a tumour in one or both loins, but usually larger on one side; the surface is irregular and feels cystic, although the kidneys feel very firm otherwise. (3) The other symptoms are those of chronic interstitial nephritis (§ 399), the urine is abundant, pale, of low specific gravity, containing traces of albumen, and occasionally blood and casts. The heart becomes hypertrophied, and the pulse indicates high blood pressure. (4) Polycystic disease may co-exist in the liver, spleen, ovaries and pancreas. The patient may have excellent health for many years, or may develop symptoms of chronic uræmia. It may give rise to an enormous tumour in the fœtus and obstruct delivery. In children, symptoms may be associated with renal rickets.

The *Diagnosis* may be difficult. When symptoms of granular kidney occur, together with a tumour in both renal regions, the condition may be diagnosed as polycystic kidney. The tumours have to be diagnosed from other abdominal tumours (§ 263). Pyelography reveals a large kidney with elongated calyces (Fig. 104).

Etiology.—The disease is usually familial. In the majority, the patients are middle-aged.

Prognosis.—The younger the patient the worse the prognosis. In those diagnosed in middle age, it is common for them to survive 20–30 years.

Treatment is similar to that of nephritis. Death may occur from uræmia or the same complications as those of interstitial nephritis. Operation must not be performed as the condition is bilateral. A surgical support may be of value when the weight of the tumour is producing symptoms.

Hydatid cyst may occur in the kidney, and may be difficult to differentiate from other cysts unless it opens into the pelvis of the organ, when the characteristic hooklets (Fig. 85) are found in the urine. The passage of vesicles may cause renal colic. The condition may be suspected if (i.) the tumour has the "hydatid thrill" on palpation; (ii.) there is evidence of cysts elsewhere; and (iii.) there is a history of residence in infected countries. (iv.) Eosinophilia may be present. The complement fixation test and the Casoni reaction aid diagnosis (§ 347).

The *Prognosis* is generally not grave. The cyst may last for years with no symptoms, or it may burst into the pelvis of the kidney. It may open into the stomach or bowel, with temporary recovery; or into the chest, which is a serious complication. It may become very large and give rise to pressure signs.

Treatment is surgical.

CHAPTER XIV

DISEASES OF THE FEMALE REPRODUCTIVE ORGANS

DERANGEMENTS and diseases of the genito-urinary organs have a widespread influence upon the physiological welfare of the individual as a whole. General diseases also have a marked influence upon the reproductive functions. The physician, therefore, must take into consideration the reproductive system when investigating the condition of the patient and especially in cases where there is evidence of endocrine imbalance. In a volume on Clinical Medicine it is essential to give some consideration to the methods of investigation and the general treatment of complications of the pelvic organs of women. The modern gynæcologist endeavours to restore reproductive function as much as possible rather than to destroy it by radical surgical treatment; therefore it is unnecessary to go into details of treatment of conditions in which surgical aid is essential.

§ 432. Recent research on ovarian and pituitary hormones has thrown much light upon reproductive function and the causation of various gynæcological conditions. Before discussing the diseases affecting the pelvic organs, it is therefore advisable to give a brief account of the various hormones secreted by the ovary and the pituitary body and their influence upon reproduction.

Before the onset of *puberty*, the ovary contains primordial follicles whose secretion is associated to some extent with the early development of the genital organs. At puberty marked changes take place in the ovary; the Graäfian follicles develop, and secrete œstrogens from the follicle cells. This secretion causes proliferative changes in the uterine mucosa, with increased growth and activity of the reproductive organs and development of the secondary sex characteristics. As the follicle develops and ripens, extrusion of the oöcyte takes place, followed by the formation of the corpus luteum, which secretes a hormone known as progesterone. When there is defective secretion of œstrin, the development of the genital organs is arrested, the secondary sex characteristics fail to appear, menstruation does not occur and sterility results. If œstrin is injected, growth and activity in the genital organs occur to some extent.

Atrophic changes occur in the uterus and other genital organs when *oöphorectomy* is performed in adults. These changes can be partly arrested, or if they have already taken place, a certain degree of proliferation and vascularity of the uterine mucosa may be restored, by implanting ovarian grafts or by giving injections of œstrin. If œstrin is injected into castrated rats and mice, desquamative changes take place in the superficial cells of the vagina, similar to those which occur during œstrus in the normal animal. Œstrin also causes a certain degree of mammary activity. It arrests the reduction of the chromatophilic cells in the cervical ganglion which appear after castration, and also of the granular cells in the anterior lobe of the pituitary.

Menopausal changes occur as a result of diminishing œstrin secretion. Symptoms arising from these changes can be relieved by giving follicular hormone preparations such as stilbœstrol, alone, or in combination with other glandular extracts.

Menstruation.—At puberty menstruation begins. The menstrual cycle has two phases: (i.) the proliferative phase, when the action of œstrogen is marked; it prepares the mucosa for the reception of the fertilised ovum and lasts from the end of menstruation till the period of ovulation. (ii.) The secretory phase, where the action of

progesterone (the corpus luteum secretion) overcomes that of œstrogen but is dependent on its sensitising action. During the first 14 days of the intermenstrual cycle, there is increased secretion of œstrogen, causing proliferation and vascularisation of the uterine mucosa. These changes are in the nature of a preparation for the fertilised ovum and resemble the decidual formation which takes place between the 14th and 16th day of the intermenstrual cycle; the extrusion of the oöcyte is followed by the formation of the corpus luteum.

The follicular hormone is secreted throughout the whole of the menstrual cycle. The amount of it which is found in the urine during the proliferative phase gives some indication of the time at which ovulation takes place. Its action is inhibited by the secretion from the corpus luteum. If fertilisation does not take place, the superficial layers of the mucosa are cast off, hæmorrhage occurs, and a fresh menstrual cycle sets in; and in the absence of fertilisation, the corpus luteum degenerates and becomes scar tissue. If fertilisation does occur, the corpus luteum develops further and its secretion—progesterone—is concerned with the maintenance of the ovum in the uterus. It is the guardian of the ovum during the earlier stages of pregnancy at least, presiding over its destiny, together with the pituitary hormones. It inhibits uterine contractions and ovulation during pregnancy and prevents abortion. The administration of corpus luteum hormone prevents the occurrence of abortion in many cases. When examining specimens of the uterine mucosa after curettage it is necessary to remember the exact date of the menstrual cycle when the operation took place; otherwise physiological proliferative or degenerative changes may be looked upon as pathological.

Hormone Therapy has made rapid progress, and its value in gynæcological practice is recognised; its use will lessen to some extent the necessity for radical operations. There is still, however, so much confusion regarding its application that further research work must be carried out to stabilise medical opinion as to its merit in suitable cases. We are still ignorant of the various relationships between the endocrines; we have no exact knowledge of the stimulating and the controlling influences which one gland exercises upon another. Research upon the relationship between the anterior pituitary secretions and the gonads has produced a large number of extracts which are widely used, but further research and experience are required before we can have definite indications for the administration of hormone substances.

The standardisation of hormones has enabled the gynæcologist to prescribe exact doses of substances whose activity is maintained at a known level. The crystallisation of the hormones of the ovary is a recent and valuable advance.

The *follicular hormone* of the ovary can be administered orally, by injections, and also by means of ointments and vaginal suppositories. It is known as œstradiol by international workers: stilbœstrol, hexœstrol and dienœstrol, in tablet form by mouth, are replacing many of the previous injections. The *lutein substance* of the ovary, however, is usually given by injection, as it is more satisfactory than by mouth.

Menstrual Abnormalities.—*Defective œstrogen secretion* at puberty leads to imperfect development of the uterus, delayed onset, and scanty, irregular periods. Spasmodic dysmenorrhœa may be due to defective uterine development, or to defective follicular hormone secretion. Premature menopausal changes may take place when the œstrogen secretion is defective. Administration of œstrogen may remedy this.

Excessive œstrogen secretion leads to marked proliferation of the uterine mucosa and increased number of the follicles. Excessive menstrual loss alternating with periods of amenorrhœa may occur in young women; in these cases œstrogen is found in the urine. The *corpus luteum secretion* is defective in this type of young patient; in older women retention cysts are often present in the ovaries—the condition is known as *Metropathia Hæmorrhagica*. Treatment consists in the administration of progesterone alone or in combination with extracts of the anterior lobe of the pituitary; a daily intramuscular injection of progesterone is given for seven days before the onset of the expected period, the object being to counteract the over-action

of œstrogen. Œstrogen is a stimulant to uterine action; progesterone has a sedative action. Should such treatment fail, it may be necessary to curette the thickened endometrium or apply radium 50 mgm. for 22 hours to the interior of the uterus. Radium reaction may cause hæmorrhage for some days or longer after its application, but the ultimate effect is satisfactory; the small dose does not interfere with menstrual function or reproduction. In older women it may be necessary to excise the cystic portions of the ovaries before cure is effected. In some cases there may be granulosa cell formation in the ovaries. Hysterectomy should not be advised.

The *pituitary* is closely associated with ovarian function. The anterior and posterior lobes differ in physiological action. (1) The secretions of the *anterior lobe* are concerned with the reproductive function; they control ovarian activity and are concerned with uterine changes and embedding of the ovum. They do not act directly on the genital organs but through the medium of the ovary. Two known hormones have been isolated—Prolan A stimulates the production of œstrogen, and Prolan B the formation of the corpus luteum and secretion of progesterone. In pregnancy Zondek and Aschheim found that pituitary hormones are present in the urine and if the urine is injected into an immature mouse, corpus luteum and hæmorrhagic changes take place. This constitutes the most reliable test for early pregnancy. Positive results are also obtained in cases of hydatidiform moles and teratomata. (2) The *posterior lobe* of the pituitary secretes a hormone which influences uterine contractions. This secretion has been divided into two forms: (*a*) one which influences uterine contractions only and (*b*) one which causes a rise in blood pressure. Pituitary secretion has no effect on a uterus after oöphorectomy; its action is also inhibited by the corpus luteum. It is, therefore, more powerful in its action in the proliferative rather than the secretory phase of the menstrual cycle. Pituitary secretion influences abortion, but its action is more marked in late pregnancy, where it sensitises the uterus for the onset of labour.

The *Thyroid* and *Parathyroids* are related to reproductive function, inasmuch as some believe that they are concerned with calcium metabolism and partly counteract the influence of œstrogen, which stimulates the excretion of calcium. A combination of corpus luteum and of thyroid extract is useful in the prevention of abortion. Parathyroid extract gr. 1/10 is of benefit in some cases of dysmenorrhœa.

PART A. SYMPTOMATOLOGY

§ 433. Diseases of the pelvic organs have both Local and General symptoms. Local Symptoms are: Irritation or swelling around the vaginal orifice, vaginal discharge, including leucorrhœa, painful menstruation (dysmenorrhœa), excessive menstruation (menorrhagia), deficient menstruation (amenorrhœa), pain in the region of the organs, acute and chronic; backache, various disorders of function (such as dysuria and dyspareunia), and tumours or swellings.

General Symptoms consist of: (1) Malaise and general ill-health. The condition of chronic invalidism caused by pelvic maladies may be altogether out of proportion to the amount of local trouble. Most often such chronic ill-health dates from pregnancy or child-birth. (2) Disorders of the abdominal or pelvic viscera often cause symptoms of dyspepsia. (3) Anæmia, due to excessive hæmorrhage, lack of fresh air and exercise from confinement indoors, or from the toxæmia which occurs with degenerating tumours or as result of the complications of pregnancy. (4) Various neuralgiæ and a general hypersensitiveness of the nervous system follow derangements of the reproductive function.

Case-taking in diseases of women differs somewhat from that given in Chapter I. The following summary will form a guide to the principal questions to be answered as a matter of routine:

1. What is the leading symptom complained of by the patient?
2. History—name, age, married or single.

(*a*) If married, how long? How many children and ages of each? Character of confinements, easy or difficult? Any complications after childbirth? Any miscarriages?

(*b*) Menstruation—age at which it commenced? (1) Regular? How often does it occur? How many days does it last? (2) Is it profuse or does the flow contain clots? (3) Is pain present, before the onset or during the period? Has pain always been present? If not, when did it begin? Where is the pain? In back, legs, or in one or other side of lower abdomen? What relation has pain to the flow? Is it continuous?

(*c*) Is there any intermenstrual discharge—duration, quantity, white, yellow, clear, thin or thick? offensive? with blood?

(*d*) Micturition—painful, frequent during day or night? Condition of bowels, regular? Are purgatives taken as a rule? Is there pain on defæcation?

(*e*) Other physiological systems must be enquired into, and whether the general health has suffered.

PART B. PHYSICAL EXAMINATION

§ **434.** An abdominal examination should be a matter of routine in all gynæcological cases.

(*a*) *An External Examination* of the abdomen by inspection, palpation, percussion and auscultation (§ 240). For a thorough examination of the pelvic organs the patient should lie on her back with knees flexed and shoulders raised; this relaxes the abdominal muscles. The degree of rigidity or contraction of the abdominal muscles can be ascertained. Is rigidity due to the cold hands of the examiner or to clumsy methods of palpation? Or is it a guard to prevent the examining hand from touching a deep-seated lesion? Is the rigidity due to nervousness, and will it pass off when the patient is more at ease? Place the warmed hands, cup-shaped, on the abdominal wall, very lightly at first. If the patient is encouraged to talk, her attention will be diverted from tender areas which may be more or less due to a condition of hypersensitiveness. If the normal areas are palpated first, the painful regions may be found to be less resistant as the examination proceeds. If there is tenderness in a particular area, is it local or referred from a deep organ? If so, the pathological lesion may be in the intestine or the posterior parietal peritoneum may be involved.

(*b*) *Pelvic Examination.*—There need be no unnecessary exposure of the patient during the vulvo-vaginal examination. A light blanket or a sheet is thrown over the knees and lower abdomen. This examination should only be undertaken in the case of married women or women who have borne children. Virgins should only be examined in exceptional cases or under an anæsthetic; if, however, a pelvic examination is neces-

sary, the rectal route should be employed, the patient lying on her left side. In nervous women this examination is of little value owing to lack of relaxation. To perform a *vaginal examination* two fingers of one or other hand are covered with a rubber glove or finger stall, lubricated with liquid soap or glycerin jelly, and gently introduced into the vaginal opening, care being taken not to touch with the thumb the sensitive anterior portion which includes the clitoris. The skin should not be soiled with the lubricating fluid. If the vaginal entrance is small, the forefinger alone should be inserted. It is useful to be able to examine with either hand. The condition of the *vaginal walls* should be noted —dry or moist, a normal pink or fiery red, as a whole or in patches, atrophic or swollen; the position and condition of the *cervix*, patulous or soft as in pregnancy, conical, firm, granular, scarred or friable. Bleeding on examination should be noted.

(*c*) *The Bimanual Examination* is next made by placing the two fingers in the anterior fornix and palpating the uterus with the external hand pressed firmly above the symphysis pubis. The size, shape, position and mobility of the uterus can be felt between the two hands. The examination may also aid in defining whether a painful area is low down or high up in the pelvic cavity. As a rule the uterus itself is not painful on palpation; pain on examination indicates congestion, adhesions, inflammatory conditions of the peritoneum or ovarian lesions. Palpation of the ovary sometimes gives a sensation of sickness rather than of pain—a valuable aid in localising its position. Tumours, tubal or ovarian swellings can also be felt. The bladder and rectum must be empty.

Difficulties in examination may be overcome by giving an *anæsthetic*. This produces relaxation of the abdomen, so that palpation of the pelvic organs is easy on bimanual examination. As the patient goes under the anæsthetic the last area of rigidity may point to the seat of the lesion. The disadvantage of an anæsthetic is that areas of tenderness are not then ascertained, and deep palpation may thus cause damage, as in cases of ectopic pregnancy or pyosalpinx.

X-ray examinations are useful in localising appendix complications, and reveal the presence of calculus, a calcified fibroid, or bony tumours; a salpingogram (after the injection of iodised oil, B.P.) reveals the condition of the Fallopian tubes. X-ray also shows the position and condition of the spine, the vertebræ and the joints, also any spinal or pelvic deformity: and pregnancy after the sixteenth week.

Instruments employed in the examination of the pelvic organs.

1. *Vaginal Specula.*—The Ferguson speculum is a tube; the bivalve or trivalve consists of two or three limbs jointed together; and the duckbill (Sims). The first is best for the examination of the os; the second for the examination of the walls of the vagina; and the third for operative measures. Note the condition of the mucous membrane, and the character of any discharge. In passing the speculum, do not forget that the vaginal canal is directed backwards and upwards; less pain is produced by quick movements in the right direction than by slow bungling. If it is necessary, apply treatment to the interior by means of a probe covered with cotton-wool; do this before withdrawing the speculum.

2. The *volsellum* is a form of hooked forceps used for drawing down one or other lip of the cervix. It is contra-indicated in those conditions in which the sound is contra-indicated, and in tubal pregnancy. Owing to its lacerating and painful effect upon the cervical tissue it should only be used when the patient is anæsthetised.

3. *The Sound* is rarely used now except when operating or examining under anæsthesia. Undoubtedly harm used to be done by passing it, without a protecting speculum, through a septic vagina into the uterus. Its use is contra-indicated in (1) pregnancy, (2) menstruation, (3) acute inflammation in the pelvis, (4) cancer, and (5) it should never be passed before making a bimanual examination. The uses of the sound are to discover: (1) the depth of the uterus, which is normally 2½ inches, and the thickness of its wall, prior to dilatation and curettage; (2) the position of the uterine cavity, when it is impossible to find it by bimanual examination; (3) the size and state of the os; (4) the presence of tumours in the uterus.

EXAMINATION of a VAGINAL DISCHARGE gives valuable information (see § 437). A glass tube, with a rubber nozzle attached, is used to remove a little discharge, from which a thin film is made. Fix in equal parts of 90 per cent. alcohol and ether, stain with hæmatoxylin, ether and water blue (Papanicolaou). Examine under the microscope. The epithelial cells of the vagina are large, flat and with irregular faint outlines; the nuclei stain deeply. When these cells are numerous there is healthy tissue in the vaginal walls, with information as to the secretion of œstrogen and also the stage of the menstrual cycle. If the epithelial cells are few in number and replaced by small round or oval cells with deeply staining nuclei, the inference is that there is denudation of the superficial or protective epithelium. (1) In the follicular phase the vaginal smear shows leukopenia and cornified squamous epithelial cells, with small pyknotic nuclei; (2) in the post-menopausal or ovari-ectomised case the smear shows many leucocytes, a predominance of non-cornified squamous cells with larger nuclei, or compact cells with large nuclei from the deeper layers of vaginal epithelium. To produce a change from the second to the first stage requires œstrogen daily, in dosage varying with the preparation used. Stilbœstrol in doses of 0·5–1·0 mgm. is commonly given; the effect does not last long.

DILATATION OF THE CERVIX may be performed by the (1) *Slow Method* (seldom employed); tents inserted and left *in situ* for some hours, or by (2) the *Rapid Method*, with Hegar's or Fenton's dilators, vulcanite or metal instruments of graduated sizes. General anæsthesia is necessary. Having inserted the posterior vaginal speculum, fix the anterior lips of the cervix with the volsellum or ring forceps, draw well down, measure the length with a sound, and insert the dilators gradually one after the other until the cervix is dilated. The curette is then used. The nature of any growth present is discovered by a microscopic examination of the scraping; such examination should never be omitted. Dilatation of the cervix is contra-indicated in cases of possible pregnancy, or cancer of the cervix. It should be performed with great caution when the tissues are softened by recent pregnancy.

PART C. DISEASES OF WOMEN, THEIR DIAGNOSIS, PROGNOSIS, AND TREATMENT

§ 435. Routine Procedure and Classification.—Having ascertained the patient's principal or *Leading Symptom*, and the leading facts as to the *History*, according to the scheme given in Part B., proceed, unless the nature of the case is not already apparent, to the *Physical Examination* (subject to the reservations mentioned in Part B.).

The diseases are considered under the various cardinal symptoms to which they give rise—viz.:

§ 436. (A) **Morbid Alterations of the Vulva.**—A few of the common alterations are enumerated here.

VULVITIS in children may be caused by the migration of thread worms, streptococcal and coli infection from the anus, uncleanliness, gonorrhœa, or bad habits. In adults it is generally accompanied by vaginitis (*q.v.*).

PRURITUS AND ECZEMA VULVÆ is sometimes very obstinate. Careful examination should always be made for pediculi or irritating discharges from the uterus, vagina, urethra or from the minute ducts near the vaginal entrance. Diabetes is a rare cause.

A CARUNCLE is a minute red irritable papilloma situated usually just within the urethral orifice, and is a frequent cause of painful micturition, painful sitting, and painful coitus. There is also a painless form. Slight prolapse of the urethra may give rise to a red swelling which may be mistaken for a caruncle, especially in the aged.

LABIAL THROMBOSIS is readily recognised. ABSCESS of the vulva sometimes follows the last named; often it follows inflammation of Bartholin's gland. HERPES is an eruption of a small group of vesicles. They readily rupture, leaving round superficial ulcers which may become infected secondarily.

NOMA, DIPHTHERIA, AGRANULOCYTIC ANGINA, CHANCRES, CONDYLOMATA, INFECTIVE WARTS, ULCERS (simple or malignant) also affect the part. LEUKOPLAKIA and KRAUROSIS are dealt with in § 651.

In the *Treatment* of *vulval* conditions cleanliness is essential, and on the whole the lack of this is one of the most frequent causes of vulvitis. *Caruncle* is treated by diathermy fulguration, cautery or by operation. *Labial thrombosis* requires surgical treatment. The treatment of *pruritus* and *eczema vulvæ* may tax every therapeutic resource. Severe cases should be kept in bed. All scratching is forbidden, and to ensure this, sleeping draughts, even morphia, may be needed during the acute stage. Sedative soothing lotions and pastes of calamin, zinc, bismuth and lead, should be used. Albucid gives relief in some cases, also anethaine ointment and nestosyl. Sitz baths with magnesium sulphate added, suit others. Dettol cream (5 per cent. in lanoline) often gives relief when smeared over the affected area. Fissured and lichenified surfaces may be painted once a week with 4 per cent. argent. nit.. The *cause* may be tracked down and removed, usually a vaginal, cervical or urethral discharge. Dietetic care and open-air exercise are essential. When local treatment fails, tests for allergic causes should be carried out. Electrical methods are most

valuable, especially X-ray and ultra-violet light, but require expert administration. Œstrogen in the form of ointment or vaginal suppositories is of value, especially in older women, provided the local cause is removed.

(*a*) *There is a white,* NON-PURULENT DISCHARGE *from the* VULVAL ORIFICE ; *the condition is* LEUCORRHŒA.

§ **437.** (B) **Leucorrhœa** is a discharge colloquially known as " the whites " ; there is no discharge in health. Leucorrhœa is usually a simple increase of the normal secretion of the genital tract, a non-infective type of discharge, not to be confused with the purulent discharge associated with inflammatory conditions of the vagina, cervix, uterus or tubes.

Diagnosis.—A local investigation is inadvisable in the case of young girls. Of recent years much has been discovered from examination of the discharge alone. The normal vaginal secretion is acid due to lactic acid formed by Döderlein's bacilli: the pH of about 4·4 deters the growth of other micro-organisms. When the discharge gives pH of 5·6 or over, this points to the presence of local infection. A smear shows whether epithelial or pus cells predominate, and the presence and type of micro-organisms may be determined by microscopical examination alone. A thin discharge is usually of vaginal, a tenacious glairy mucus of uterine or cervical origin. A foul smelling discharge, worse after the period, usually indicates *B. coli* and streptococcal, sometimes also staphylococcal infection of the cervix or uterus.

Etiology.—Leucorrhœa is common (1) at puberty, (2) before menstruation, (3) with sexual excitement ; and often accompanies (4) debility, anæmia and (5) local congestion due to undue exertion, constipation, gastro-intestinal disorders and other causes of pelvic venous stagnation. (6) Malnutrition due to deficiency of mineral and vitamin constituents in the food.

Treatment.—Remove any local cause ; improve the health with exercise, fresh air and other general tonic treatment. Douches are usually unnecessary ; the best is lactic acid ♏ 120 to Ōii ; dettol pessaries (5 per cent. in glycerin jelly), freshly prepared lactic acid pessaries, or penicillin compound pessaries are of benefit. Stilbœstrol 1 mgm. daily by mouth may help.

(*b*) *There is a* PURULENT DISCHARGE *which comes from the* VAGINA ; *the condition is* **vaginitis, acute** or **chronic.**

In ACUTE VAGINITIS the discharge is profuse, yellow or greenish, sometimes blood-stained, attended by dysuria and local signs of inflammation. The chief *Causes* of acute vaginitis are: (1) Traumatism, due to pins, peas, and worms in children, or in the adult an irritant pessary, a foreign body (contraceptive appliances, etc.), too strong douches or excessive coitus ; (2) infection with *B. coli*, streptococci, staphylococci, micro-organisms and fungi of various kinds ; (3) gonorrhœa, which is hard to diagnose from other infections of the vagina except by the microscopic examination of the discharge ; (4) extension from adjacent parts, such as the urethra or Bartholin's glands ; (5) a diphtheritic form ; and

(6) agranulocytosis (§ 155). Acute vaginitis of gonorrhœal origin is dangerous because of the liability to extend to the uterus, tubes and peritoneum and to the bladder and kidney.

Treatment.—Rest, hot hip-baths and douches of potassium permanganate (10 grains to the pint), corrosive sublimate or various silver or aniline dye preparations, and after a few days some astringent lotion such as sulphocarbolate of zinc, Tr. iodine (♏60–Ōi), Jeyes' creolin (♏60–Ōi), protargol (4 per cent.) may be swabbed on the wall as the speculum is withdrawn. The oral administration of a sulphonamide is of value in these cases.

Acute Gonorrhœal vaginitis.—*Local treatment:* the entire surface, every crevice and fold of the vagina is swabbed daily and carefully with a mercurial salt, then dried and packed with gauze. The urethra, cervix and ducts are separately dealt with. *General treatment:* chemotherapy is now replacing local treatment, and usually cuts short the infection in a very few days. Sulphathiazole and sulphadiazine are the most effective sulphonamides and are used in doses of G. i t.i.d. for one week. Penicillin, by three-hourly injection, gives better results, but at least 300,000 units in all must be given; this course may have to be repeated. Care must be taken that this does not mask a coincident syphilitic infection.

In Chronic Vaginitis there is a thick, continuous, opaque discharge, usually with local signs of inflammation. The *Causes* are (1) antecedent acute vaginitis; (2) various constitutional conditions, such as general debility, errors of diet, such as excessive protein intake, diabetes, old age, alcoholism, anæmia, and convalescence from fevers; (3) new growths in the vaginal walls, such as epithelioma; (4) irritant foreign bodies and other causes mentioned under Acute Vaginitis.

Trichomonas Vaginitis due to a flagellated protozoon is a common cause. It causes intense pruritus, intertrigo, and acute pain on urination. The vagina is red and tender, the vault filled with a thin, yellow or greyish-yellow purulent fluid, frothy, offensive, often acid in reaction. The origin of the parasite is unknown; many cases are complicated by other infections. Relapse after each period, with a uterine discharge, is common.

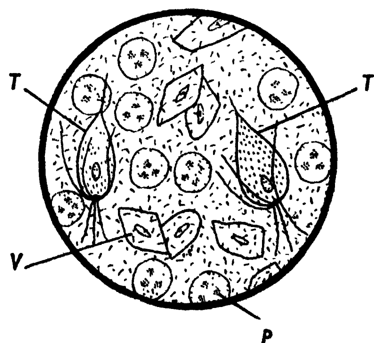


Fig. 109.—A Diagram drawn from a Microscopic Specimen of Vaginal Secretion. It shows Trichomonas Vaginalis Organisms (T) with Flagellæ: also Pus Cells (P) and Vaginal Epithelial Cells (V).

Treatment.—(1) Treat any constitutional disease and remove foreign bodies and new growths. (2) Deal with any primary cause originating in the cervix or body of the uterus. (3) For *trichomonas vaginitis*, insert high into the vagina each night two stovarsol vaginal tablets: after a week they should be used on alternate nights for a further 2–3 weeks. Lest this drug irritates the vulva, apply a wool tampon: and if relapse occurs,

repeat the treatment. (4) For other vaginal discharges, or to prevent infection after vaginal laceration, compound pessaries of penicillin with 0·1 per cent. flavizole give the best results. When considered desirable, local applications through a Ferguson's speculum of liq. iodi mitis or a solution of silver nitrate (5·0 per cent.) may be used. An insufflation of silver picrate powder (1·0 per cent.) each 5-7 days has been most helpful, but must not be used in the later months of pregnancy. Hot sitz baths produce a beneficial effect. Gonorrhœal cases demand special treatment (see Acute Vaginitis). When *vaginitis continually relapses*, seek and treat any source of re-infection in the cervix, urethra or Bartholin's glands.

SENILE VAGINITIS may occur in elderly women and with the menopause. The vaginal lining shows atrophy and red patches; the discharge may be blood-stained, and adhesive bands near the cervix are often found. The glycogen content of the vaginal epithelium is diminished. The cervix and urethra may be involved. Senile vaginitis responds to vaginal pessaries, diathermy or ionisation, and in some cases to œstrogen medication alone.

VULVO-VAGINITIS IN CHILDREN used to require very lengthy treatment. Œstrogen preparations stimulate the formation of glycogen in the vaginal lining and of lactic acid in its secretion; both of which aid nature's defence mechanism and are often curative. For gonorrhœa the sulphonamide group of drugs and penicillin have shortened the course of the infective discharge to a matter of a few days. According to the age of the patient, sulphadiazine ½ G. is given four to six times a day for five days, then three times daily for another five days.

DISCHARGE OF **Uterine Origin** may be due to endocervicitis, endometritis, cancer (see Hæmorrhage), salpingitis, inflammation around the uterus (see Pelvic Pain)—and constitutional causes.

I. In ENDOCERVICITIS, or inflammation of the cervix, the discharge is more or less constant, and usually consists of *glairy material* like white of egg, but it may be muco-purulent. Other symptoms are: (1) The cervix is swollen, and may present retention cysts, but usually on examination with the speculum one sees an "erosion" or catarrhal patch, which may bleed slightly on pressure. (2) When the tissues around the cervix are congested, there is tenderness on palpating the cervix, often menorrhagia, dysmenorrhœa, and backache; (3) general malaise and other signs, as with other septic foci. Cervicitis may have to be *diagnosed* from cancer of the cervix. Here the age is not much guide, as cancer of the cervix may appear in a young patient. Cancer is hard to the touch and is friable, readily breaking down and bleeding when touched, and there is usually a blood-stained discharge. Microscopic examination of scrapings will determine the diagnosis. When fixity of the uterus and cachexia have appeared, the diagnosis is simple. For *Causes* and *Treatment* see below.

II. In ENDOMETRITIS, or hyperplasia of the lining membrane of the body of the uterus, the discharge comes in gushes when the patient rises or walks about. Endometritis is usually accompanied by both menorrhagia and dysmenorrhœa, and general pelvic discomfort and pain. The general health may be poor. Bimanually, the uterus is found to be enlarged; the cervix is often hypertrophied and inflamed. Sometimes there is a history of recurring abortions or of sterility. Endometritis must be

diagnosed from cancer, especially if there is blood in the discharge. Owing to the risk of delay, in doubtful cases curettage should be performed and the scrapings examined.

The *Causes* of endocervicitis and endometritis are classified thus: (1) Bacterial invasion—gonorrhœal, streptococcal, staphylococcal, diphtheroid, coliform and other infections, spreading upwards; from laceration at childbirth, retained products after labour or abortion, or dirty instruments; (2) congestion of the uterus, as in displacements, tumours, injury, subinvolution, tumours of the adnexa, excessive coitus and constipation. Endocervicitis of non-inflammatory or infective origin is sometimes due to œstrin deficiency. Microscopic examination of vaginal smears gives important information about the ovarian follicular influence on the pelvic tissues and also about the effects of œstrogen administration (§ 434).

Treatment.—Chronic discharge from the uterus requires general treatment, together with penicillin compound vaginal suppositories, especially if there is menorrhagia. Attention to the diet, with avoidance of much meat, regularity of the bowels and open-air exercise all help.

Endocervicitis, with or without erosion. Pass a Ferguson speculum and swab away the tenacious mucus with wool soaked in liq. potassæ; then apply picric acid, or a mercurial or silver salt several times a week. In this class of case copper or zinc ionisation 2 per cent., 10 to 15 m.a. for ten minutes twice a week for three weeks, is effective. If there is co-existing tenderness and swelling of the cervix, this should be preceded by pelvic diathermy, twice a week. If the body of the uterus is affected also, the probe often passes readily beyond the cervix, in which case intra-uterine ionisation by an expert gives excellent results.

For *endometritis*, antiseptics applied to the interior of the uterus at regular intervals has been the method of treatment in vogue for years. It may succeed in mild cases, but without free drainage it is of little value. The best results are obtained by free drainage. Dilatation should be carried out, under anæsthesia, by Hegar's dilators. A self-retaining rubber catheter is introduced into the cavity of the uterus and kept in position by a catgut suture to the tissue at the external os. Glycerine is injected slowly into the tube by means of a serum syringe until it is seen to flow into the vagina. The vagina is loosely packed with gauze and the tube brought out through an opening in a pad of gamgee, which is kept in place by a T-bandage. Glycerine is injected several times a day into the uterus for four or five days, until the catgut is absorbed and the tube falls out. This is a modification of Remington Hobbs' method.

§ 438. (C) **Dysmenorrhœa** is pain during the menstrual period. Various classifications have been made: three main types correspond to three sets of clinical symptoms: (1) SPASMODIC, due to disorderly and spasmodic muscular contractions of the uterus; (2) INFLAMMATORY, due to some mechanical abnormality or inflammation of the pelvic organs; (3) MEMBRANOUS, due to the passage of membranes or casts from the uterine cavity.

(1) SPASMODIC DYSMENORRHŒA accounts for the majority of cases in young women. The pain occurs during the first few hours or days of the menstrual flow: it may resemble colic or spasm, or it may be a continuous ache; and it may be accompanied by sickness, headache and general malaise. The pain may be referred to the pelvic joints and to the legs. It is most frequent in young single women or in married women associated with sterility. It does not as a rule begin at the onset of puberty but about the age of 18 years or older. It is frequently associated with a sedentary occupation, such as office work, and with deficient exercise and open air; it also occurs when there is overstrain or lack of occupation or interests. Dysmenorrhœa in young women often depends on the general health, as it may disappear when active life is taken up in the country. It can rarely be traced to over-exertion or to physical work. Local causes may be stenosis of the cervix and imperfect development of the uterine muscle; childbirth often cures such cases. The uterus may be in a position of pathological anteflexion and disturbed polarity may occur. The passage of abnormally large clots, thickening of the endometrium and congenital or acquired retro-displacements of the uterus are other causes.

Treatment.—In the case of single women a vaginal examination should not be made unless some pathological lesion is suspected or medical treatment has been ineffective. Examination per rectum is of little value in such cases. The vaginal examination should be made under anæsthesia; if local treatment such as dilatation or curettage is indicated, it can be carried out at the same time. Displacements in girls should be treated by daily exercises, such as those of the knee-chest position; pessaries should not be inserted. *General* treatment such as vitamin full diet, avoidance of constipation, and open-air games or exercises, especially deep abdominal breathing, should be advised: dancing is a substitute for those who are unable to get much open air. Warm baths often relieve the pain if taken before the onset of the period and are usually also beneficial during the period. Considerable success is achieved in dealing with girls of the leisured classes when interesting occupation is found and less attention given to the attractions of rest and warmth at the time. In some cases, however, owing to the severity of the pain, the girl is incapacitated, and special treatment is then imperative.

Remedial treatment at the time of the period consists in hot bottles, hot drinks with sal volatile or essence of peppermint. Antipyrin, phenacetin and caffeine citrate succeed in some cases. Aspirin, liquor sedans, bromides have varying success. Alcohol should never be prescribed: in resistant cases pethidine is useful—(5–10 mgms. orally t.d.s. given the day before the onset of a period and longer if necessary). As dysmenorrhœa in girls is due in some cases to underdevelopment of the uterus, the administration of œstrogen either orally or by injection has proved valuable. Intramuscular injections of œstrogen may be difficult to arrange; hence the synthetic form, stilbœstrol, taken by mouth in doses

of 1–2 mgm. daily for 14 days, commencing on the first day of a period, has advantages. It is doubtful if luteal extracts are of value, as recent work shows that the luteal phase occurs if stimulation of the follicular phase is obtained. Testosterone by mouth or by injection are said to relieve pelvic congestion and pain. Spasmodic dysmenorrhœa is sometimes aided by luteal extract by mouth or injection. Hormone treatment is useless when the genital organs are much underdeveloped. Iron is given when there is anæmia, also the vitamin B complex is helpful. The teeth should be attended to; treat all digestive disturbances, especially constipation. Dilatation of the cervix has cured cases in which this measure is indicated. In cases with other signs of disorder of the sympathetic nervous system, sympathectomy has succeeded.

(2) Inflammatory or Congestive Dysmenorrhœa.—The pain usually begins a few days before menstruation and may be relieved or aggravated at its onset. The pain is continuous, and varies according to the position of the inflamed area which is its cause—*e.g.*, peritonitic adhesions, metritis, subinvolution, fibroid, ovarian cysts, salpingitis or displacement. Stenosis may occur after operation on, or too strong diathermy coagulation of the cervix. Exercise and movement aggravate, whilst rest improves this type of dysmenorrhœa.

Treatment consists in rest and warmth. Heat can be applied in the form of hot sitz baths and prolonged hot vaginal douching, given by a nurse. Sedatives, such as those above mentioned for spasmodic cases, give relief during menstruation. Adhesions are aided by pelvic diathermy. Surgical treatment may be required for the causal lesions.

(3) Membranous Dysmenorrhœa is uncommon. One type occurs in virgins, another is caused by changes in the endometrium due to infection after childbirth. The pain is associated with the passage of portions of or a complete cast of the uterus at frequent intervals. The pain is similar to that of labour pains and is due to the contraction of the uterus in its efforts to evacuate the membrane or cast. There is slight hæmorrhage; at first the pain is not severe but increases and reaches its height as the membrane is being passed. The cast may be solid or may be hollow and triangular in shape like the cavity of the uterus. In some cases the openings for the Fallopian tubes and the internal os may be seen. Under the microscope fibrin is seen with leucocytes and red blood-corpuscles and remnants of uterine glands and vessels. The cast is distinguished from extrusions from the uterus in cases of tubal gestation or uterine abortion by the absence of decidual cells or chorionic villi. There is the history of recurrent attacks unassociated with the early symptoms of pregnancy.

The *treatment* is difficult. Dilatation and curettage have variable success. Relief may be obtained after curettage has been performed twice. In other cases œstrogen therapy has helped.

Endometrioma.—Dysmenorrhœa may be caused by the presence of endometriomata. This growth is similar in character to the endometrium and is said to be carried as a *rest* or graft from the uterus. It takes on menstrual changes during the period, with congestion, swelling and hæmorrhage. The pain may be localised to one side or may be generalised. It occurs before the onset and at the beginning of the flow and is relieved thereby. It is more frequent in women about the early thirties. Removal of the tumour is the only cure.

Mittel Schmertz.—This pain occurs midway between the periods and comes on as a rule after twenty years of age. It is said to be caused by ovulation. This is difficult to prove and all inflammatory lesions must be first excluded. Sedative drugs are used, as in dysmenorrhœa. If pathological lesions are found, the appropriate treatment should be carried out.

§ **439.** (D) **Hæmorrhage.**—*Menorrhagia* indicates an excessive flow at the monthly period; *Metrorrhagia* indicates irregular hæmorrhage from the uterus, irrespective of the period. It is difficult to separate these, as their causes are more or less identical, and they often occur together. Hæmorrhage from the *vulva or vagina* is usually slight in quantity, and its cause readily discovered by inspection. Hæmorrhage from the *cervix* is usually due to polypi, cervical erosion or malignant disease; rarely, it is due to ulceration, syphilitic or tuberculous, or to injury by a pessary. All of these are made out on inspection. Hæmorrhage after coitus is suggestive, especially in older women, of malignant disease or a polypus hanging from the cervix. Slight bleeding from a cervical erosion may be due to œstrogen deficiency.

Local causes of hæmorrhage from the *uterus* are: Endometritis, fibrosis or metritis, fibroids, polypi of the uterus, inflammation of the adnexa and in the pelvis, subinvolution of the uterus, congestion consequent on cardiac or lung disease, malignant disease, retroverted uterus incarcerated in Douglas' pouch, ovarian tumours (occasionally), inversion of the uterus, and extra-uterine fœtation. Flexions and versions of the uterus rarely cause symptoms unless attended by pelvic inflammation or adhesions. *Constitutional* causes are considered in § 440.

In women *over thirty-five* any of the above causes may give rise to hæmorrhage, but malignant disease must be excluded. The sudden supervention of *hæmorrhage* with *acute pain* suggests a miscarriage or an extra-uterine fœtation (§ 446). In women *past the menopause* some lesion of the uterus, especially cancer or uterine fibroid, is nearly always present.

Many of the above mentioned causes of uterine hæmorrhage are dealt with elsewhere. The following are considered here: (I.) Certain Constitutional conditions; (II.) Uterine Fibroid or Polypus; (III.) Chronic Subinvolution (in persons under forty); (IV.) Metritis; (V.) the Menopause; and (VI.) Malignant Disease (in persons over thirty). These conditions will therefore be differentiated here.

§ **440.** I. Hæmorrhage may depend upon certain CONSTITUTIONAL CONDITIONS. (1) Certain women of plethoric build, usually with florid countenances, may have too profuse periods all their lives, and excessive flow on any trivial exciting cause. (2) When associated with hypertension it may be the natural method of relieving this. (3) Prolonged lactation, too many and too frequent pregnancies; (4) residence in tropical climates; (5) acute specific fevers, septic foci, purpura and other blood conditions; (6) mental overstrain, especially with a sedentary life. Strong emotion may cause a single heavy hæmorrhage, possibly through its action on the pituitary. (7) Congestion, as with heart or liver disease;

also after sudden change of temperature or over-exertion. (8) Endocrine imbalance, as at the onset of puberty, when it is said to be due to variations in the œstrogen secretion. The flow may occur every two or three weeks, though the amount may not be increased, and it may be readily excited, as by a hot bath or a day of unusual exercise. Other endocrine causes are deficiency of thyroid and unbalanced production of pituitary and ovarian hormones. There results hypertrophy of the endometrium, a condition seen especially in **metropathia hæmorrhagica,** where also occur follicular cysts of the ovary. It is believed to be due to excessive œstrogen stimulation and absence of luteal influence. There is loss of the menstrual rhythm, profuse and lengthy bleeding with, in some cases, periods of amenorrhœa, also. See § 432.

§ **441.** II. Hæmorrhage due to a UTERINE FIBROID. The symptoms vary with the position of the tumour. These tumours may be submucous, interstitial, or subserous. When the fibroid is *submucous* or *interstitial,* the symptoms of uterine fibroid are (1) menorrhagia and metrorrhagia; (2) discharge and sometimes dysmenorrhœa. (3) On examination with the sound the uterine cavity is found to be enlarged; and (4) on bimanual examination enlargement of the uterus, which is usually hard and bossed from the presence of more than one fibroid, can be detected. The submucous variety tends to become polypoid, remaining attached to the uterus by a pedicle. The *subserous* fibroid may present no symptoms for many years and may even then be discovered by accident. Amenorrhœa may accompany such cases quite as often as menorrhagia, and the latter is never profuse. In short, pressure symptoms may be the earliest indication of a subserous fibroid. In uterine fibroids of all kinds the rate of growth, though it varies somewhat, is nearly always slow; but as the tumour increases there are symptoms of pressure upon the surrounding organs, such as frequent micturition, varicose veins, backache, neuralgia in the legs, indigestion, or hydronephrosis. Fibroids, especially when very large, tend to undergo degenerative changes which give rise to symptoms of toxæmia.

UTERINE POLYPUS. The most common forms are fibroid polypi and mucous polypi. Placental and fibrinous polypi occur, the first after labour or abortion, arising from retained portions of the placenta, the second from the stump of a growth previously removed. When very small, polypi can be made out with certainty only by dilating the os and exploring the interior. When the polypus is larger, or springs from a lower site, examination with the speculum may reveal it hanging from the os into the vagina. After a time it may slough, and cause an offensive discharge.

§ 442. III. SUBINVOLUTION, or the non-return of the uterus to its normal size, is a very frequent cause of menorrhagia after labour or abortion. After a confinement the uterus begins to diminish in size, and at the end of about two months resumes its normal length of 3 inches. In cases of subinvolution we find (1) on vaginal examination that the uterus is enlarged; (2) it tends in most cases to be retroverted and lower than normal; (3) the patient generally complains of backache, bearing-down pain and discharge; and (4) lassitude, weakness, general malaise and anæmia.

Etiology. (1) Toxæmia occurring during pregnancy or the puerperium; (2) retained membranes or portions of placenta; (3) pelvic inflammation; (4) delayed labour or over-distension of the uterus; and (5) the practice of not suckling the infant. Therefore it occurs chiefly after numerous and rapid pregnancies.

§ **443.** IV. METRITIS or FIBROSIS is a condition in which the uterine tissue is thickened, tense, and hard or flabby. Profuse menorrhagia is the chief symptom; there is usually a feeling of weight and dysmenorrhœa, and the uterus is felt to be enlarged and firm. It is caused by infection, rarely syphilitic, usually of gradual onset, and may occur at any age. It may be due to endocrine deficiency.

§ **444.** V. THE MENOPAUSE, or climacteric, is the epoch at which the sexual activity of the female undergoes involution, and the menses, which are the sign of that activity, cease. This may take place in three ways: (*a*) They may cease gradually, without any disturbance of the general health; (*b*) quite suddenly; (*c*) there may be a series of hæmorrhages.

The existence of this cause of menorrhagia or metrorrhagia can only be recognised by the attendant phenomena. (1) The age varies considerably, between thirty-five and fifty-five, the average being about forty-five. (2) "Flush storms," which consist of a hot stage, a cold stage, with or without shivering, and sometimes a stage of perspiration. With many healthy women these flushes appear only when there is a septic focus or intestinal cause co-existing. (3) Nervous phenomena at this time are extremely varied—irritability, restlessness, and depression. (4) While fibroids and other gross lesions sometimes undergo involution at this epoch, the case should be carefully watched lest carcinoma develop. When menstruation ceases at the normal age of menopause, the patient should be assured that the occurrence is physiological: constitutional symptoms can be alleviated by intramuscular injections of œstrogen, or by stilbœstrol by mouth (1 mgm. daily for several weeks, then 0·5 mgm. daily). At and after menopause, atrophic changes in the genital organs cause the vaginal folds to become smooth, often with some vaginal discharge; the influence of ovarian secretion is estimated by microscopical examination of epithelial smears. When the ovaries have been surgically removed, menopause is much more sudden and disturbing.

§ **445.** VI. MALIGNANT DISEASE of the uterus is clinically met with in four forms: (*a*) Cancer of the cervix, chiefly met with in multiparæ, between the ages of twenty-five and seventy; (*b*) cancer of the body, which is chiefly met with in nulliparæ, between the ages of fifty and sixty; (*c*) sarcoma of the uterus, which is rare, unless we include under that term certain fibroids which appear to take on the malignant features of spindle-celled or large round-celled sarcoma; and (*d*) chorion epithelioma, a very rare form following parturition.

The symptoms differ in the first three varieties. (*a*) CANCER OF THE CERVIX usually runs a somewhat more rapid course in younger women. (1) On digital examination the os feels hard, friable, granular; it is so characteristic that this feature and the blood-stained discharge upon the finger are alone, in experienced hands, sufficient to diagnose the disease. (2) In a later stage examination reveals a mushroom-like growth ("cauli-

flower excrescence ") or crater-like depression, readily breaking down and readily bleeding. It tends to spread to the vaginal wall, to the utero-sacral ligaments, broad ligaments, and body of the uterus, leading to fixation of the uterus and a hardness which is easily made out on palpation. (3) Metrorrhagia and menorrhagia are present. (4) In the intervals between the marked hæmorrhages there is a continuous watery discharge, usually pinkish-brown, often with a very offensive odour. (5) Local pain is usually a late symptom, but, like the wasting and the cachexia, is sure to supervene sooner or later. Pain points to invasion of the cellular tissue by the growth.

(*b*) CANCER OF THE BODY of the uterus is chiefly met with in nulliparæ over fifty years of age. Bleeding occurs at a later stage than in cancer of the cervix. The symptoms are: (1) A watery discharge, usually coming in gushes; (2) metrorrhagia, and in the intervals pinkish brain-like discharge; (3) on bimanual examination the uterus is found to be enlarged. (4) If the passage of a sound is attempted, considerable hæmorrhage may take place. It should not be used in cases with much bleeding and offensive discharge. (5) Later on, as the disease extends to the broad ligaments, the uterus becomes fixed; this fixity to the educated finger is very characteristic of the disease. (6) The cachexia, pain and other general symptoms resemble those of cancer elsewhere. The diagnosis from senile endometritis or a degenerating fibroid can be made only by microscopic examination of the discharge or a scraping taken for the purpose.

(*c*) SARCOMA OF THE UTERUS is a relatively rare condition. Its symptoms do not differ materially from those of uterine fibroid, except in the rapidity with which the case progresses, and the liability to deposits elsewhere.

(*d*) CHORION EPITHELIOMA is characterised by bleeding in the late puerperium or after the removal of a vesicular mole. The Aschheim-Zondek test is positive, and a curettage of the uterus with subsequent biopsy confirms the diagnosis. The ovaries show cystic degeneration. *Treatment* consists in early removal of the uterus. In some cases, the metastases clear up when the primary focus is removed.

§ 446. **Extra-uterine Pregnancy** (or Ectopic Gestation) may become manifest by menorrhagia, metrorrhagia, or amenorrhœa. The term is applied to the condition where pregnancy takes place outside the uterus, as a rule in the Fallopian tube, but sometimes in the ovary. The tube usually ruptures at the second or third month after fertilisation, either into the broad ligament (extra-peritoneally) or into the peritoneal cavity. The ovum in some instances is extruded through the abdominal opening of the tube into the peritoneal cavity, forming the so-called Ectopic Abortion.

Symptoms.—(1) In many cases paroxysmal pains are experienced in one iliac fossa; (2) in about 70 per cent. of the cases there is a history of amenorrhœa for some weeks or a month over time, followed in most cases by a history of irregular hæmorrhages from the uterus. A membrane or cast may be discharged from the interior of the uterus at the same time. (3) Other symptoms of early pregnancy, such as morning sickness, are but rarely present. (4) On bimanual examination a swelling is found in the fornix, and the cervix is soft as in early pregnancy. In most cases, however, none of the above symptoms may be noticed by the patient, and advice may not be sought until the time of rupture of the tube, when the patient consults us for *severe pain* and *hæmorrhage.* Extra-peritoneal rupture is attended and followed by the symptoms of pelvic hæmatocele; intra-peritoneal rupture by the symptoms of perforative peritonitis (§ 243). If the rupture takes place about the fourth week the

shock is not so severe, and the hæmatocele often remains extra-peritoneal. The prognosis and treatment are discussed under Hæmatocele (§ 451).

The *Prognosis of Hæmorrhage* depends upon the cause. Uterine bleeding of itself is not fatal to life, but some forms are very intractable, and lead to considerable anæmia, debility, discomfort, and inability to fulfil the duties of life. (1) The undue bleeding at the MENOPAUSE and of SUBINVOLUTION tends to spontaneous recovery, and that which is due to CONSTITUTIONAL conditions is usually amenable to treatment; so also, in many cases, is that due to PELVIC INFLAMMATION, or such cases may develop CHRONIC METRITIS. With endocrine imbalance, as in METROPATHIA HÆMORRHAGICA, the outlook is not favourable (see § 432). (2) METRITIS is one of the most intractable causes, though it responds to local treatment. (3) The prognosis in a case of FIBROID tumour depends upon its position. The submucous varieties and mucous polypi (§ 441) are readily treated, but if neglected these may slough, and produce death by exhaustion and septic intoxication. The subserous form may cause little trouble for many years, and then chiefly by pressure symptoms. The interstitial form is the most serious, and if there be much loss of blood and consequent prostration the patient is seriously incapacitated. Fibroids, even if occurring near the menopause, should be treated surgically, because they rarely disappear spontaneously. Curettage is not permissible. Intensive doses of X-rays have succeeded when operation was refused. (4) CANCER is the most serious of all the causes of hæmorrhage. Cancer of the body of the uterus is not so grave as cancer of the cervix. The chance of recovery depends upon the diagnosis of the disease and its treatment *at an early stage.* If cancer of the cervix is discovered before it has spread to the parts around, or if cancer of the body is taken in hand while the uterus is still freely movable, radium or operation offer a fair prospect of recovery. The results of treatment by radium and deep X-ray therapy are giving much satisfaction and many gynæcological surgeons are employing this method of treatment instead of, or after operation. The prognosis of extra-uterine pregnancy is discussed in § 451.

Treatment of Hæmorrhage.—(*a*) Symptomatic, in all forms. To relieve the hæmorrhage calcium lactate or gluconate in full doses is helpful. Ergot, especially in the form of ergotamine tartrate, by mouth or injection, adrenalin, tinctura hamamelidis, tonics, quinine, are all useful. In some cases injections of progestin are valuable. 1 c.c. of a standardised solution of pituitary extract may be injected intramuscularly or adrenalin applied locally in severe cases. The results of administration of testosterone are still inconclusive. Injections of antuitrin S. are also useful, and the injection of glycerin into the uterus is of value. X-rays in expert hands may cure interstitial fibroids and metritis. Copper ionisation is most useful when there is an infective discharge from the cervix or uterus. Small doses of radium are especially useful in the treatment of uterine hæmorrhage. Menorrhagia of puberty is sometimes difficult to treat; injections or oral

doses of œstrogen may succeed by improving the development of the uterus. In others luteal therapy may benefit by antagonising the effect of the follicular secretion. If these treatments fail a small intra-uterine dose of radium gives excellent results—50 mgm. for 22 hours, administered by an experienced radiologist. Hysterectomy should never be carried out in these cases. (*b*) Remedial treatment is directed to the cause. (*c*) In all cases general measures are required—the food must be nourishing, exercise must be avoided near the period, and the patient must rest in bed while the flow is profuse. Strong purgatives must not be used, but it is extremely important to avoid constipation. Septic foci and constitutional causes must be sought for and remedied. For the menorrhagia of the menopause, bromides, calcium salts and liver preparations are recommended. Anterior pituitary extract given by injection, for several successive days, aids cases of hæmorrhage without a mechanical cause. Thyroid is beneficial in some cases, both in old and young, even when hypothyroidic symptoms are few or absent. Blood transfusion in small amounts is of value.

§ **447.** (E) **Amenorrhœa** is that condition in which the catamenia are either deficient or absent. The term *primary* amenorrhœa is applied to the condition in which menstruation has never occurred, as in rare cases where there is a congenital absence of the organs concerned, and also in cases of infantile uterus and undeveloped ovaries. The condition is fully discussed in § 432. *Apparent* amenorrhœa is that form in which there is a feeling of fulness in the breasts and abdomen every month, but the menstrual flow is retained behind an imperforate hymen, an occluded os or vagina. *Physiological* amenorrhœa is the cessation of the menses which occurs in pregnancy, during lactation and at the menopause. In *secondary* amenorrhœa, the flow, after having been once established, ceases or becomes deficient for a time.

In Pregnancy, the physiological cause of amenorrhœa, the *General Symptoms* are as follows : (1) Morning sickness is usually one of the earliest, coming on about the first or second, and ceasing at the fourth month; frequent micturition is also a sign. (2) The mammæ present a dark areola around the nipple, they become enlarged and after the third month contain colostrum. The *Local Signs* are : (1) On digital examination there is *a softness* of the os which is unmistakable to the educated finger; (2) a gradual increase in the bulk of the uterus is early apparent. These are the earlier symptoms. From the third and fourth month we have a series of unmistakable signs—viz., (3) about the eighteenth week fœtal movements can be felt by the physician, and (4) the fœtal heart-sounds (at the rate of 120 a minute) can be heard on auscultation, usually midway between the umbilicus and one or other anterior superior spine; and (5) ballottement can be made out about the fifth or sixth month. (6) X-ray examination will assist.

The *diagnosis* of early pregnancy is often difficult. Since the introduction of the Aschheim-Zondek and other tests the accuracy of diagnosis has

been made almost certain. It depends upon the presence of anterior pituitary hormone in the urine (§ 926).

The *Causes* of SECONDARY AMENORRHŒA may be divided into constitutional and local causes. (*a*) *Constitutional* causes are the most frequent. Endocrine imbalance as a cause of amenorrhœa is considered in the introductory paragraph to this chapter (§ 432); to this may be ascribed the amenorrhœa following a sudden change of abode or mode of life, anxiety, stress or mental shock. It also occurs with tuberculosis and anæmia and after severe illness, during prolonged lactation, chronic poisoning with cocaine and opium, and sometimes with pyrexia. (*b*) The most important *local* cause is an ovarian tumour, with which menstruation is often absent or irregular. Other causes are a chill during menstruation, inflammatory conditions in the pelvis, superinvolution of the uterus, and extra-uterine fœtation.

Treatment of constitutional causes includes plenty of fresh air, exercise, and healthy living. Warm hip and foot baths at the expected time are useful. Keep the bowels regular; iron, calcium and vitamin D preparations are beneficial. Adopt general tonic treatment in young unmarried girls; only after these have failed should local causes be investigated. When the uterus is normal in size, the ovaries can be stimulated by anterior pituitary injections, together with small doses of thyroid. When the uterus is undeveloped œstrogen by injection or by mouth, as stilbœstrol, may be of use, but fails if marked mal-development is present. Pelvic galvanism and diathermy succeed in some cases of secondary amenorrhœa.

SUDDEN SUPPRESSION of the catamenia is a form of amenorrhœa which requires special treatment. The flow has probably come on normally, and then suddenly ceases on the second or third day, and the patient suffers a good deal of general discomfort. In such cases the patient should put her feet in hot water or a mustard bath, or sit in a warm hip-bath, and then should get into a thoroughly warm bed with hot bottles and take hot drinks. Subsequently saline purgatives in small doses, and general attention to the health are indicated. When the time of the expected period again comes round, repeat the same procedure.

§ **448.** (F) **Pelvic Pain.**—Pain in and about the pelvis is one of the commonest symptoms of disorder of the female reproductive organs. "Bearing down" is often spoken of; and "backache" or pain over the sacrum is so constant a feature of uterine disorders that it has come to have that association in the minds of the laity. The position and character of pelvic pain vary with the different maladies, but its degree is largely influenced by the temperament of the patient. Reference has already been made to painful menstrual periods (dysmenorrhœa), but the causes of a continuous pain (without reference to the menstrual period) are usually due to acute or chronic inflammatory lesions of the reproductive organs which have a toxic or bacteriological origin. Referred pain is frequently present in acute gynæcological lesions because the parietal peritoneum is involved through the somatic nerves.

CLINICAL INVESTIGATION OF **Acute Pelvic Pain.**—To ascertain the significance of pain in a given case attention must be paid to the general mental and nervous condition of the patient. The method of procedure in the investigation of abdominal pain in § 242 should be studied here.

(1) The HISTORY of the ONSET, and the AREA of PAIN give valuable indications as to the nature of the lesion. If the pain begins at the umbilicus and later extends to the right iliac fossa suspect appendicitis. If the pain starts in the lower abdomen and later settles into one or both iliac fossæ, suspect tubal disease, which is often associated with pelvic peritonitis (§ 449).

(2) TENDERNESS is present either in the skin (hyperæsthesia) or in the deep structures (deep tenderness), in association with congestion or inflammation of underlying organs. In appendicitis there is usually deep tenderness over MacBurney's point, but if the appendix is in the pelvis the tenderness may be much more marked on vaginal or rectal examination. Morley states that if the maximum tenderness is close to the anterior superior spine, the appendix is to the outer side of the normally situated cæcum; but if the maximum tenderness is on the lower part of the right rectus, close to the middle line, the appendix is hanging over the brim of the pelvis. Any other inflamed organ in the pelvis may cause much more tenderness by vaginal or rectal examination than by abdominal palpation. And see § 247.

(3) RIGIDITY is generally associated with deep-seated tenderness and usually indicates an acute inflammatory condition. (4) Inquire as to MENSTRUAL IRREGULARITY, which gives important indication as to pregnancy or abortion. (5) VAGINAL DISCHARGE, if acute or severe, may point to extension upwards of gonorrhœal or other sepsis. (6) RECTAL OR VAGINAL EXAMINATION must never be omitted, as it detects tenderness, swelling, discharge or displacement.

UTERINE PAIN is rare apart from pregnancy. (1) It is associated with alteration in menstruation and (2) is usually spasmodic in character, due to irregular uterine contractions.

SPASMODIC PAIN associated with UTERINE HÆMORRHAGE may indicate ABORTION, a RUPTURED TUBE, or TUBAL GESTATION (§ 446). The condition of the patient in the latter is usually much more serious, as shock and internal hæmorrhage may be present. Shoulder pain is sometimes an indication of tubal gestation. Examination of the pelvis will clear up the diagnosis.

SEVERE PAIN and COLLAPSE follow TORSION OF THE PEDICLE of an OVARIAN CYST. The tumour will be felt on palpation.

Pain in the left iliac fossa associated with signs of peritonitis may be due to DIVERTICULITIS (§ 321).

If there is difficulty in diagnosis between a tubal lesion and an appendix, the insertion of a glycerine drain in the uterus will cause cessation of spasmodic pain in salpingitis, whereas if the appendix is involved, uterine drainage has no effect.

EXAMINATION under an ANÆSTHETIC may clear up the diagnosis. In the case of the appendix, prompt surgical treatment may be indicated, but tubal conditions react to expectant treatment, for the time at least.

General Treatment of Pelvic Pain.—It is unnecessary to lay stress upon the fact that successful treatment of pain lies in the proper recognition of its cause. When the accurate diagnosis has been made the appropriate treatment will suggest itself. If there are displacements of the pelvic organs, tumours, etc., surgical treatment may be the only form which will give relief. If, however, the cause is unknown, or if there is an inflammatory lesion, medical treatment should be carried out until there is a definite indication for surgical interference. Pain is relieved by the administration of sedatives, as indicated in Chapter IX. The phenacetin group give relief, also aspirin and tab. codein co. (veganin). Morphia is used only

in severe conditions, owing to its tendency to mask symptoms, and thus delay proper surgical treatment. Chloral hydrate and bromides aid cases with hypersensitive nerves; and are useful because they help to reduce the blood pressure. Local treatment, especially for inflammatory or septic lesions, is called for. The most efficient form of treatment is the application of heat in the form of electric light or sitz baths. Prolonged hot vaginal douching should be carried out by a nurse, not by the patient herself. When the acute stage has passed, pelvic diathermy is of value.

The **pelvic pain** *came on* **acutely** *and recently; it is accompanied by more or less* CONSTITUTIONAL DISTURBANCE—PELVIC PERITONITIS, INFLAMMATION of the UTERINE APPENDAGES, PELVIC HÆMATOCELE, ACUTE CYSTITIS, or some other INFLAMMATORY CONDITION within the pelvis, may be suspected; the reader should first turn to § 448.

If the PAIN *has come on* VERY SUDDENLY *with* FAINTNESS *and* NAUSEA, turn first to PELVIC HÆMATOCELE, § 451; *if it be accompanied by* METRORRHAGIA, it suggests MISCARRIAGE, or EXTRA-UTERINE FŒTATION (§ 446).

§ **449. Pelvic Peritonitis** is a frequent cause of pelvic or lower abdominal pain. It is due to septic infection causing an inflammatory condition of the peritoneum covering the pelvic organs. Exudation may be present and in chronic cases adhesions may lead to a matting together of the uterus and appendages. The terms peritonitis and cellulitis have been used to indicate different lesions; but these conditions are almost identical. The involvement of the cellular tissue may be an extension from the pelvis especially in puerperal cases. In the case of septic lacerations of the cervix the cellular tissue may become involved first and the peritoneum later. The symptoms of pelvic sepsis may be acute or chronic.

Symptoms of ACUTE PELVIC PERITONITIS: (1) Severe pain across the lower abdomen; (2) on examination, distension and tenderness of the abdomen and (3) a tender swelling may be felt. (4) The legs are flexed; the patient lies on her back. (5) The quick pulse and high temperature indicate the severity of the condition. Vomiting may occur. (6) On bimanual examination there is great tenderness, but little may be made out. Later, when exudation and adhesions have taken place, (7) a swelling may be felt behind the uterus, pushing it forwards, and the uterus cannot be moved, owing to adhesions. (8) There is usually hæmorrhage or mucopurulent discharge from the cervix.

Symptoms of CHRONIC PELVIC PERITONITIS: (1) pain in the lower abdomen in one or both iliac regions. (2) Backache is usual; the pain is constant, bearing down in character and is much worse at the menstrual period. (3) Chronic ill-health is usually marked. (4) On bimanual examination the uterus may be found to be retroverted and fixed by adhesions, posteriorly or to one or other side, and (5) there is tenderness due to adhesions, especially when in the ovarian region.

When the CELLULAR TISSUE is involved in chronic conditions the pain (1) may be referred to one leg (which may be drawn up to relieve the pain) and on examination the swelling in the pelvis may be limited to one side.

(2) Backache is marked. (3) The pain is dragging in character when there are posterior peritonitic adhesions. (4) Pus may be found in acute or subacute septic conditions of the cellular tissue. Abscesses may point towards the vagina or rectum, or upwards in the direction of Poupart's ligament. Pus may also be found in the Fallopian tubes and ovary as the result of septic infection of the uterus.

Etiology.—(1) Infective processes from lacerations during labour or sepsis during the puerperium. (2) Septic abortion, which in most cases has been induced, is a frequent cause of streptococcal infection. (3) Direct infection downwards from the appendix by the colon bacillus, or (4) upwards from the cervix by the gonococcus or other organisms.

Course and Prognosis.—(*a*) In *acute pelvic peritonitis* the acute symptoms should subside in a week; if widespread adhesions are present, part of the exudation will be absorbed, and part will remain, giving rise to the symptoms of chronic pelvic peritonitis. The prognosis will depend (i.) upon the extent of the inflammation, and (ii.) its cause. If it is the sequel to an acute attack with widespread adhesions, the patient, if untreated, will probably have chronic pelvic pain, menorrhagia, a discharge and dysmenorrhœa all her life, with resulting chronic invalidism and nervous symptoms. Sterility is usual. If due to extension from a diseased organ, the patient will be subject to relapses with acute pain after any imprudence in the way of chills or over-exertion. If the fever continues for four or five weeks pus has formed, and the patient will be invalided until the pus finds an exit (which may not be for months). The swelling felt in one lateral fornix becomes larger, pushing the uterus to one side, and later on a firm lump, which may extend to the iliac fossa, is felt along Poupart's ligament. The pus may point in the iliac fossa or follow the line of the vessels into Scarpa's triangle; or it may burst into the vagina, bladder, rectum, or peritoneal cavity. When the cellular tissue is involved adhesions and fibrous tissue are formed rather than pus. They do not interfere with pregnancy, and may be absorbed in time, but anteflexion or version of the uterus is a common result of the contraction of the utero-sacral ligaments.

Treatment.—Acute pelvic infections must be treated by (1) absolute rest in bed; (2) hot fomentations or turpentine stupes to the abdomen: or the electric light cradle should be used at intervals; (3) in every case a sulphonamide and/or penicillin should be given; (4) saline purges; (5) morphia, if necessary, to relieve pain. (6) Drainage of the uterus by glycerin gives satisfactory results (§ 436). Watch for the formation of pus and deal with it surgically. If pus cannot be detected by palpation, the leucocyte count will settle the diagnosis: drain if possible by the vaginal route. *Preventive* treatment consists especially of: (1) cleanliness of the hands and surroundings in cases of labour or abortion; (2) prevention of extension of sepsis from the cervix or perineum; (3) free drainage of the uterus. Treatment of *chronic* pelvic infection is by hot sitz baths, hot douches given by a nurse, tampons or pessaries of ichthyol and glycerine. Pelvic diathermy in mild doses allays pain and disperses adhesions. Cold

or damp and undue exertion in walking or standing must be avoided; and a certain daily interval of rest in the recumbent position should be ordered. In toxic or septic conditions, where there are intestinal symptoms, colonic lavage is beneficial. Belladonna and trasentin allay intestinal spasm. Drastic purgation is not advisable for gynæcological conditions; give, rather, confection of senna, liquid paraffin, elixir of cascara, magnesium sulphate in small doses or mycolactin. If symptoms persist, surgical advice should be sought. When suppuration has occurred, the pus must be evacuated by free incision, preferably per vaginam. For further treatment a volume of gynæcology should be consulted.

§ 450. **Infective Conditions of the uterine appendages** may also be a cause of pelvic pain. It is not always possible to separate the inflammatory affections of this region.

Oophoritis is inflammation of the ovary, and should be distinguished from ovarian neuralgia. The *Symptoms* of oophoritis are so frequently accompanied by those of pelvic peritonitis that it is difficult to differentiate them. Indeed, *acute oophoritis* is found solely with acute pelvic peritonitis or cellulitis (*q.v.*). *Chronic oophoritis* may be recognised by (1) severe pain at the pelvic brim, extending down the thigh of the affected side; (2) pain increased by any pressure on the pelvic viscera (*e.g.*, by much standing, constipation, or flatus in the abdomen, and in severe cases by sitting); (3) menorrhagia and dysmenorrhœa, because endometritis so often accompanies oophoritis; and (4) dyspareunia. (5) The ovary is usually prolapsed, and therefore, per vaginam, a walnut-sized swelling is found at the site of the ovary, to one side of or behind the uterus, acutely tender to touch, which causes a "sickening" pain. *General* symptoms, referable for the most part to the nervous system, often supervene. The *Causes* of (1) acute oophoritis are sepsis after labour, abortion, or surgical operation; (2) chronic oophoritis may be due to the same causes as peritonitis, to certain fevers (*e.g.*, mumps), or to infection after a chill, with suppression of menstruation.

Salpingitis (inflammation of the Fallopian tubes) occurs in three forms, hydro-, pyo-, and hæmato-salpinx. (i.) When the fimbriated end of the tube is closed by adhesions, the exudation within, unable to escape, tends to accumulate in the tube instead of escaping by the uterine opening (hydrosalpinx); (ii.) when the tubes are filled with pus (tuberculous, gonorrhœal, or septic) the condition is named pyosalpinx; (iii.) when the tubes are filled with blood, hæmatosalpinx.

The *Symptoms of salpingitis* are (1) pain across the lower part of the abdomen, usually greater on one side, often shooting down one leg; (2) on examination a sausage-shaped swelling is found, usually double, running from the lateral fornices to Douglas' pouch; (3) as peritonitis usually accompanies it, the uterus is less mobile than normal; (4) dysmenorrhœa, discharge and menorrhagia. (5) General symptoms—in hydrosalpinx there may be none, but pyosalpinx is accompanied by fever. In a pyosalpinx of sudden onset (often gonorrhœal), the fever may be very high and the symptoms those of acute peritonitis. *Causes.*—(1) Acute salpingitis is usually due to streptococcal or gonorrhœal infection extending upwards. Sometimes *B. coli* and streptococcus fæcalis appear to travel by direct extension from an inflamed appendix. (2) The commonest form of salpingitis in young single women is tuberculous. It is generally bilateral, and apparently enters by the blood or by extension from tuberculous peritonitis. It is generally chronic. (3) A chronic or subacute vaginitis or endometritis extending upwards may result in salpingitis, especially after abortion. See pelvic peritonitis (§ 449) for other causes. (4) Hæmatosalpinx is due usually to a ruptured extra-uterine pregnancy.

Prognosis.—In *oophoritis* this depends on the extent of the inflammation. If there is much matting the case is really one of peritonitis. If the inflammation is confined to the ovary the prognosis is favourable, provided the cause be removable and the

patient is not neurotic. With *salpingitis* sterility may result; when the infection has died out, Rubin's inflation test, and X-ray after iodised oil B.P. (lipiodol) injection give information as to the patency of the tubes. Pyosalpinx is dangerous to life, as it may at any time burst into the peritoneum. Tuberculous salpingitis is very chronic, and less painful than the other forms. In all forms there is a tendency to relapse, and to peritonitis by extension rather than to spontaneous cure.

Treatment.—Acute and chronic *oophoritis* are treated like peritonitis (*q.v.*), together with hot applications to the hypogastrium when the pain is severe. Constitutional treatment is important. In *acute salpingitis*, with pus and the condition certainly diagnosed, some recommend laparotomy and removal of the tube; others consider that rest in bed in the Fowler position with a sulphonamide followed by penicillin injections are best. Glycerin drainage of the uterus should be tried in every case before abdominal section is contemplated. There is a great element of risk in operating in the acute stage, owing to dissemination of infection. In *chronic salpingitis*, rest, penicillin suppositories, or ichthyol and glycerin tampons may be tried for several weeks. If this treatment and pelvic diathermy fail, it may be necessary to remove the tubes.

§ 451. **Pelvic Hæmatocele** is an effusion of blood either into the peritoneal cavity (intraperitoneal) or into the connective tissue of the broad ligament (extraperitoneal), usually due to a ruptured tubal pregnancy (§ 446). Here there is a *sudden onset* of (1) severe pain, starting in one iliac fossa and soon spreading over all the lower part of the abdomen, accompanied by (2) faintness, perhaps collapse with (3) nausea, and in some cases vomiting. (4) There may be some uterine hæmorrhage, with discharge of a cast of the interior of the uterus. (5) On examination, the uterus, in the intraperitoneal variety of pelvic hæmatocele, is found pushed forwards behind the pubis, while in the extraperitoneal variety the swelling is smaller, and causes a lateral displacement of the uterus as in pelvic cellulitis. The intraperitoneal variety, if large, forms a lump which can be felt, on bimanual examination, both in Douglas' pouch and above the pubes, and the abdomen is tender and distended. After forty-eight hours, adhesions form and the uterus is fixed, and other signs of pelvic peritonitis may then ensue. The temperature begins to rise in twenty-four hours after the onset of pain—that is to say, when the pelvic peritonitis commences.

Diagnosis.—If the bleeding is (*a*) intraperitoneal, the hæmorrhage is rapid and excessive; (*b*) if extraperitoneal, it is usually slow and limited in amount and tends to become encysted. (*a*) In the former, in addition to the symptoms of abdominal pain with collapse, there are the symptoms caused by hæmorrhage, viz., pallor, restlessness and air-hunger. The diagnosis from a *ruptured viscus* (§ 243) is very difficult at first. (*b*) When there is a smaller amount of bleeding, there may be acute pain and collapse, as above, but the symptoms may subside after a few hours, and attacks of pain may recur at intervals for days. The local signs resemble *pelvic cellulitis*, from which it may be diagnosed by a history pointing to extra-uterine pregnancy, and by the fact that pyrexia is absent at the onset, and there is pallor and a low tension pulse.

Prognosis.—If hæmorrhage be large, death has been known to occur in about an hour. In smaller hæmorrhages adhesions due to pelvic peritonitis or cellulitis follow, and the exudation may be (i.) entirely absorbed, or (ii.) may go on to suppuration with a danger of general peritonitis. When due to extra-uterine pregnancy, an extra-peritoneal is not so immediately serious as an intraperitoneal hæmorrhage. Secondary rupture may occur into the peritoneum. In rare cases the fœtus may live till full time, when the patient goes through a spurious labour, after which the placenta beco mes absorbed and the fœtus mummified, causing no symptoms.

Treatment is operative, except in the encysted variety, when operation is not so urgent.

The **pain** *is of a* **chronic** *character, is of considerable duration, and is* UNATTENDED *by* PYREXIA. Almost any of the different diseases mentioned in this chapter may be suspected. Examination may reveal METRITIS,

ENDOCERVICITIS, CHRONIC PELVIC PERITONITIS (§ 449), or a UTERINE DISPLACEMENT; or careful bimanual examination may reveal a PROLAPSED OVARY or an INFLAMED TUBE. UTERINE DISPLACEMENTS and PELVIC TUMOURS alone remain to be considered. PROLAPSE OF THE UTERUS is a cause of dragging pain, especially in its early stages.

§ 452. **Uterine Displacements.**—The normal position of the uterus is one of anteversion, with slight anterior flexion. The uterus undergoes physiological displacements according to the fulness of the bladder and rectum. In itself a displacement leads to no symptom; the symptoms so often associated with displacement are due in the majority of cases to the inflammatory processes in or near the uterus which have caused the displacement. Tumours and inflammatory exudation in the pelvis may cause LATERAL or UPWARD DISPLACEMENTS of the uterus.

FORWARD DISPLACEMENTS (ANTEFLEXION).—On bimanual examination the os is found to be high up, and the fundus is felt unduly far forward. In single women a stenosis of the os or an elongated cervix with spasmodic dysmenorrhœa may accompany a forward displacement of congenital origin. As above stated, *Symptoms* may be entirely absent, and attention is first drawn to the condition when other mischief, such as pelvic inflammation, endometritis, parametritis, or a history of dysmenorrhœa, sterility, or constantly recurring abortions, is present.

Etiology.—(1) A congenitally ill-developed uterus is often displaced forwards. A forward displacement is diagnosed as pathological as distinct from physiological, when there is lessened mobility of the uterus, and pain on attempting to move it. Forward displacements are found in association with (2) pelvic peritonitic adhesions, and (3) cellulitis affecting chiefly the utero-sacral ligaments.

Prognosis.—Anteflexion is a frequent concomitant of sterility. Its treatment is extremely troublesome, but if consistently and carefully carried out a radical cure is certainly to be expected unless the condition is due to a considerable degree of pelvic peritonitis or cellulitis, when the prognosis depends upon the removability of these conditions.

Treatment.—Treatment must be directed to any pelvic peritonitis or cellulitis present (*q.v.*). Ichthyol tampons and hot sitz baths with purgative treatment work wonders in the slighter forms. Bimanual massage is sometimes practised where the anteflexion is due to contraction of the utero-sacral ligaments. Dilatation of the cervix has aided some cases.

BACKWARD UTERINE DISPLACEMENTS consist of *retroversion* and *retroflexion*. In a backward displacement there is also a certain degree of descent of the uterus. Retro-displacements in themselves cause no symptoms; sometimes they are congenital. On examination the finger detects the forward displacement of the cervix, which is usually somewhat lower than normal. The uterus is not palpable in the anterior fornix, whereas a lump is felt in the posterior fornix, which is found to be the uterus because it is movable with the cervix, and can be felt to be continuous with the cervix.

Symptoms arise when pelvic adhesions are present, or when the displaced organ interferes with other organs in the vicinity. In such conditions, a retroverted uterus gives rise to (1) pain in the back and the lower abdomen of a bearing-down, dull, aching character; (2) dysmenorrhœa and menorrhagia; (3) constipation and painful defæcation. (4) If pregnancy occurs, the sickness of the early months is excessive, and after the fourth month there may be retention with dribbling of urine.

Diagnosis.—The diagnosis of a backward displacement is not difficult, but the diagnosis of the cause may be obscure. It is important first of all to determine whether the uterus is freely movable or not, as the prognosis and treatment differ.

Etiology.—(i.) Congenital; (ii.) the dragging of adhesions consequent on pelvic peritonitis; (iii.) changes in the uterine tissues, such as subinvolution, or tumours in the walls; (iv.) relaxation of the ligaments, as after pregnancy; (v.) sudden fall or strain; and in a few cases (vi.) a habitually over-distended bladder. Several of these causes may act in combination; thus, subinvolution together with relaxation of the ligaments causes a retroversion with a certain amount of downward displacement of the uterus, as pointed out in Prolapse (§ 455).

Prognosis.—(1) So long as the uterus is freely movable and not enlarged, there may be no symptoms until pregnancy occurs. Most often, perhaps, constantly recurring abortions take place. (2) In time retrodisplacements are apt to lead to congestion and enlargement of the uterine body, with endometritis, cervical erosion, and prolapse of the ovaries. Adhesions may follow the chronic inflammation of the tubes and ovaries. (3) Where the uterus is bound by adhesions, there is a condition which, according to Playfair, is "not fatal, but tends to life-long discomfort."

Treatment.—(1) If the displacement is giving rise to no symptoms, treatment is not required. If a backward displacement gives rise to pain, knee-elbow exercises should be advised three times daily and later a Smith-Hodge pessary inserted. If there are adhesions which prevent replacement, a course of sitz baths with glycerine and ichthyol tampons, or of pelvic diathermy frequently facilitates replacement. If the symptoms continue, examination under anæsthesia should be carried out, followed by operation where necessary. If there is retroversion of a gravid uterus, rest in bed with the pelvis raised and frequent posturing in the knee-elbow position is usually successful. Retention of urine must be dealt with. Rarely is it necessary to perform an operation in these cases and induction of abortion should not be advised. In unmarried women, where the health of the patient is seriously affected by the displacement, the best treatment is operative, when necessary. Gilliam's suspension of the uterus by the round ligaments is satisfactory. In unmarried women no local treatment should be carried out; pessaries are an objectionable method.

§ 453. (G) The following are some of the more important **Pelvic Tumours and Vaginal Swellings**: (*a*) *Internal tumours*—(1) uterine fibroid; (2) cervical or uterine polypus; (3) cervical or uterine cancer; (4) retroverted uterus; (5) pelvic cellulitis;

(6) ovarian tumour; (7) pyosalpinx; (8) appendix abscess; (9) pelvic hæmatocele; (10) hydatid of the pelvis. (*b*) *External swellings* or swellings about the vulva may be due to (1) prolapse of the uterus; (2) inversion of the uterus; (3) prolapse of the vaginal walls (cystocele and rectocele); (4) cysts or tumours of the vaginal wall—*e.g.*, of Bartholin's gland; (5) uterine polypus with a long pedicle; (6) local conditions of the *vulva*, such as abscess, hæmatoma, or labial thrombosis (§ 436); (7) cysts of the vaginal wall, usually found on the anterior wall, about the size of an egg and painless; (8) hernia.

Most of these various conditions have already been fully referred to, but three conditions which may appear as external swellings remain to be described—PROLAPSE OF THE VAGINAL WALLS, PROLAPSE OF THE UTERUS, and INVERSION OF THE UTERUS.

§ 454. **Prolapse of the Vaginal Walls** is very common in multiparæ, and especially affects the anterior wall. It is then named cystocele, because of its close connection with the bladder; indeed, the anterior vaginal wall may draw down the posterior wall of the bladder along with it. Prolapse of the posterior wall may occur, and when the rectum is prolapsed also, it is named rectocele. But, as the rectum is not so intimately attached to the posterior vaginal wall, a prolapse of that wall is not usually a rectocele. The only symptom in addition to the swelling may be difficulty in passing water until the prolapsed part is pushed up. The diagnosis from a cyst of the vaginal wall is made by passing a sound per urethram and with one finger in the vagina, feeling the point of the instrument in the bladder. The chief predisposing cause of prolapse of the vaginal wall is a ruptured perineum.

For the *Treatment* of the two conditions, see below.

§455. **Prolapse of the Uterus** is its displacement downwards. Three degrees of displacement are described: (i.) The organ may occupy a position somewhat lower than normal; (ii.) it may have partly or entirely passed through the vaginal orifice (procidentia); and (iii.) in extreme procidentia it lies entirely outside the vulva, the body lying in the inverted vaginal wall.

In slighter cases the vaginal wall is seen coming down on asking the patient to strain. In severer degrees the cervix can be seen and the body of the uterus and the ovaries can be felt. The other symptoms of prolapse of the uterus are: (i.) The uterus is enlarged, the cervix is frequently hypertrophied, there may be accompanying endometritis or endocervicitis; (ii.) in early cases there may be incontinence or frequent micturition; later, there is difficulty in passing water till the prolapsed organ is pushed up; (iii.) sometimes there is a weight or a bearing-down feeling in the pelvis, but more often no pain is complained of, only the discomfort of the lump during walking and sitting. In the early stages, on the other hand, backache may be a prominent feature. (iv.) The uterus is usually retroflexed. (v.) Leucorrhœa is usually troublesome. Ulceration of the external parts is apt to supervene on procidentia.

Etiology.—(1) As in prolapse of the vagina, (i.) a ruptured perineum; (ii.) a relaxed condition of the parts after labour, usually associated with sepsis; and (iii.) a laborious occupation which demands much muscular strain, such as that of a washerwoman. The exciting causes are (i.) increased intra-abdominal pressure, such as occurs with undue muscular strain, sagging of dilated intestine, or undue deposit of abdominal fat. (ii.) The increased weight of the uterus in cases of chronic subinvolution or tumours of the walls.

Treatment.—Preventive treatment is most important. All lacerations occurring during delivery should be stitched up at the time. For chronic cases, rest, tampons, massage and electrical treatment are of value. A well-fitting two-way elastic stretch corselet with strong shoulder-straps and suspenders is very beneficial in supporting the intestine and preventing downward pressure upon the pelvic organs. Belts can be worn when fitted to the patient's requirements: some belts owing to want of proper support of the sagging intestine, are of more harm than use. Pessaries may be tried in some cases, but operative measures give the best permanent results.

Inversion of the Uterus.—Sudden inversion of the uterus may occur in the third stage of labour, when the fundus is relaxed, but here we are concerned only with the

chronic form of inversion, a very rare condition. It may be the sequel to acute inversion if the patient survives the shock, or to the dragging of a tumour. The fundus alone may be inverted through the os, or the whole uterus may be inverted. (1) The swelling is red, bleeds readily, and is tender. (2) The sound cannot be passed the normal distance, if at all. (3) Bimanually the fundus is found absent; and if a sound is placed in the bladder in the middle line and the finger in the rectum these can be made to meet without any uterus being felt. (4) There may be symptoms of bearing-down, menorrhagia, and leucorrhœa. (5) The orifices of the Fallopian tubes can sometimes be distinguished. A *Diagnosis* may have to be made from fibroid polypi, in which the fundus is not absent from its usual position.

Prognosis.—There is no tendency to spontaneous cure. The *Treatment* is operative; the reader should consult a text-book on Gynæcology and Obstetrics.

§ 456. (H) It is proposed to discuss briefly the causes of the following symptoms for which the physician may be consulted: (*a*) DISORDERED MICTURITION (Retention. Unduly Frequent, Painful, or Difficult Micturition and Incontinence); (*b*) PAINFUL DEFÆCATION; (*c*) PAIN ON SITTING; and (*d*) DYSPAREUNIA.

(*a*) **Disordered Micturition** is dealt with more fully in kidney diseases (§§ 420 to 422); here only a few of those special to the female will be mentioned.

I. RETENTION OF THE URINE.—The *Causes* peculiar to women are impacted fibroids, malignant disease of the cervix involving the vagina, tumours of the vagina, a retroverted uterus (especially about the fourth month of pregnancy), and other conditions causing obstruction of the urinary passage consequent on pressure over the mouth of the bladder. The condition is also found in reflex retention after operations on the perineum and in hysteria.

II. FREQUENT MICTURITION may be caused in women by (i.) pressure on the bladder by early pregnancy, an enlarged anteflexed uterus or a tumour; (ii.) a vascular caruncle of the urethra; (iii.) acute cystitis; (iv.) cystocele; (v.) pelvic inflammation, especially during the early stages; (vi.) calculi and crystals; and (vii.) various nervous conditions.

III. PAINFUL MICTURITION is found especially in connection with urethral caruncle, cystitis, and in the early stages of pelvic inflammation or oophoritis.

IV. INCONTINENCE OF THE URINE is found (i.) in vesico-vaginal or vesico-uterine fistula; or (ii.) after dilatation of the urethra has been performed—*e.g.*, as a preliminary to lithotrity.

V. DIFFICULT MICTURITION is found (i.) after labour, when the parts are swollen and bruised; (ii.) with prolapse of the uterus, in which case the symptom is relieved on pressing upwards the prolapsed parts; (iii.) all causes of incomplete obstruction.

(*b*) **Painful Defæcation** may be due to (i.) retroverted and retroflexed uterus, especially when bound down by adhesions; (ii.) an incarcerated retroverted pregnant uterus; (iii.) pelvic inflammation when acute; (iv.) oophoritis; (v.) prolapsed ovary; (vi.) coccydynia; (vii.) a fibroid or other uterine tumour pressing upon the rectum and (viii.) rectal disease, *e.g.*, hæmorrhoids, proctitis or fissures.

(*c*) **Pain on Sitting and Coccydynia** are often associated with painful defæcation. (1) The commoner *external* causes of painful sitting are (i.) a vascular caruncle of the urethra; (ii.) vulvitis and all other acute conditions of the vulva; (iii.) hæmorrhoids or fissures of the anus. (2) The *internal* causes of painful sitting may depend upon (i.) an increased pressure within the pelvis—*e.g.*, pelvic inflammation, or any tumour within the pelvis; (ii.) injury or inflammation affecting the sacro-sciatic and the sacro-coccygeal ligaments; (iii.) a movable condition of the sacro-iliac joints after parturition; or (iv.) a rheumatic condition of the same joints. (v.) Dislocation or malunited fracture, inflammation, or " neuralgia " of the coccyx is also a recognised cause of the condition.

Diagnosis.—The diagnosis of pelvic inflammation is discussed elsewhere. *Neuralgia* of the coccyx is known by the fact that the coccyx is sensitive to the touch. It may be connected with constipation or disorder of the rectum. Injury of the sacro-sciatic or sacro-coccygeal ligaments is known by: (i.) the history of pain often dates

from childbirth, or from the injury which produced it; (ii.) pain is produced by pressure on the ligaments, which tightens them; and (iii.) there is an absence of swelling or dislocation of the bone. *Dislocation of the coccyx* has no pain or tenderness, and is known by the fact that the bone, in most conditions, is displaced backwards. When the dislocation is found to be forward, it is much more painful, so that the patient usually sits on one ischial tuberosity—*i.e.*, sits sideways. In a *movable condition* of the joints there is a history of accident or of forceps delivery at confinement. In slight cases it may be very difficult to diagnose except by means of X-rays. *Rheumatism* is known by the absence of other local signs and by the shifting character of the pain, and perhaps the fact that the patient has other manifestations of rheumatism.

Prognosis and Treatment.—Vulvitis and pelvic inflammation are discussed elsewhere. Inflammation and neuralgia of the coccyx are usually cured by laxatives, hot baths, sedative and electrical applications. Injury which has affected the ligaments may also be cured by laxatives and hot baths, but the improvement is slower. Some advise in extreme conditions the division of the ligaments. Dislocation of the coccyx, if backward, may be a cause of no great inconvenience, but if recent may be reduced at the time; if of old standing it should be left alone. A forward dislocation, on the other hand, is much more troublesome, and may require the removal of the coccyx. A movable condition of the joints tends to recover spontaneously. It may be necessary to make the patient rest for a time, and afterwards to walk with a tight bandage across the pelvis.

(*d*) **Dyspareunia** (painful coitus) arises from a variety of causes. (1) The most frequent is a functional spasm of the sphincter vaginæ (vaginismus), often associated with a general neurotic state. In these circumstances the attempt to pass a speculum will sometimes elicit the same spasm, but may also be a means of cure. (2) Other local conditions should be carefully looked for, such as a vascular caruncle of the urethra, vulvitis, or vaginitis (see above). Fissures or small ulcers of the vulva or the anus may be causes of discomfort, which remain undiscovered for months and perhaps years. (3) Oophoritis or a prolapsed ovary may produce considerable pain on deep penetration. (4) Pelvic cellulitis or peritonitis (especially when associated with endocervicitis), and retention of foreign bodies. (5) Masturbation. (6) There may be, though this is relatively rare, a disproportion between the individuals concerned.

Prognosis and Treatment.—The condition of dyspareunia is apt to lead to considerable discomfort, not only to the individual, but to home life in general, and may have far-reaching consequences. When the physician is consulted he must make a very careful and minute examination of the vulva in a good light. Spasmodic contraction on touching the parts may give the diagnosis of some nervous or physical cause. If there is vaginismus, manual dilatation under anæsthesia, with the subsequent wearing of vaginal dilators in graduated sizes, cures most cases. Psychotherapeutic treatment and adjustment of marital relationships may be required in cases of neurosis. Cocaine ointment and suppositories and small doses of bromide are helpful. Childbirth frequently cures vaginismus.

§ 457. (I) **Backache.**—Pain in the back may accompany various chest diseases; for these see § 103. We are here concerned with the pain in the lumbar region which is so frequently complained of, especially by women. Chronic backache in women used to be attributed as a rule to some abnormal condition of the reproductive organs and the patients came under the care of the gynæcologist: a common condition, cervicitis, may have pain referred to the back. Since backache has been studied by the orthopædic surgeon, it has been found to be due most frequently to strain of the sacro-iliac, lumbo-sacral and other joints. The condition frequently started with undue stretching during labour. Badly balanced conditions of the spine contribute to the symptoms, which may be

confused with gynæcological lesions. These cases improve if treated orthopædically.

Physical Examination.—When the patient complains of backache, the physician should make a thorough examination of the region over which the pain is felt. For the adequate performance of this examination it is essential that the patient should be stripped. If the clothes are removed only so far as the waist, important physical phenomena may be overlooked. Note first whether there is any curvature of the spine, displacement, tumour, or redness. By palpation endeavour to make out the presence and position of any tenderness or swelling. Examine next the precise position of the pain; whether it is unilateral or bilateral; whether it is accompanied by tenderness or not; whether it is aggravated by the movements of certain muscles or joints or is accompanied by muscle spasm; whether it radiates along the course of any nerve. Examine the sacro-iliac joints and the costo-vertebral joints, and whether pressure over those joints elicits pain. An examination should be made next of the viscera; dilatation of the cæcum or a " dropped " colon may be present; vaginal and rectal examinations may reveal disorders in these regions. The urine may reveal kidney disease. In the absence of such causes, X-ray examination should be made. The history of the onset of pain, and of the concomitant symptoms at the time of the onset, may give important clues in the diagnosis.

Causes of Backache.—Sometimes the case belongs to the province of the gynæcologist, as in *Pressure pain* and *Bearing-down pain.*

(1) *Pressure pain* may occur as the result of *displacement of the uterus,* although this is infrequent unless some other complication is present. *Pelvic tumours* may also give rise to pain.

(2) *Bearing-down pain* may indicate uterine enlargement and displacement (§ 452), *e.g.*, retroflexion, retroversion and prolapse, or tumours pressing on the rectum. It is also frequent with inflammatory lesions in or near the uterus. Constipation is not an infrequent cause. Pain is present in advanced malignant conditions of the uterus.

(3) Backache occurs in many *acute diseases,* in most of the acute specific fevers, notably small-pox and influenza, and its cause is then recognised by pyrexia and other general symptoms.

(4) *Functional Causes.*—In nervous individuals, whose general health is below par, fatigue is usually evidenced by backache. It is frequently met with after childbirth, after infectious diseases, after operations, and often associated with constipation. The condition is mainly nutritional and is treated by attention to the diet, with cod liver oil or other vitamin preparations; gentle exercise, fresh air, regulation of the bowels, and calcium are beneficial. Suitable corsets may be necessary.

(5) Lumbago is known by: (i.) a history of a sudden onset, usually when stooping; (ii.) the pain is increased by movement of the lumbar muscles, and is relieved by local warmth; (iii.) tender nodules are palpable near the origin and insertion of the muscles affected.

(6) CURVATURE OF THE SPINE, whether it be due to Pott's disease or to simple lateral curvature, is a cause of backache. The later stages of Pott's disease (tuberculosis of the vertebræ) show an angular curvature and come under the notice chiefly of the orthopædic surgeon. The early stages are frequently overlooked, as no symptom may be present except pain. X-ray reveals the cause. Prolonged rest and general treatment as in other forms of tuberculosis are required. The slighter forms of lateral curvature are a frequent cause of backache, especially on standing. This cause of pain, especially in the early stages, is often missed, because of the neglect of the physician to examine the spine with the patient stripped.

(7) SECONDARY DEPOSITS, especially from carcinoma of the breast, and a SPINAL TUMOUR give intractable backache. X-ray examination is essential.

(8) **Sacro-iliac strain or subluxation** may be caused by a jerk when stepping off a kerb, or when stooping to lift a heavy object. Pain is felt on and off for a time, then is continuous and spreads over the buttock and leg. Symptoms: (i.) pain and tenderness over the joint is made out on palpation, or when the ilium is pressed inwards by the physician; (ii.) pain is elicited by flexing the thigh on the abdomen while the knee is fully flexed; (iii.) the patient sometimes stands on one leg, and may complain of pain passing down one sciatic nerve; (iv.) there is usually a history of strain. When a strain is present, rest the back and strap it for support; otherwise heat, massage, exercises and often manipulation are necessary.

(9) Pain low in the back may be due to **sacralisation**—*i.e.*, adhesions or ankylosis between the fifth lumbar vertebra transverse process and the sacrum. Rheumatic or other fibrositis compresses the nerve and sets up referred sciatica. Even neuritis, with muscular wasting, may be caused.

(10) **Osteo-arthritis** is known by: (i.) signs of the disease elsewhere; (ii.) the pain is made worse by coughing or sneezing; (iii.) it often radiates down the sciatic nerve.

(11) A DISPLACED INTERVERTEBRAL DISC (§ 825) is now a well-recognised cause.

(12) Backache may be due to disease connected with the KIDNEYS, such as perinephric abscess, pyelitis and pyelonephrosis, movable kidney, tumour and stone. An examination of the urine may first lead the physician to suspect the kidneys.

(13) Other ABDOMINAL TUMOURS, such as retroperitoneal sarcoma, aneurysm, and tumour of the spine, cancer of the stomach and rectum.

(14) GALL-STONES and a DUODENAL ULCER may rarely give rise to pain in the back before the pain works round to its usual position in front.

(15) **Spondylitis** or inflammation of the vertebral joints may be mentioned as a cause of backache: rarely it follows typhoid fever or syphilis. The "*typhoid spine*" appears a variable time after typhoid fever. There is pain and tenderness, sometimes starting pains along the nerves, occasionally paresis and wasting. The diagnosis from *polyneuritis* and *tuberculous disease of the vertebræ* is made by a positive Widal reaction, and albumin in the cerebro-spinal fluid. X-rays show osteo-periostitis. Kyphosis may result if the condition is not treated by rest.

(16) RECTAL disease and hæmorrhoids, in some cases.

Treatment.—Appropriate treatment can be carried out when the cause is known. When no obvious lesion is found, and especially with women who have borne children, a well-fitting belt or elastic corset is beneficial, especially when worn continuously, night and day: it should be kept in position by suspenders and shoulder straps and not end at the waist line. If no gynæcological cause can be found, orthopædic advice should be sought.

§ 458. (J) **Sterility** is that condition of a woman who under ordinary circumstances of reproduction, does not bring forth a living child. Natural sterility may be either

absolute or *relative.* In absolute sterility conception cannot take place without treatment; in relative, sometimes called "one-child sterility," a fœtus is cast off before viable or one child only is born. Whether a woman will be sterile or not is practically decided within three years of marriage; only 7 per cent. bear children after that time.

Etiology.—(1) In 25 per cent. of cases a single absolute cause will be found. Any condition causing dyspareunia or vaginismus, any deformity, mal-development, inflammatory condition, new growth, displacement or obstruction to coitus may result in failure to conceive. (2) 75 per cent. of infertile marriages are due to a totality of multiple infertility factors, which, in themselves, may be of little importance. If a fertile couple be investigated, one or two of these infertility factors will be found, but in an infertile couple the number varies between two and eight factors; investigation and treatment is thus directed to removing as many as possible of these, so that the fertility level of the sterile couple may be raised well above the threshold of conception. In an ordinary case with four or five infertility factors, the removal of two or three may result in conception occurring. Thus it follows that any one of several therapeutic measures may be successful.

Female Infertility Factors.—*Local* genital factors are the more common, such as (1) minor degrees of genital hypoplasia indicated by a relatively long cervix and a small firm uterine body; (2) hostile viscosity of the endo-cervical mucus; (3) tubal blockage; (4) absence of or mechanical impediments to ovulation such as small cystic ovaries; (5) uterine displacements. Retro-displacements reduce fertility, anteflexions generally form part of an endocrine factor. (6) Important *general* causes are endocrine deficiencies of the thyroid, anterior pituitary and ovary; (7) chronic intoxications, particularly focal, such as appendicitis and chronic cholecystitis; (8) dietetic errors such as insufficient protein and absence of fresh foods; (9) general debility and anæmia.

Investigation.—The female. This is best carried out in the mid-menstrual period (1) First enquire into the medical history:—(i.) General questions concerning age, duration of marriage, any history of past abdominal inflammation such as appendicitis, salpingitis or septic abortion. (ii.) Any discharge requires careful investigation. (iii.) Whether menstruation is normal, whether it started late, whether it is infrequent, excessive or small in amount. (iv.) Frequency of coitus. If more than twice a week, the spermatozoa may not have time to mature and fertilisation is less likely. (2) Next examine the wife: (i.) look for any evidence of endocrine disturbance suggesting thyroid deficiency, or virilism, and note the general development; (ii.) test the acidity of the vagina with nitrazene paper. About the mid-menstrual phase the pH of the vagina should be 4·5—higher acidity will need correction. (iii.) Examine the vaginal mucosa: if it is thick, rugose and pink, ovulation is probably occurring. When it has more the appearance of a vaginal mucosa at the menopause, then fertility is reduced as ovulation is less likely. (iv.) Examine the vaginal discharge: if it shows a semi-solid ground-rice appearance, it is indicative of hyper-acidity. Frank pus may be present the result of inflammation, most probably due to the trichomonas. There may be excessive mucus from the cervix. At the mid-menstrual phase this should be clear and watery; turbid mucus at this time indicates impaired fertility. At other times it is more gelatinous and turbid. The cervix may show an erosion. (3) Bi-manual examination may reveal gross abnormalities of the uterus but more likely a disturbance of the utero-cervical index only. If the examination is being carried out in the week preceding the period, a diagnostic scrape may be taken from the uterine cavity which will indicate the presence or absence of ovulation in that particular month. An isolated negative finding indicates the absence of ovulation that month only and is not proof of permanent absence of ovulation. Any tubal swelling would suggest tubal occlusion. (4) If nothing abnormal is detected the husband should be tested.

Male Infertility Factors are most often constitutional states producing relative deficiencies in the semen, such as general infective and toxic states, dietetic errors, obesity, endocrine disturbances and drug abuse. Local causes are less common.

Male infertility is present in at least one-third of all cases and in one-third of these again, aspermia will be present.

Investigation.—Two tests are necessary. (1) After four days' abstention a fresh specimen of semen is examined and should show in the region of one hundred million motile sperms per c.c., with less than 20 per cent. of abnormal heads. Less than sixty million or more than 20 per cent. abnormal heads indicates male infertility, but cases of pregnancy have occurred with much lower counts. (2) The post-coital test of Huhner. If live and active spermatozoa are found penetrating the cervical mucus two hours after coitus, the husband can be excluded.

Treatment.—I. If both parties, as a result of these investigations, appear to be normal and are under thirty, and if a reasonable time for conception to occur has not elapsed, give them some general advice and ask them to report in six months. (i.) Advise against too frequent coitus. (ii.) Likely days for ovulation to occur should be given, such as the fourteenth day prior to the onset of the period. (ii.) If there is a history of profluvium seminis, or the vaginal fornices are shallow, advise elevation of the buttocks after coitus.

II. Assuming the husband to be normal, the wife may have to be treated for (i.) Defective production of a normal ovum: (ii.) Obstruction to its entry to the tubes: (iii.) Failure of penetration of the spermatozoa and (iv.) Failure of the ovum to imbed. Any organic cause must be removed. In the VAGINA, vaginitis due to Trichomonas infestation is a frequent cause. A high acidity vagina with the typical ground-rice discharge and a low pH due to high œstrin activity should be treated with a sodium bicarbonate douche two hours before coitus. CERVICAL infection with erosions and cervicitis are very frequent causes of sterility. Dilatation and linear cauterisation are indicated. If thin watery mucus is not present in the cervical canal at the supposed time of ovulation it suggests deficient circulating œstrin. The treatment should consist of giving increasing doses of stilbœstrol from the end of the period until the thirteenth day of the cycle. If failure of ovulation is proved, synapoidin injections given from the tenth to the thirteenth day of the cycle inclusive, may induce ovulation.

UTERUS.—(*a*) If hypoplastic, stilbœstrol given following the period to the twelfth day with progestin later in the cycle, may be helpful. For failure of the ovum to imbed, curettage is indicated. (*b*) Tubal occlusion. Try repeated insufflations. Lipiodol injections will indicate the site of occlusion (this should not be carried out during the week following the period). (*c*) Endocrine disturbances, as indicated by obesity and amenorrhœa. Thyroid deficiency, indicated by a low basal metabolic rate must be dealt with. Thyroid given to the limit of tolerance if successful in reducing the weight and producing normal menstruation, is often followed by conception.

Artificial Insemination is indicated where coitus is impossible due to male deformity or where the cervical discharge is proved to be persistently lethal to the sperms. Careful attention to the health and hygiene of adolescents, with a view to preventing genital hypoplasia, the correct teaching of sex hygiene to avoid pelvic congestion, the eradication of venereal disease and the avoidance of useless local treatment such as curettage for discharge, will help to reduce considerably the number of infertile marriages.

CHAPTER XV

PYREXIA

MICROBIC DISEASES

WHEN a patient is suffering from some general or constitutional derangement, he complains of a vague "feeling of illness" (*i.e.*, malaise), or of "weakness" (debility, asthenia). He feels "generally" ill, and perhaps looks ill, but may be unable to mention any localising symptom, such as pain in the side or palpitation. Now, the first thing to do in such circumstances is to ascertain whether he is feverish or not, because all such conditions may be divided into two large clinical groups: A. **Debility with pyrexia,** which includes the Acute Specific Fevers and disorders in which there exists some localised inflammation; and B. **Debility without pyrexia,** which includes the different forms of Anæmia and various toxic and nutritional disorders. The latter will be dealt with in Chapter XVI. In this chapter we are concerned solely with the various conditions attended by elevation of the body temperature.

§ 465. **Definitions.**—The term **Acute Specific Fever** (or Specific Febrile Disease) has been applied to those fevers which are due to a specific or special poison, introduced into the body from without, and which run a definite course. If the poison was contracted from a previous case, but without contact with the patient, it was said to be an *Infectious* disease (*e.g.*, scarlatina); if the disease was produced only by actual contact with a person suffering from the malady, it was called *Contagious* (*e.g.*, syphilis); but these terms have always been used somewhat loosely and indifferently, and it would be better not to attempt any such distinction but to speak of them collectively as *Infective.* It would be out of place to enter here into the question of the nature of this poison; but there is direct or inferential proof in all the acute specific fevers that it is of microbic or parasitic origin. At first the organisms themselves were supposed to be the active agents of these diseases, but now in most cases the *causa vera* of the pyrexia and other symptoms is known to be a toxin or toxins which are produced by the microbe. This branch of knowledge has made enormous advances during the last three quarters of a century (*cf.* §§ 519 *et seq.*).

Bacteriology is dealt with in Chapter XXI. The chief **clinical characteristics** which cause us to suspect a disease of being **microbic in origin** are:

1. The occurrence of the disease in question in an *epidemic* form—*i.e.*, in the form of an outbreak, or as a series of cases which suggest that the patients contracted the disease either from one another or from a common source, the infection being conveyed to them through the air, the water or other ingesta, or by the bite of an insect.

2. Two features are common to all infective diseases: (i) *Pyrexia* is present at some time during the course [1]; and (ii.) in many cases the disease runs a more or less *definite course*—definite onset, gradual increase (fastigium) to an acme, defervescence, gradual or sudden, followed by complete restoration to health, or death.

3. The constant presence in the blood, tissues or excretions of the patient of a *microbe* or *protozoon*, which is not there normally.

[1] Some diseases have become so attenuated (*e.g.*, rubella and chicken-pox) that pyrexia may at times be absent, although most of the other clinical features are present.

The *pathological proof* that a particular microbe is causally related to the disease consists in applying certain experimental tests (see § 519).

4. The fact that the attack is more or less protective against subsequent infection.

Epidemic, Endemic, and Sporadic are terms by which it is usual to express the relative prevalence of infectious diseases. A disease is said to be *Epidemic* when a large number of cases arise by infection from a common source or from one another at one time, followed by an interval in which relatively few arise. Thus epidemics of measles, scarlet fever, and diphtheria arise in the Metropolis and elsewhere from time to time. A disease is said to be *Sporadic* when it occurs only in isolated cases. Thus we speak of a sporadic case of mumps when no other cases of it have been known to occur about the same time and in the same district. An *Endemic* disease is one which is constantly present in a certain district. Thus measles is endemic in London, malaria in Central Africa, and cholera in India.

PART A. SYMPTOMATOLOGY

§ 466. Pyrexia and Symptoms which may attend it.—Pyrexia may in some instances be unattended by any symptoms, but in nearly all cases the patient whose temperature is elevated complains of feeling "chilly," or he may have shivering or rigors; or perhaps he feels "burning hot." Headache, restlessness, and vague pains in the limbs and back are also common symptoms, in addition to the malaise or weakness. His skin is hot and dry to the touch, his pulse and respiration are rapid, his appetite is bad, tongue furred, and bowels constipated, his urine scanty and high coloured: in young children vomiting and convulsions may herald a pyrexial illness. In severe cases of fever there is great prostration, considerable mental dulness, and there may be delirium, or the "typhoid state." By these symptoms we suspect the presence of pyrexia, and the suspicion is confirmed, and the degree of fever ascertained, by the clinical thermometer (see below). Infective diseases pass through various STAGES which have many features in common: in severe cases, and often in association with high temperatures, RIGORS, DELIRIUM, and the "TYPHOID STATE" may occur.

§ 467. Incubation and other Stages of Acute Specific Fevers.—Particularly in epidemics, the infective or specific fevers conform to a common pattern and run a *definite course* (*e.g.*, measles). However, it must be remembered that the same organism may at times give rise to dissimilar diseases: *e.g.*, the same strain of hæmolytic streptococci may produce acute tonsillitis, scarlet-fever or puerperal fever in three different individuals.

It is a curious fact that a person does not develop the disease directly after he has been exposed to infection. The interval is called the stage of *incubation*. The patient is usually quite well during this stage, but there may be transient fever ("illness of infection") for a few hours after exposure. The incubation period varies in different diseases (Table XXIIA). During at least part of this time a healthy person who has been exposed to infection needs to be isolated ("placed in quarantine"), to see if he will develop the disease. A glance at the first column in the table will show that a period of THREE WEEKS will cover the incubation of all the

TABLE XXIIA.—SHOWING INCUBATION, DATE OF ERUPTION, AND DURATION OF INFECTION OF THE PRINCIPAL INFECTIVE DISORDERS.

DISEASE.	INCUBATION PERIOD.	DAY OF DISEASE ON WHICH RASH APPEARS.	INFECTIOUS PERIOD, or period during which the *patient* need be isolated.
Varicella.	10 to 21 days, average 14.	The rash is usually the 1st symptom noticed.	Till all scabs have separated, or 14 days, whichever is the shorter.
Scarlet Fever.	2 to 4 days, average 2½.	**1st** or **2nd**.	From commencement of illness till an indeterminate date, which varies in different cases. Average 4 weeks. Rhinorrhœa, and possibly otorrhœa, may retain infection for 6 months or more.
Small-pox.	12 days.	**3rd.**	From commencement till not a trace left of scabs or desquamation. Most virulent during vesiculation, pustulation, and scabbing. 3 to 8 weeks.
Measles.	7 to 14 days, average 10.	**4th.**	*Great* in early period *before* rash out Till rash has faded: usually 1 week after rash appears.
Rubella.	14 to 19 days.	**1st to 4th.**	5 to 6 days from commencement.
Typhus.	12 to 14 days.	**4th or 5th.**	Probably 3 to 4 weeks.
Typhoid and Paratyphoid.	8 to 21 days, usually 10 to 14.	Average **2nd week.**	Several weeks after pyrexia has ceased. "Carriers" may retain their infection for many years.
Dengue.	2 to 6 days.	Initial rash 1st day. Terminal rash 4th.	
Diphtheria.	1 to 6 days, usually 2 to 4.	None.	Until 3 swabs from nose and throat, and any ear discharge, fail to grow the organism.

The period of incubation of the other microbic disorders so far as we know is given approximately below. This is important, as the duration of quarantine depends on the period of incubation. In cases with a relatively long incubation period, such as mumps, chickenpox, measles and rubella, it is not necessary to isolate contacts for the first week after exposure.

Malaria, 12 hours and upwards.
Erysipelas, 1 to 7 days.
Cerebro-spinal fever, 1 to 3 days.
Influenza, 1 to 3 days.
Pneumonia, 1 to 3 days.
Anthrax, 2 or 3 days.
Gonorrhœa, 2 or 3 days.
Plague, 3 to 7 days.
Glanders, 3 to 18 days.
Tetanus, usually 3 to 21 days.
Mumps, 3 to 28 days (average 17).
Relapsing fever, 4 to 10 days.
Glandular fever, 5 to 12 days.
Whooping-cough, 7 to 14 days.
Malta fever, about 9 days.
Cholera, under 14 days.
Yellow fever, under 18 days.
Syphilis, 15 to 25 days.
Hydrophobia, 40 days or more.
Tuberculosis, probably some weeks.
Infective Hepatitis, 17 to 35 days.

eruptive fevers. The actual *invasion* or development of the symptoms of the disease is usually more or less abrupt, except in typhoid fever, whooping-cough, and sometimes measles. *Prodromal symptoms* at the onset of the disease proper may indicate that a disease is commencing, but not permit an exact diagnosis. An *eruption* appears upon the skin within the next four days (except in typhoid fever) in those diseases which develop a rash, and which are called on that account the EXANTHEMATA. (Enanthemata are the lesions seen on the mucous membranes). The fever and other symptoms go on increasing until the *acme* is reached. *Remissions* indicate temporary diminution of symptoms, and *recrudescences* aggravation of the disease. Finally, the last stage—the stage of *defervescence* supervenes, and gradually the patient convalesces unless a *relapse* occurs.

§ 468. **Rigors** often indicate the sudden onset of pyrexia. A rigor is an attack of shivering attended by elevation of temperature and great acceleration of pulse rate, rapidly followed (usually) by sweating and a fall in the temperature. Such an attack may vary widely in severity from a simple feeling of "chilliness down the back, like cold water," to a shaking of the whole body, so that the patient shakes the bed beneath him. Severe rigors occur typically and *regularly* in the course of malaria, also at frequent but *irregular* intervals throughout the course of septicæmia and pyæmia. In childhood, rigors are often replaced by convulsions.

1. First, ascertain that the shivering is not of purely nervous origin, because a trembling much resembling a rigor may occur as the result of pure fright or from slighter causes in nervous people.

2. Procure, if possible, a series of temperature records, because rigors occur in association with several conditions which can only be differentiated in this way.

Causes.—The causes of rigors are very numerous, but they are best approached in a general way as follows:

(*a*) Coming on in a person *previously healthy*, one should always suspect the advent of some acute illness. In children the eruptive fevers are sometimes ushered in with either convulsions or rigors. In adults, septicæmia, pneumonia, pyæmia, peritonitis, the eruptive fevers, malaria or influenza may be suspected.

(*b*) *Septic Infection.*—When rigors *supervene in the course of an illness* of any kind, abscess or pent-up pus in some position should always be the first thing thought of. *Before the days of the thermometer the doctor used to rely upon shivering and sweating as an infallible indication of the formation of pus.* For instance, in a case of pleurisy with an effusion, which has hitherto been serous, the occurrence of shivering indicates that the contents of the chest have become purulent (empyema). Similarly, a rigor occurring with otitis media suggests extension to the mastoid cells, or may point to lateral sinus thrombosis. Rigors occurring in a case of cardio-valvular disease indicate the occurrence of infected emboli, or the supervention of malignant endocarditis. Shiverings and sweatings may

occur during the course of tuberculosis and many other conditions mentioned under the Causes of Intermitting Pyrexia (§ 509). If no obvious cause for an attack of shivering appears, we may suspect some internal suppuration, such as appendicitis, or ulceration in some part of the urinary, biliary, or alimentary canals. If the rigor is due to a collection of pus, there will be found a definite leucocytosis.

(c) The *passing of a catheter* is often followed by a severe rigor, and sometimes the temperature goes suddenly up to 105° or 106° F., and as suddenly falls again. Sudden obstruction in the biliary or renal passages is often attended by rigors, followed by a feeling of heat and sweating, and the temperature may go up to 105° F ; these examples are probably due to bacterial invasion through minute abrasions. A rigor, too, may be set up by the intravenous injection of some chemical substance (*e.g.*, neoarsphenamine) or a therapeutic serum, such as diphtheria antitoxin, or after blood transfusion. Therapeutic use is made of this by injecting T.A.B. vaccine, sulphur and foreign proteins to produce pyrexia.

(d) *Neurasthenic* and *hysterical* patients often have shivering attacks, without pyrexia. An attack of shivering may also constitute a symptom of *vaso-motor disorder*. Thus it is a symptom of the reaction which follows, and often forms part of the " flush-storms " chiefly met with at the climacteric, without elevation of temperature.

(e) *Cholecystitis* may cause short attacks of shivering without pyrexia.

The *Prognosis* and *Treatment* belong to the several causal conditions, but in any case the patient should be kept warm in bed with a hot-water bottle to his feet: aspirin, bromide or morphia will soothe the nervous system.

§ **469. Delirium,** or incoherence of thought, is another symptom which frequently accompanies pyrexia. The older authors used to describe three varieties of delirium: (1) Delirium ferox, in which the patient is very violent and maniacal; (2) typhoid delirium, in which the patient lies on his back muttering, with subsultus tendinum ; (3) delirium tremens, in which there is great sleeplessness, hallucinations and tremors, not necessarily due to alcohol. The nature of the delirium is not always constant in any given disease. For clinical purposes, the *causes of delirium* may be divided into two groups—FEBRILE and NON-FEBRILE. It is important, therefore, to take the temperature at once in every case of delirium. Alcoholic subjects and children, especially if neurotic, are predisposed to delirium when attacked with only slight fever.

a. Febrile Delirium may arise under four circumstances:

1. ACUTE LOCAL INFLAMMATION in some part of the body, such as pneumonia. It is advisable, therefore, to examine all the organs.

2. DISEASES OF THE BRAIN (Encephalitis), or OF THE MENINGES, such as tuberculous meningitis. The latter is accompanied by headache, vomiting, retraction of the head, intolerance of light, and paralysis of cranial nerves.

3. All the ACUTE SPECIFIC FEVERS are liable to be accompanied by

delirium. The tendency, however, varies considerably, though it is usually directly related to the height of the temperature and the nervous stability of the individual. It is important to bear this in mind, because, as a prognostic indication, delirium occurring in a disease like measles or acute rheumatism, in which it is rare, has a much more serious meaning than when it occurs in pneumonia, for instance, where it is more usual (see Table XXIII).

4. Certain cases of DELIRIUM TREMENS of a SEVERE KIND are accompanied by an elevation of temperature. Indeed, the prognosis in this affection may largely depend upon the temperature. We must be careful to exclude local inflammations in such cases, for they are apt to come on very insidiously. In the worst cases of ACUTE DELIRIOUS MANIA also the temperature may be considerably elevated (see *b* 6, below).

TABLE XXIII.—SHOWING THE RELATIVE FREQUENCY OF DELIRIUM IN THE VARIOUS INFECTIVE FEVERS.

Frequent in—	*Occasional in—*	*Rare in—*
Confluent Small-pox	Remittent Fever	Influenza
Typhus	Yellow Fever	Mumps
Lobar Pneumonia	Small-pox (modified)	Dysentery
Typhoid Fever (after 1st week)	Measles	Cholera
Meningitis	Relapsing Fever	Acute Rheumatism
Encephalitis	Scarlet Fever	Diphtheria
Erysipelas	Malaria	Rubella
Plague		Varicella
Malignant Endocarditis		
Septicæmia		

b. Non-febrile Delirium may arise under six conditions:

1. DELIRIUM TREMENS (Delirium e Potu) is, as just mentioned, usually unattended by a rise of temperature, and is undoubtedly the commonest cause of non-febrile delirium. It is recognised by the history, the muscular tremors, sleeplessness, and the characteristic hallucinations.

2. CHRONIC RENAL DISEASE, and especially chronic interstitial nephritis, gives rise in its advanced stages to a muttering delirium or incoherence, which thus becomes a symptom of the gravest import, and generally heralds coma and death. The delirium is due to uræmia, and occurs in other renal diseases.

3. POST-FEBRILE DELIRIUM (Post-Febrile Mania).—During the convalescence of pneumonia and other exhausting diseases, especially such as run a protracted course, and have been attended with a high degree of pyrexia, mental symptoms may develop. These symptoms, which usually make their appearance without any warning, give great uneasiness to the friends. Nevertheless, by means of good food, tonics, and fresh air, such mental symptoms will entirely disappear. Before venturing on a prognosis, however, inquiry should always be made for any family history of mental disease, for a hereditary tendency greatly

lessens the chance of recovery. The condition is recognised by the history of the previous malady. Sometimes the mental derangement consists simply of loss of memory, especially for the names of persons and things, but more often the mind " wanders " and there are delusions.

4. Reflex Delirium.—Trousseau mentioned cases of children with intestinal worms who had delirium, and described several cases which were caused by tickling the soles of the feet. The transient delirium connected with the severe pain of childbirth is possibly of the same nature.

5. Deliriant Drugs should always be suspected when delirium develops suddenly in a person in health, especially children in the country, in the absence of any of the foregoing causes. The most important are belladonna, hyoscyamus, hyoscine, cannabis indica, stramonium, and others of the solanaceæ, antipyrin, camphor in rare cases, œnanthe crocata, cocculus indicus (with which beer used to be adulterated), poisonous fungi, and sometimes salicylic acid and its salts, especially if adulterated, when given in large doses. Delirium may ensue when a patient is recovering from the effects of poisonous gases. Morphia in some people invariably produces delirium.

6. Acute Mania sometimes comes on very suddenly, and only differs from " delirium ferox " or maniacal delirium in not being referable to some bodily disease or toxæmia. Delirium occurs in the advanced stage of many mental diseases. We identify these conditions by (1) the temperature is not as a rule elevated; (2) it affects a person previously in good health; and (3) the exclusion of any organic lesion by a careful examination of the nervous and other systems. As regards the temperature there is an exception in the rare and serious condition known as " acute delirious mania," in which marked pyrexia is present (and see § 893).

Prognosis.—Febrile delirium is not necessarily a grave symptom when it is associated with a *disease in which its occurrence is usual*—*e.g.*, pneumonia—and especially when the cause is only temporary; but its presence adds considerably to the gravity of a case if the occurrence of delirium is unusual (see Table XXIII), for it indicates a very severe attack, or the occurrence of complications, or both. *Non-febrile* delirium is a grave symptom in chronic renal disease. The prognosis is serious as regards mental recovery in all patients who have a hereditary tendency to mental disorder. In acute mania the prognosis is grave.

Treatment.—It is necessary to provide a nurse or attendant, and restraint may be called for. *Remedial Treatment.*—An ice-bag to the head for an intracranial inflammation; good nourishing food for mania and post-febrile delirium; a brisk purge for uræmia. *Symptomatic treatment* consists of the administration of sedatives, such as somnifaine, hexobarbitone (evipan), nembutal, chloral, calcibronat, the bromides and paraldehyde (injected). Opium and morphia require caution, especially if there is liver disease. In delirium tremens, it is most helpful in some cases by procuring sleep, but in others it only aggravates the maniacal condition. Periodical sponging with cold or ice-cold water often has a

steadying effect. In post-febrile delirium and other conditions where the brain is suffering from malnutrition, opium in small doses is a most valuable remedy, and may be given without fear if the liver and kidneys are healthy.

§ 470. **The Typhoid State** may be described as a condition of semi-consciousness or unconsciousness (coma) attended by elevation of temperature and muttering delirium, due to toxæmia. The name of this condition was derived from its frequent association with typhus, but it is met in many other fevers. With reference to the question of pyrexia, it should be stated that the comatose condition, due to renal disease (uræmia), advanced liver disease (cholæmia), and various poisons (particularly opium), has sometimes been described as the typhoid state, but these are apyrexial conditions, and it is preferable to include only those with pyrexia. In short, the typhoid state corresponds clinically to a state of coma *plus* pyrexia and muttering delirium.

Symptoms.—The typhoid state is always secondary to some febrile condition, in the course of which it arises: the height of the temperature and its persistence depend chiefly upon the nature of the primary disease. The first *mental symptom* usually noticed is sleeplessness with delirium, generally of the muttering variety, but by and by stupor supervenes, which gradually deepens. The mental faculties are obscured, but the unconsciousness is not always so complete as one would imagine. The profound disturbance of the nervous system is evidenced by prostration, restlessness, subsultus tendinum (muscular twitchings), floccitatio or carphology (picking at the bedclothes), unconscious evacuation of bladder and bowels, and, in extreme cases, convulsions. The *physical condition* is indicated by the pale and often cyanosed colour: the tongue is dry, brown, furred and tremulous: and sordes collects upon the lips and teeth. The pulse is rapid, feeble, and irregular, and the heart-sounds distant. The respiration is usually rapid, but shallow. The pupils are dilated, but the patient does not see. Nevertheless, he looks about at imaginary objects—"coma vigil." Dysphagia, diarrhœa and stertorous breathing are very serious indications of profound stupor.

Diagnosis.—(1) The "*typhoid state,*" as above mentioned, may be distinguished from *coma* by the presence of pyrexia, and the absence of evidences of renal or liver disease, apoplexy, or other cause of the coma. (2) Certain acute *inflammations of the brain and meninges* are attended by pyrexia, and offer considerable difficulty—particularly with tuberculous meningitis (§ 727). The presence of papillœdema, head retraction, paralysis of the cranial nerves on the one hand, and the signs of the primary malady which has produced the typhoid condition on the other, are evidences upon which we can rely in many instances.

Causes.—Patients with an alcoholic history or with chronic nephritis are predisposed to the development of the typhoid state.

1. The Acute Infectious Fevers are the commonest causes, and particularly typhoid and typhus fevers. The Typhoid State occurs as an ordinary symptom of a grave attack in the course of these two diseases

and in some others (see Table XXIV). In another group of diseases it occurs only occasionally, and in others it is rare. If it arises in either of these latter groups, it indicates either (1) a very severe variety of the disease, or (2) some serious complications; and, in any case, that the patient is likely to die.

2. Certain other INFLAMMATORY OR INFECTIVE DISORDERS with local manifestations may be attended by the typhoid state, such as acute lobar pneumonia, acute pulmonary tuberculosis, ulcerative endocarditis, acute meningitis, and encephalitis lethargica.

3. Certain acute IDIOPATHIC DISEASES may, in rare instances, be attended by the typhoid state, such as acute gout and very intense forms of delirium tremens. It is extremely rare in acute rheumatism, unless accompanied by peri- or endo-carditis.

TABLE XXIV.—RELATIVE FREQUENCY OF THE TYPHOID STATE IN DIFFERENT DISEASES. ALCOHOLIC SUBJECTS AND PATIENTS WITH CHRONIC NEPHRITIS ARE PREDISPOSED TO THE TYPHOID STATE.

Frequently met with, especially towards the end, in—	*Occasionally met with in—*	*Rare in—*
Typhoid (Enteric) Fever	Scarlet Fever	Cholera
Typhus	Measles with broncho-pneumonia	Variola (modified)
Confluent Small-pox (unmodified)	Cerebro-Spinal Fever	Dysentery
Erysipelas (severe)	Anthrax (Internal)	Malaria
Septicæmia (including Malignant Endocarditis and Osteomyelitis)	Remittent Fever	Relapsing Fever
Meningitis—especially tuberculous	Undulant (Malta) Fever	Acute Rheumatism
Encephalitis, especially E. lethargica		
Lobar Pneumonia		
Acute Miliary Tuberculosis		
Acute Glanders		
Acute Anthrax		
Remittent Fever		
Cerebral and Hæmorrhagic Malaria		
Yellow Fever		
Plague		

Diagnosis of the Cause.—The clinical investigation should be conducted on the same lines as in cases of pyrexia. Is it due to *local* or *generalised* inflammation? First, every organ in the body should be thoroughly examined so as to exclude local disorders. Secondly, we proceed to the diagnosis of the general fevers from one another, and, if possible, obtain a series of temperature records. In cases where the cause of the typhoid condition is obscure, septicæmia, especially with endocardial involvement, should always be suspected, and its origin carefully sought.

Prognosis.—The typhoid state, like delirium, has a less serious import in diseases such as typhoid fever, in which it is frequently met with. But it is always a grave condition, and indicates profound cerebral and general toxæmia. Occurring in the course of scarlet fever, erysipelas, or measles, it often indicates pulmonary or cardiac complications, and is proportionately serious. As regards symptoms, the profundity of the stupor is a measure of the intensity of the toxæmia, and dysphagia, uncontrolled diarrhœa, stertor, or convulsions are generally lethal signs.

The Treatment of a condition such as this arising in the course of so many diseases must necessarily vary, and our first duty is *to ascertain what disease is in operation.* The toxæmia is partly bacterial and partly the result of disordered metabolism and elimination. The indications are (1) to eliminate the toxins by diuretics, diaphoretics, and aperients; and (2) to stimulate and support the patient's strength by nutriment and stimulants. The use of alcohol in the treatment of fevers as in other branches of medicine has of late years considerably declined. As regards symptomatic treatment, if the delirium be very violent, sedatives such as chloral or bromide, in large doses, even up to 40 grains of each, are indicated. Opium should be avoided, as it prevents the elimination of the poison. For the treatment of Hyperpyrexia, see § 524.

PART B. PHYSICAL EXAMINATION

The clinical investigation of pyrexial disorders consists of (1) CLINICAL THERMOMETRY; (2) AN EXAMINATION OF THE ORGANS; and (3) BACTERIOLOGICAL INVESTIGATION.

§ 471. **Clinical Thermometry and Types of Pyrexia.**—The temperature is ascertained by means of the clinical thermometer: readings are usually taken in the mouth or the axilla. Mouth temperatures must be taken before meals, for food, drinks or mouth breathing cause false readings: a half-minute thermometer must be kept in the mouth for at least one minute, and in the axilla for ten minutes, to give accurate records. The temperature may also be taken in the rectum, where it may be ½° to 1° higher than in the mouth. In children the thermometer may be held in the groin, the thigh being flexed to the abdomen for the purpose. The normal temperature of the body varies between about 97·8° and 99° F.; average 98·4° F. It is lowest about 4 A.M. and highest about 8 P.M. It tends to be lower in old age and higher in infancy, especially after an attack of crying. The temperature is often subnormal after a loss of blood, during convalescence, in cardiac failure, and in all states of collapse. The latter is sometimes the direct result of toxæmia.

A temperature of 100° is regarded as slight fever.
" " 102° " moderate fever.
" " 104° " high fever.
" " 105° and upwards is regarded as hyperpyrexia.

THE TEMPERATURE CHART.—*Very little information can be derived from a single observation of a patient's temperature, and in all cases of pyrexia one must know the course which it runs from day to day and hour to hour.* In most cases of fever it is hardly possible to come to any conclusion without seeing a "chart" of the case—*i.e.*, a series of records. In all cases of pyrexia the temperature should be taken **and recorded** morning and evening; and in all acute cases it should be taken four-hourly. In cases of suspected tuberculosis and some other affections it may be advisable to obtain hourly records throughout the day, otherwise slight eleva-

tions may be missed. The pulse, respiration and blood pressure should also be observed, especially in abdominal inflammations, extensive broncho-pneumonia, and after severe attacks of diphtheria, where the temperature alone does not give us a true idea of the amount of mischief which is going on. In broncho-pneumonia the rapidity of respiration is often the most reliable indication. The onset of pyrexia may be gradual, as in typhoid fever or diphtheria, but more often it is sudden and may be accompanied by a rigor, as is sometimes seen in pneumonia or small-pox. Remember that the *onset is apt to be very sudden* in scarlatina, erysipelas and small-pox; it is *gradual* (taking perhaps two or three days) in measles, typhoid fever and pertussis. During the next few days the temperature

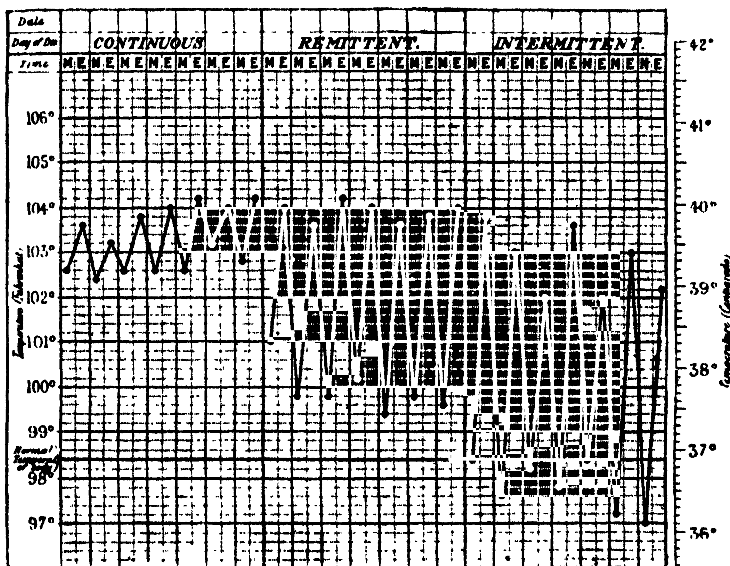


FIG. 110.—TYPES OF PYREXIA.—Continuous pyrexia showing only the normal variations in the morning and evening. Remittent pyrexia showing a drop of several degrees each day. Intermittent pyrexia where the temperature comes down to normal at some time every day.

generally increases until the *acme* is reached. The termination may be gradual, when it is said to terminate by *lysis*, as in typhoid; or pyrexia may terminate suddenly by *crisis*, as in some cases of lobar pneumonia and relapsing fever.

Types of Pyrexia.—In the absence of any eruption, the COURSE OF THE TEMPERATURE is our best, and may be our only, guide. It is usual to describe three types of pyrexia, according to the course which the temperature pursues from day to day (Fig. 110); (i.) *Continued or Continuous Fever*, where the temperature remains elevated for a considerable period, and where the *diurnal variation often does not exceed the normal diurnal variation*—viz., one, or at most one and a half degrees; (ii.) *Remittent Pyrexia*, when the diurnal variation is greater than the normal diurnal

variation, but where the temperature never comes down quite to normal; (iii.) *Intermittent Pyrexia*, where the temperature at some time of the day is normal or subnormal, and at another time of the day, usually in the evening, it is raised one, two, or more degrees. But for clinical purposes the two latter may be grouped together, and thus we have TWO GROUPS of fevers—one in which the pyrexia is practically CONTINUOUS, and another in which there is a remission, or INTERMISSION, once or oftener during the twenty-four hours, usually in the morning.

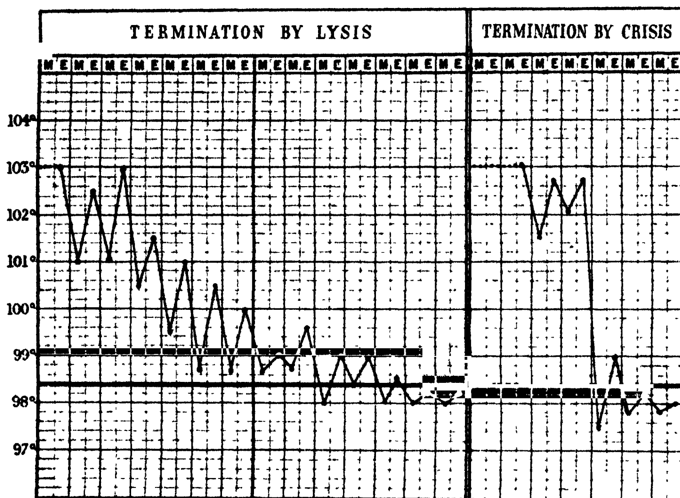


FIG. 111.—TERMINATION OF PYREXIA BY LYSIS AND CRISIS.

The following are useful facts to remember regarding temperatures: (i.) The sudden advent of high fever in a previously healthy person without other symptoms indicates, in England, Scarlet Fever, Influenza, Small-pox, or Erysipelas, and sometimes Pneumonia. A very gradual advent is suggestive of Typhoid Fever. (ii.) A fresh rise after the temperature has begun to fall indicates a complication or a relapse. (iii.) A sudden fall in the course of a fever (especially Typhoid Fever) may indicate internal hæmorrhage, perforation of one of the viscera, or profuse diarrhœa. (iv.) A considerable rise in diseases usually non-febrile, such as tetanus, delirium tremens, cholera, cancer, epilepsy, apoplexy, etc., generally indicates a fatal termination.

§ 472. Subnormal Temperature.—The temperature of the surface of the body, as indicated in the mouth or axilla, is rarely more than one or two degrees below normal. When it is below 96° the condition usually amounts to collapse. Subnormal temperature is not so important, for purposes of diagnosis, as elevation of temperature; but in the first four instances given below it may aid us in their differentiation. Subnormal temperature adds to the gravity of the prognosis in most wasting disorders. With regard to treatment, temperature readings below the normal are indications for the administration of stimulants, nourishment, and the application of external warmth.

Causes.—1. Subnormal temperature as an indication of *lowered vitality* occurs in normal circumstances in the aged, in whom the temperature is habitually several fractions of a degree below normal.

2. The temperature drops suddenly in *internal hæmorrhage* and in *abdominal rupture* or *perforation*: rupture of an abdominal cyst, traumatic rupture of the liver, spleen or kidney, or perforation of a peptic ulcer or of an ulcer of the bowel are usually attended by other and more distinctive signs (§ 243). In typhoid fever this sudden fall may be the only indication of these serious complications.

3. In all severe *abdominal inflammations* prostration and collapse are marked features, and the temperature may in some cases be subnormal, although there may be considerable constitutional disturbance, as shown by the prostration, and the rapid pulse (§ 239).

4. Subnormal temperature occurs in several other disorders in which it is not of much diagnostic significance, because we depend upon other signs for their identification. Thus, the temperature of the body is lowered (i.) when there is an excessive withdrawal of heat from the body, as in cases of inanition or exposure combined with privation, or with extensive weeping skin eruptions; or when large quantities of fluid are evacuated, as in severe diarrhœa or cholera (when the temperature may be 90° in axilla, though 105° in rectum); (ii.) in states of inanition or cachexia—*e.g.*, during convalescence from fevers, Addison's disease, cancer (especially of the alimentary canal), diabetes, and chronic mental disorders; (iii.) when there is deficient oxygenation, as in cases of congenital heart disease, cardiac failure, alcoholism, jaundice, uræmia, and myxœdema; (iv.) in some diseases of the central nervous system, such as tuberculous meningitis, the onset of cerebral hæmorrhage, or cerebral tumour; and (v.) in poisoning by phosphorus, morphia, phenol, and other drugs.

5. In all states of COLLAPSE the temperature is considerably lowered (2° or more). Indeed, this is one of the chief means by which it may be distinguished from syncope.

§ 473. **Examination of Organs.**—All the viscera must be carefully examined in accordance with the Scheme of Case-taking, pp. 6 and 7, so that local causes for the pyrexia may be excluded. Examination of the urine or the stools may reveal an unsuspected cause of pyrexia. For *clinical* purposes there are two large groups of causes of pyrexia: (*a*) **local inflammation**: such as pleurisy, appendicitis, abscess of the liver, etc., and (*b*) **general bacteræmic** or **toxæmic conditions**, like scarlet fever, rheumatic fever, and streptococcal or coli infection.

If any local inflammation is found, turn to the chapter dealing with the disease of that part. But it must still be remembered that some constitutional disease (*e.g.*, some specific fever) *may* be present, of which the local disease is a complication. Thus pneumonia, which would be discovered in the course of our examination, is a frequent complication of typhoid fever; and endocarditis of rheumatic fever. There are two features which may lead us to suspect a combination of disorders such as this: (1) The signs and symptoms of the local disorder may be of an aberrant type (*e.g.*, see Aberrant Types of Pneumonia, § 122); and (2) the constitutional disturbance presented by the patient would be greater in degree or different in kind than would accompany the local disease if it were the only disease present.

§ 474. **The Examination of the Blood** often affords most valuable information, and it may be useful to make a complete blood-count or stain a film (§§ 530 and 531), to take blood for the purpose of culture or to determine the Widal and Wassermann reactions (§§ 922 and 924). In certain cases the sedimentation rate test (§ 927) is also useful.

PART C. THE DIAGNOSIS, PROGNOSIS, AND TREATMENT OF PYREXIAL DISORDERS

§ 475. **Routine Procedure and Classification.**—In cases of pyrexia we must investigate, as in other cases, three points:

First, THE LEADING SYMPTOM complained of by the patient will be one or more of those mentioned in §466.

Secondly, THE HISTORY OF THE ILLNESS. The *date* when the symptoms commenced—*i.e.*, the PRECISE DURATION OF THE ILLNESS—is a most important matter. A few of the fevers—*e.g.*, typhoid fever and diphtheria—commence insidiously; but the majority are ushered in suddenly, very often with an attack of shivering (a rigor). Throughout the entire course of every case of fever the physician should have constantly in mind the "day of the disease,"[1] so that he may know what events to expect at that particular period of the case. In typhoid fever, for instance, on the fourteenth day, or a little later, the diurnal range of the temperature should commence to be more marked, and during the next few days special care should be exercised to avoid hæmorrhage or perforation.

Thirdly, THE EXAMINATION OF THE PATIENT comprises three important matters: (1) Physical examination; (2) is there, or has there been, an eruption? and (3) the temperature and its course.

(1) EVERY ORGAN must be systematically examined (Scheme of Case-taking, pp. 6 and 7), and as carefully and thoroughly as the patient's condition will allow, in order that we may DETECT OR EXCLUDE ANY LOCAL DISEASE. This is important, because all cases of pyrexia are associated with or due to some **local inflammatory disease,** or some **generalised febrile disorder** (*e.g.*, **typhoid fever**), or both.

(2) WHETHER THERE IS OR HAS BEEN ANY ERUPTION is the next question. The first of the groups (*vide infra*) into which all fevers may be divided comprises those in which a rash distinctive of the disease appears within the first four days (with one exception) after the illness. The day on which it appears in each disease should always be in mind (Table XXIIA).

(3) THE TEMPERATURE **and its course** is the next thing to investigate; and it is of the greatest importance to obtain a CHART or succession of readings, after the manner described in § 471. The DURATION of the fever is of assistance in diagnosis, especially when it has lasted longer than two or three weeks.[2]

The **classification** of pyrexial disorders may conveniently be based upon

[1] The fourth day of a disease is the third day *after* its commencement. Thus the eruption of measles appears on the fourth day, and, supposing the patient were taken ill on a Monday, the eruption would appear on Thursday.

[2] Excluding diphtheria and the exanthemata, it is found that the majority of short fevers, of a few days' duration, are due to "common colds," "rheumatism," "constipation," and "influenza." "Colds," including bronchitis, influenza, tonsillitis and pharyngitis, 4,164; acute appendicitis, 1,504; acute arthritis, 1,016; salpingitis, 871; pneumonia, 803; lymphangitis, 365; sinusitis, 259; erysipelas, 241; poliomyelitis, 227.—R. C. Cabot, "Differential Diagnosis." London, 1919.

the results of our examination—namely, the eruption, if present, and the course of the temperature.

GROUP I.—ACUTE EXANTHEMATA OR ERUPTIVE FEVERS—*i.e.*, fevers which are characterised by AN ERUPTION (*i.e.*, a RASH) distinctive of each disease appearing on one of the first four days of the illness (§ 476).

GROUP II.—CONTINUED FEVERS—*i.e.*, fevers in which the temperature runs a more or less continuous course, and which present NO ERUPTION during the first four days (§ 492).

GROUP III.—INTERMITTENT FEVERS—*i.e.*, fevers in which the temperature runs an intermittent (or remittent) course, and which present NO ERUPTION (§ 509).

If the physical examination reveals signs of disease of some particular organ, reference should be made to § 473, and to the chapter on diseases of that organ.

GROUP I. THE ACUTE EXANTHEMATA OR ERUPTIVE FEVERS

In all the diseases in this group the onset of the pyrexia is more or less abrupt, and in the majority a well-marked GENERAL ERUPTION appears during the *first four days* of the illness.[1] The course of the pyrexia varies considerably in the disorders in this group.

Common.		*Rare.*	
I. Chicken-pox (first day) ..	§ 476	VIII. Dengue (first day) ..	§ 483
II. Scarlet fever (second day)	§ 477	IX. Classical Typhus (fourth or fifth day)	§ 484
III. Erysipelas (second day) ..	§ 478	X. Rocky Mountain fever	§ 485
IV. Small-pox (third day) ..	§ 479	XI. Scrub typhus (rash fifth to seventh day)	§ 486
V. Measles (fourth day) ..	§ 481	XII. Q Fever	§ 487
VI. Rubella (first to fourth day)	§ 482	XIII. Trench Fever	§ 488
VII. Typhoid fever (usually eighth to tenth day), influenza, cerebro-spinal meningitis, plague, and other members of Group II, occasionally present early rashes.	§ 493	XIV. Rickettsial Pox	§ 489
		XV. Anthrax	§ 490
		XVI. Acute glanders	§ 491

In each of the acute exanthemata the ERUPTION has special and DISTINCTIVE CHARACTERS, which, together with the DAY OF THE DISEASE on which the eruption appears, may enable one to differentiate the members of this group from one another. SCARLET FEVER may be regarded as the type, but it will be convenient to take them in the order in which the eruption appears. TYPHUS is hardly ever seen, and DENGUE is not met with in England. ANTHRAX and GLANDERS are, like hydrophobia, derived from animals.

Some DRUGS IN COMMON USE may give rashes and pyrexia ("drug fever"): common examples are the sulphonamides and the barbiturates which at times can mimic the eruptive fevers closely.

§ 476. I. Varicella (synonym: **Chicken-Pox**) may be defined as an

[1] Incomplete forms (formes frustes), in which the rash or other characteristic symptoms are absent, may occur especially during an epidemic.

acute contagious disease, manifested by an eruption of successive crops of limpid vesicles, usually accompanied by slight exacerbations of fever. It is in most cases a trivial disorder of childhood. It was differentiated from small-pox by Heberden in 1767, but its autonomy was disputed for nearly a hundred years later.

Symptoms.—Especially in young children the characteristic rash is generally the first sign noticed. In older children and in adults *prodromal symptoms* precede this rash for the first twelve to twenty-four hours and give rise to a temperature even to 101°–102°, malaise, headache, backache and sometimes a prodromal scarlatiniform, morbilliform or urticarial rash. In any case within twenty-four hours the *characteristic eruption* appears: this consists of dark pink, slightly raised, ovoid, or somewhat pyramidal papules, which in the course of a few hours become vesicular. The typical vesicle is at first a thin-walled, translucent, unilocular, glistening bleb, which contains a clear fluid in the most superficial layer of the skin: some of the lesions are ovoid and in the direction of the folds of the skin. After a day or so the fluid is invaded by staphylococci, causing the fluid to become opaque: the vesicle meanwhile loses its tension and dries into a scab which within ten to fourteen days separates, leaving a pigmented scab but rarely extensive scarring. Some of the papules do not proceed to vesiculation at all, but dry up. The essential feature of this eruption is that it *comes out in successive crops*, and so we see different stages of the rash on the same area of skin: this process rarely exceeds four days and is often less. The earliest lesions often appear on the mucous membranes of the palate and cheeks, which should always be inspected: on the skin, first the back, and then the front of the chest and abdomen are invaded: soon the whole body is affected, including the face and limbs, but as the lesions spread away from the centre, so they become much less numerous. Hence the density of the lesions is much less on the forearms and hands than on the upper arms, is less on the lower legs and feet than on the thighs, and is less on the upper face and scalp than on the lower face and neck. On the arms and legs, the *flexor* rather than the *extensor* surfaces are affected. The number of lesions can be very variable: in some the whole body seems to be covered, in others only isolated vesicles are to be seen.

The whole disease seldom lasts longer than ten days, and may be so trivial as to pass unnoticed by the patient. The temperature rarely exceeds 103° F., and mild cases may be afebrile throughout. A case ceases to be infectious after the primary scabs have separated. The incubation period is usually about a fortnight, with limits from ten to twenty-one days (see Table XXIIA, p. 557). A *quarantine period* is unnecessary, but child-contacts should be kept under regular observation for twenty-one days.

Varieties.—A *non-eruptive form* (varicella sine varicellis) may occur, but abortive lesions may have been missed in some of these cases. *Varicella bullosa* and *V. ulcerosa* occur most commonly in children with a concomitant infection with virulent streptococci as in those who have

simultaneous impetigo contagiosa or scarlet fever. *V. gangrenosa* occurs when the lesions are infected by hæmolytic streptococci or by C. diphtheriæ. *V. hæmorrhagica* in which bleeding occurs into and between the vesicles, and from the mucous membranes, is very rare but usually fatal.

Diagnosis.—Modified Variola is the chief disease from which it has to be differentiated, although this should not be difficult, because in small-pox (i.) the rash comes out definitely on the third day; (ii.) it does not appear in successive crops; (iii.) its favourite situations are the distal extremities; (iv.) the evolution of the pock is much less rapid; and (v.) the constitutional symptoms are very definite and characteristic; and see p. 571. *Herpes zoster* is distinguished by the limited area, and grouping of the vesicles (§§ 635, 826). *Pemphigus* is identified by the size and chronic character of the blebs, but a bullous or pemphigoid form of varicella may occur. *Dermatitis Herpetiformis* is very chronic, its vesicles occur in groups, and irritation is severe. In *Scabies* the chest and abdomen are not the most affected areas, and oral lesions are never seen.

Etiology.—Varicella is essentially a disease of childhood, but adults are not exempt; even elderly persons may be attacked.[1] It occurs in epidemics, for the most part of limited extent, though it is endemic in London. One attack usually confers immunity, but there are many reported cases of second and even third attacks. Other infectious fevers predispose to it; attacks following scarlet fever are apt to be severe. The disease is transmitted mainly by droplet infection, but can be carried by feeding utensils and by the hands and clothing of contacts. The disease can be inoculated, though not so constantly as small-pox. The vesicle fluid has been found by C. R. Amies and others to contain elementary bodies which are much smaller than those of small-pox and which are agglutinated by the serum of patients convalescent from varicella.

There is a close relationship between the virus of chicken-pox and of herpes zoster, and the elementary bodies of the two appear identical (Amies). A patient with herpes zoster can cause contacts to develop chicken-pox after the usual incubation period, and more rarely the reverse occurs. Yet an individual who has had chicken-pox is not protected against herpes zoster.

Prognosis.—An attack is usually over in a week or ten days, but it is apt, particularly in adults, to be followed by weakness which indeed may be more troublesome than the disease itself. Death is very rare apart from the hæmorrhagic form and secondary infection (see *varieties*). Rare *complications* include encephalitis, meningitis, myelitis, neuritis, arthritis and fibrositis, from all of which recovery is usual.

Treatment.—The itching is generally the chief trouble. The child should be prevented from scratching the pocks. The early application to each crop of papules or vesicles of 2–3 coats of a paint (cresol 0·5, tannic acid 12·5, collodion flexile 100) decreases the amount of pustulation and subsequent scarring (Mitman). The oral lesions need frequent mouth washes. Quinine and arsenic are the best remedies for the resulting

[1] J. D. Rolleston, "Varicella in Old Age," *Brit. Med. Jour.*, 1932, ii, 1007.

weakness. When the lesions are infected by hæmolytic streptococci or by C. diphtheriæ, give full doses of penicillin and diphtheria antitoxin. Isolation need not be maintained for more than fourteen days from the first appearance of the eruption.

§ **477. II. Scarlet Fever** (synonym: Scarlatina) used to be one of the most serious, and one of the commonest, of the eruptive fevers. It is still very prevalent, especially in those under ten years of age, though its severity has undergone remarkable mitigation in this country during recent years. It may be defined as an infective febrile disease due to a hæmolytic streptococcus, attended by inflammation of the tonsils, a punctiform eruption on the skin, and followed by desquamation. There are six characteristic *Symptoms*. (1) After a period of incubation which varies from one to seven days, though usually two to four, there is a *sudden advent of high fever* to 100°–103°, reaching a maximum on the second or third day (Fig. 112). As with other hæmolytic streptococcal infections, the pulse is rapid, 120 or over: headache and muscular pains are usual. *Vomiting* with the initial rise of temperature occurs in 80 per cent. of cases. In the absence of complications, the temperature gradually subsides to normal about the fifth or sixth day. It does not, as in small-pox, subside when the rash comes out. (2) A *sore throat*, with enlarged lymph glands at the angles of the jaw, is complained of or seen on the first day. The tonsils are inflamed and often develop a follicular exudate on both sides, which can be removed without causing bleeding: the fauces become uniformly red or scarlet in colour, whereas the palate shows a punctate redness. Sore throat occurs with several of the exanthemata. In scarlet fever it is the tonsils and pharynx that are affected (rarely the larynx); in measles the larynx is chiefly affected; in small-pox both the larynx and pharynx are involved. The inflammation may become very severe, and is always attended with more or less glandular swelling. (3) The *eruption* is the next symptom and is remarkably regular in its appearance, twenty-four to thirty-six hours after the advent of the pyrexia. It has two elements—a generalised red blush, disappearing on pressure, and a number of minute points (punctate erythema) slightly raised and redder than the surrounding skin. The flush is first seen on the face, and is rapidly followed by the punctate rash which starts on the neck and quickly spreads to the chest, trunk and upper arms. The forearms and hands, and the legs are not affected at first, but within the first twenty-four hours the whole body is covered. There are certain special points to notice: (*a*) the face is flushed, but has no punctiform rash. (*b*) In contrast to measles and German measles, the face in scarlet fever usually shows a *circumoral pallor*. (*c*) Punctate hæmorrhages may be seen, especially in the flexures of the elbows (Pastia's sign). (*d*) Miliary sudamina may occur if the rash is severe. The rash continues to be well marked until the fourth or fifth day of the disease, but disappears earlier if antitoxin has been given: slight staining may remain. (4) In the early stages the *tongue* becomes reddened along the edges, and covered with a thick white fur—" Straw-

berry and cream tongue." Soon the brilliant red papillæ show through, and as the fur separates leaves a bright red denuded surface, resembling a ripe raspberry. Some confusion has arisen from the term "strawberry tongue" having been applied to both these conditions. (5) *Desquamation* ("peeling") is apt to occur with any severe skin inflammation, but it is more characteristic in this than in any other fever. It begins about the fourth day, and continues for four to six, or eight weeks—first on the face, and, following the order of the rash, *last on the palms and soles*, the complete desquamation of which may be very tedious. In the latter position the flakes are large; elsewhere they are small and shreddy. (6) The *blood*

Fig. 112.—Scarlet Fever.—Arthur M——, æt. 5. A typical mild case, especially as regards the initial symptoms, the rash, the tongue, and the desquamation. The various incidents are shown on the chart.

shows a polymorphonuclear leucocytosis and moderate eosinophilia. According to some observers, the Wassermann reaction is positive during the acute stage, though this is denied by others.

The primary cause is a hæmolytic streptococcal infection, usually of the throat, and sometimes arising from wounds (surgical scarlatina) or from the uterus (puerperal scarlatina). The rash indicates the production of an erythrogenic toxin by the infecting organism. Thus additional signs of the disease are available for (7) a hæmolytic streptococcus can be isolated from the primary source of infection (usually the throat and nose). A positive result may be obtained in non-scarlatinal cases, but a reliable negative report excludes the disease. (8) The *Schultz-Charlton* reaction or

Extinction Sign consists of a blanching of the eruption within eight to twenty-four hours of intracutaneous injection of 0·2 c.c. of a 1 in 10 dilution of scarlatinal antitoxin. It is absent in non-scarlatinal eruptions. (9) A *positive Dick test* (§ 656) becomes negative after an attack of scarlet fever.

Varieties.—There are four chief varieties: (1) The *Benign*, simple or ordinary type as above described. Various symptoms—*e.g.*, rash, fever or sore throat—may be absent and these cases are spoken of as *latent* or *formes frustes*. (2) *Modified* scarlet fever follows the administration of scarlatinal antitoxin within the first one to two days. Within forty-eight hours of the serum the temperature settles to normal, the intensity of the sore throat and rash is considerably lessened and desquamation may not occur. (3) In *Septic Scarlet Fever*, Scarlatina Ulcerosa or Anginosa, the ordinary symptoms are aggravated by a septic infection of the throat, with an exudate which may spread beyond the tonsils and may produce local ulceration even of the fauces and palate. From this focus septic material is absorbed, the upper cervical glands may suppurate and the middle ears become involved. The rash is often faint, but a blotchy or gyrate eruption frequently appears on the face and limbs in the second or third week. (4) In the *Toxic* form the patient is seized with high fever, delirium, and marked cardio-vascular weakness; the vomiting persists, the rash is very intense, but the throat symptoms often ill-marked, and the patient dies during the first week. Toxic scarlet fever of such intensity as to deserve the name *Malignant*, with low muttering delirium, usually a marked rash, and death without complications in a few days, is a very rare variety.

Diagnosis.—The diagnosis of scarlatina is not difficult in typical cases. The abrupt advent of high fever, accompanied by vomiting and sore throat in a child who has not had the disease, is always extremely suggestive, and if the disease is prevalent the diagnosis is almost certain. During the first few days the greatest difficulty is sometimes experienced in the diagnosis from *tonsillitis*, and especially that variety due to other strains of hæmolytic streptococci. Vomiting is more common in scarlatinal cases, and a careful watch must be kept for the rash and for subsequent desquamation. *Diphtheria* has no punctate rash, though a flush may be seen on the chest and arms, but the characteristic membrane appears on the throat and the tongue remains coated. In doubtful cases it is best to act as if the graver disease were present (see Table X, § 156). *Measles* is associated with marked catarrhal symptoms in the eyes, nose and bronchi, and Koplik's spots are usually present. The characteristic differences between the rashes of the two diseases are best seen on the limbs. *Dengue* (*q.v.*) is accompanied by severe articular pains and a morbilliform eruption on the fourth day; the diagnosis is easier when the eruption is present. The scarlatinal rash is distinguished from the diffuse prodromal erythema of *small-pox* by the fact that the latter starts in the groins or axillæ, and invades the oral circle if the rash is diffuse, and lumbar pain is usually complained of. *Enema rashes* and *Epidemic*

Exfoliative Dermatitis are sometimes mistaken for scarlatina. A *septic rash* may be scarlatiniform, but is distinguished by fever of a pyæmic type, the presence of a septic focus, and the absence of characteristic punctation. The erythema of *belladonna poisoning* is accompanied by great thirst, dryness of the fauces, and dilatation of the pupils. *Copaiba* or *Sulphonamide rashes* and those due to so-called "*ptomaine poisoning*" may be a source of confusion.

Etiology.—The disease is highly infectious, especially at the onset and during the early stages. It is due to a hæmolytic streptococcal infection with an organism belonging to Lancefield's Group A, and capable of producing a toxin which causes the characteristic skin rash. The infection is propagated through the air for short distances as a droplet-infection from other cases, from healthy carriers, or from an infection derived from a case recently discharged from hospital ("return cases"). More rarely the organisms are conveyed by dust, or by direct contact with an infected spoon, fork or the nurse's fingers: outbreaks due to infected milk have been recorded.

The patient used to be regarded as infectious until desquamation had ceased, a period averaging four to six weeks, or even longer. There is no evidence that the desquamation of scarlet fever is ever infectious, traditional belief notwithstanding. On the other hand, the infection may survive in the mucous discharges from the throat and nose, and possibly the ears, for many weeks, long after the peeling has completely finished. One attack usually gives immunity for life. *Relapses* or second attacks are believed to arise in those who have developed a poor immunity from the first attack (shown by a persistently positive Dick test), and who are infected by a different serological type of hæmolytic streptococcus.

A hæmolytic streptococcus has been proved to be the causal organism, on the following grounds: (1) inoculation of an apparently pure culture has produced scarlet fever in volunteers; (2) intracutaneous injection of a filtrate of the culture gives a strongly positive reaction in susceptible subjects (Dick test); (3) preparation of a serum by immunisation of a horse with the scarlatinal type of *Streptococcus hæmolyticus* has a curative effect. The presence or absence of a rash depends on the susceptibility of the individual and the capacity of the organism to produce a highly active erythrogenic toxin.

The *Dick Test* is performed by injecting intradermally one skin test dose of scarlatinal exotoxin, contained in 0·2 c.c. of fluid. In eight to twelve hours, there appears a small circular erythematous area which reaches its maximum in eighteen hours after injection, then rapidly fades. To avoid pseudo-positive reactions, a control test should be carried out simultaneously. A true positive result is found in 70 to 100 per cent. of cases of scarlet fever in the first three days of the disease, as well as in susceptible persons. The test possesses some diagnostic value: conversion of a positive reaction in the acute stage into a negative reaction at the end of the week or fortnight indicates that the disease is scarlet fever.

Prognosis.—The disease has become very much milder in Great Britain, and during the last seventy years the case fatality in the London Fever Hospitals has fallen from 13·5 to under 1 per cent.: it still remains serious in other parts of the world. The danger is greater in those under five

years: untoward symptoms arise when the throat infection is severe, the temperature above 105°, when cardio-vascular toxæmia is marked and with persistent vomiting. Delirium at night is more or less usual in severe cases, but violent delirium or stupor is a bad sign. The septic, toxic, malignant and hæmorrhagic forms always cause anxiety. Otherwise the disease is noteworthy chiefly for its *Complications* and *Sequelæ.* These may cause death even after slight attacks. (1) Some degree of upper cervical adenitis is usual: more marked changes and even abscess formation occur in about 5 per cent. (2) Otitis media is regarded as one of the most important complications, attacking 2–3 per cent. of all cases, and leading occasionally to permanent deafness, while mastoiditis and its complications may follow. (3) Acute nephritis (1–2 per cent.) appears usually at the end of the third week, very rarely after the fourth. It is much less common if children are kept strictly in bed from the commencement of the disease: it usually shows itself by slight pyrexia, albuminuria and the presence of casts. This may soon clear up or may proceed to more severe acute nephritis and occasionally to uræmia: chronic nephritis may follow. (4) Scarlatinal rheumatism occurs in the third week, and is due to supervening acute rheumatic fever, often with carditis. (5) Acute sinusitis, ulcerative stomatitis and broncho-pneumonia are relatively rare. Among the *sequelæ* subacute rheumatism and chorea are occasionally found.

Treatment.—With the milder cases now prevailing, it is no longer necessary to insist on treatment in a fever hospital, so long as the patient can be isolated and nursed at home (*Hygienic* treatment is considered in §§ 522 *et seq.*). Strict bed rest for at least three weeks is necessary even in the mildest cases, to prevent complications: a well-ventilated room is essential. Aspirin is useful as a gargle and to swallow in the initial stages: kaolin poultices form a useful application to the cervical glands. *Serum treatment* by the injection of 3,000–12,000 units (intramuscularly) of scarlatinal antitoxin, shortens and lessens the initial toxic symptoms: the dose may be repeated after twenty-four hours with advantage. Penicillin and the sulphonamides have no effect on the toxæmia but are of great value for the septic complications of the disease: they may be given in addition to the antitoxin. If an abscess forms in the neck, or in the middle ear, incision will be necessary. For the treatment of acute nephritis or acute rheumatism, see §§ 397, 582. *Isolation* should be carried out for a period not exceeding four weeks, in uncomplicated cases. The occurrence of return cases, *i.e.*, cases of scarlet fever arising in the same family within a month of the patient (primary case) being sent back to it, is most frequent in the cold months of the year, between the ages of 8 and 10, and within the first fortnight of the patient's return, especially if the primary case has suffered from rhinitis or otitis while in hospital.

Prophylaxis can be promoted by three methods: (1) *Chemotherapy.* A daily dose of sulphadiazine (G. i) for twelve days has stamped out an epidemic among naval recruits. (2) *Passive immunisation* by the injection

of 3,000 units of antitoxin (intramusc.) produces temporary immunity for ten to fourteen days. (3) *Active immunisation* consists in administering to Dick-positive persons five graduated doses of scarlatinal toxin at weekly intervals, after which immunity has usually been conferred. Unfortunately reactions to these injections are common. A Dick test one month after the last dose should have become negative.

§ **478.** III. **Erysipelas** may be defined as an acute febrile contagious disease, characterised by a progressive marginated redness and tumefaction of the skin, usually attacking the face, or the neighbourhood of wounds. (1) *The Stage of Invasion.*—After an incubation period of one to seven days the advent is abrupt, as in scarlet fever and small-pox. The temperature on the evening of the same day may be 103° to 104° F., or more. Vomiting is very common, and so also are muscular pains, especially pain in the back,[1] like that of small-pox. (2) The *Eruption* begins about twenty-four to thirty-six hours after the advent of fever, as a tense red spot on the face (facial erysipelas) or at the site of an abrasion (which may be microscopic). It often commences just within the external nares on one side at the junction of the skin and mucous membrane. It enlarges, spreads, becomes bright red and tender: where the skin is loose as in the eyelids or the scrotum, œdema is well marked. Thin-walled bullæ may form in the centre of the inflammatory area. The advancing edge is sharply defined and raised, the receding edge indefinite. The eruption may vary in duration from three or four days to a fortnight: it is materially shortened by chemotherapy. Delirium at night is not unusual. Convalescence becomes established, and desquamation occurs in the course of one to three weeks. During this last stage albumen may appear in the urine, if it has not appeared before.

Diagnosis.—Erysipelas is to be diagnosed from *erythema* complicated by cellulitis, in which the margin is less raised, and there is less fever. In *herpes* of the first division of the fifth nerve vesicles occur in groups, are limited to one side of the face, and are unattended by fever.

Varieties.—(i.) Although erysipelas and cellulitis are often classified as separate diseases, spread of the infection from the skin to the subcutaneous tissues may give a combination of both (erysipelo-cellulitis). (ii.) Phlegmonous erysipelas or gangrenous erysipelas are severe varieties with suppuration or extensive sloughing. (iii.) Erysipelas neonatorum is a very fatal variety; death may be due to peritonitis by inflammation spreading along the umbilical cord. (iv.) Erysipelas of the fauces is a severe variety, the eruption spreading to, or starting in, this situation. The disease may spread to the larynx and cause fatal dyspnœa.

Etiology.—It is a highly contagious malady due to a local infection with a hæmolytic streptococcus. Persons are predisposed to it, especially alcoholics, by wounds and unhygienic conditions. Infants and persons

[1] This is not usually mentioned as characteristic of erysipelas, and the first case I was called to I mistook for small-pox on this account. I have never met with a case in which it was absent, excepting in second or third attacks of the disease.

over forty are most liable. Even in so-called idiopathic cases the organism is probably introduced through a minute and hardly visible scratch. The presence of a wound is the strongest predisposing cause, and it spreads amongst surgical patients with great rapidity. One attack gives no immunity; on the contrary, it predisposes, and some elderly people are liable to an attack of facial erysipelas every year.

Prognosis.—The usual course is favourable, but the disease is more dangerous in infancy or old persons, alcoholic or plethoric patients, and those affected with chronic diseases, especially nephritis. Hyperpyrexia, persistent vomiting, lividity of the rash, and typhoid delirium are untoward symptoms. *Complications* include septicæmia, broncho-pneumonia and acute nephritis. Death may occur by coma or syncope.

Treatment (Hygienic Treatment, see §§ 522 *et seq.*). The treatment of erysipelas has been revolutionised by the administration of sulphonamide drugs and penicillin, which have a remarkably favourable effect on the duration of the spread of the local lesion, the length of primary pyrexia and of the toxæmia. The dosage of any of the sulphonamides is 1 G. by mouth every four hours until recovery, and 1 G. t.i.d. for another seven days. Local applications are unnecessary, but on the face and eyelids local bathing gives relief.

§ **479.** IV. **Small-pox** (Variola) is a highly contagious eruptive fever, the eruption passing through the stages of papule, vesicle, pustule, and scab. It essentially exists in THREE FORMS. (A) A virulent or classical form, not now seen in Great Britain—Variola Major. (B) A mild form in the unvaccinated, the type chiefly seen in Great Britain and N. America in recent years—Variola Minor or Alastrim. (C) Small-pox modified by previous vaccination—Modified Small-pox.

(A) VARIOLA MAJOR. The *Symptoms* fall into two groups: (1) *Prodromal.* After a definite incubation period of twelve days, characteristic constitutional symptoms appear—viz., sudden advent of shivering and high fever (101°–104° F.), with severe headache and *pain in the back.* The most noticeable features of this primary fever are the severity of the pain in the back (which, in my experience,[1] is present even in the mildest cases), and the frequent occurrence of vomiting: cough and bronchitis are common. *Prostration is marked,* the face becomes grey in colour, the conjunctivæ suffused and the tongue furred. The mind may be active and the patient sleepless, or a toxic delirium with mental confusion may be present. During the stage of primary fever there is, as a rule, no eruption, but in some cases a prodromal rash makes its appearance on the second day. This may be (i.) erythematous, generally found in the groins or other folds, occasionally it covers the whole body, in which case the outlook is grave; (ii.) morbilliform, usually occupying the apron area, but also occasionally diffuse; or (iii.) a profuse hæmorrhagic eruption sometimes appears on the anterior surface of the

[1] Report on the Warrington Small-pox Epidemic, by Dr. T. D. Savill; Blue Book of the Royal Commission on Vaccination. Eyre & Spottiswoode, London, 1895.

abdomen and thighs and indicates a very severe attack. After two or two and a half days, these initial symptoms disappear, the temperature drops, and on the third day the true small-pox eruption appears. (2) During the *Eruptive stage*, the temperature at first remains much lower—the patient, indeed, may feel comparatively well. The earliest lesions are often visible in the mouth and involvement of the larynx and pharynx causes a sore throat. On the skin, for the first few hours (of the third day) there is a macular eruption which rapidly gives place to a crop of papules of *shotty hardness* which can be felt even more readily than they can be seen, like small shot beneath the skin (Plate I): each papule is surrounded by a pink areola. They first appear on the forehead and on the fronts

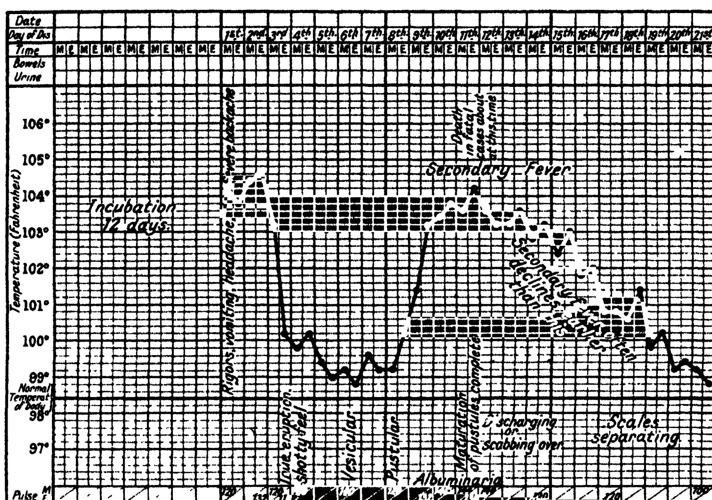


FIG. 113.—UNMODIFIED SMALL-POX.—Severe confluent case, unvaccinated, terminating in recovery. The various incidents are shown in the chart.

of the wrists, and then the eruption travels over the whole body, the chest, abdomen, groins and legs being least affected: this papular stage is complete in forty-eight hours, and the papules then more or less simultaneously become vesicular (on the fifth or sixth days). The eruption *comes out in one crop* and is never multiform in any given area of skin as in varicella. Some of the papules, however, may abort and not proceed to vesiculation. Each vesicle enlarges, and by the seventh or eighth day has become pustular: with this a secondary suppurative fever develops, which may last six to eight days and be attended by rigors (Fig. 113). In typical cases, unmodified by vaccination, each vesicle presents a depressed centre which is held down by a bridle (umbilication). The next day (eighth day) the bridle ruptures, and each pustule becomes hemispherical, about as large as a split pea, with an inflamed and indurated

base, and at this time considerable œdema of the skin is present. These pustules gradually dry into scabs, which separate about the fifteenth to the twentieth day, though in some situations, such as the scalp, forehead, and sides of the nose, considerably later, leaving patches of congested skin, and in severe cases a pitted cicatrix. The extent of the eruption and the amount of inflammatory induration varies considerably. Sometimes only the face and wrists present a few spots; sometimes the whole body is covered. The eruption is always most profuse where the skin has been irritated by any cause. The eruption on the legs always presents a proportionate retardation of development, since it appears last in this situation. Consequently, before certifying a patient as free from infection, the soles of the feet should be carefully examined, and should the thick epidermis be found to harbour any dried-up remnants of obsolescent pocks, these should be carefully dug out and removed before the case can be regarded as free from possible infection.

Varieties.—It is sufficient to describe four varieties according to the severity of the disease, the severity of the symptoms corresponding very closely with the character and extent of the eruption on the face: (1) *Mild or Discrete*, (2) *Confluent*, and (3) *Malignant or Hæmorrhagic.* During the eruptive stage, a hæmorrhagic prodromal rash may be associated with hæmorrhages within and beneath the skin, and from most, if not all, of the mucous membranes. Death ensues early, even before the vesicles appear (Fig. 115). In a second variety, which is less fatal, hæmorrhages occur into and between the pustules. (4) A *non-eruptive form* (variola sine variolis) may occur as in the other acute exanthemata, and may be mistaken for influenza. It occurs among contacts recently vaccinated.

(B) Variola Minor (synonym: Alastrim) is the term applied to true small-pox occurring in an unvaccinated person in which the severity of the disease has been considerably lessened (as compared with *V. major*) due to diminished toxicity of the organisms: the typical lesions tend to abort and the secondary fever is relatively slight. It is the type which has been most prevalent in Great Britain in the last twenty-five years.

Symptoms.—(1) The incubation period is often prolonged beyond the characteristic fourteen-day period of *V. major*: fifteen to seventeen days to the appearance of the rash is quite common. (2) The prodromal symptoms are usually slight. (3) The distribution of the typical lesions is the same as in *V. major.* (4) The papules appear more slowly, but the lesions mature more quickly. This may give a deceptive appearance of crops of lesions such as are met in chicken-pox. (5) A considerable number of papules and vesicles abort, and so secondary infection and its associated fever is slight.

Etiology.—The identity of *V. minor* with ordinary small-pox is shown by (1) vaccination is equally protective against both: (2) the distribution of the eruption is the same: (3) the serum agglutinations of *V. minor* and *V. major* are equally effective against the Paschen bodies derived from the lesions of *V minor* and *V. major.*

(C) Modified Small-pox (synonym: Varioloid) is the disease which arises when a previously vaccinated person developes *V. major*: immunity is only partial, and may be the result either of unsatisfactory vaccination, or of vaccination many years previously.

Symptoms.—(1) The primary fever and early symptoms are often indistinguishable from *V. major*, and the eruption appears on the fourteenth day. (2) Certain portions of the eruption abort and do not pass through all stages. (3) As a consequence several stages of the eruption may occasionally be seen on the same portion of skin. (4) The general eruption may be very scanty, and may consist of not more than a dozen papules, which may not even undergo vesiculation. (5) There is little, if any, secondary (suppurative) fever (Fig. 114).

Table XXV.—Differentiation between

	Chicken-Pox	Small-Pox
Age	Especially children.	Any age.
Degree of illness	Slight.	More severe, often with prostration.
Prodromal symptoms	Usually slight.	Severe for 2–3 days: high temp., headache, backache, vomiting.
Prodromal rash	Uncommon: most on chest and arms.	More common: especially groins.
Main Eruption:—	On *First day*.	On *third or fourth* days.
Temperature of eruption	Rises as rash appears.	Settles as rash appears.
Typical rash	Evolves quickly: successive crops for 4 days: several stages on one portion of skin.	Evolves slowly: one crop only.
Earliest and most profuse skin lesions	Chest and abdomen.	Forehead, wrists and forearms.
On limbs	Flexor surfaces especially.	Extensor surfaces especially.
Early stage	Superficial papules becoming vesicular in a few hours.	Deep shotty papules becoming vesicular in 2 days.
Umbilication of vesicles	Absent.	Present.
Shape of vesicles	May be oval.	Circular.
Pustulation of vesicles	Within four days.	Seventh or eighth day of illness.
Scars	Absent or slight.	Marked.
Secondary Fever	Absent or slight.	Marked.
Vaccination against small-pox	Ineffective.	Successful vaccination prevents the disease.

Diagnosis of Smallpox.—There are three important diagnostic features: (i.) Sudden advent of high fever, often with a rigor; (ii.) headache, *backache*, and vomiting at onset of the disease, of which there should always be a history, even in the mildest cases; and (iii.) the character and distribution of the papules and vesicles. The main diagnostic points and the differentiation from *varicella* are set out in Table XXV. *Measles* is the disease which is most often mistaken for variola in the early stages of the case, and therefore two plates of these diseases are presented side by side (Plates I and II). Measles is distinguished by the redness of and the running from the eyes, with other signs of catarrh, and the presence of Koplik's spots (§ 481) on the buccal mucous membrane. The rash, too, is macular rather than papular, and the individual spots as they increase in size spread out in patchy coalescence. In febrile *roseola* or lichen, the fever lasts only twenty-four hours, the efflorescence appears all over the

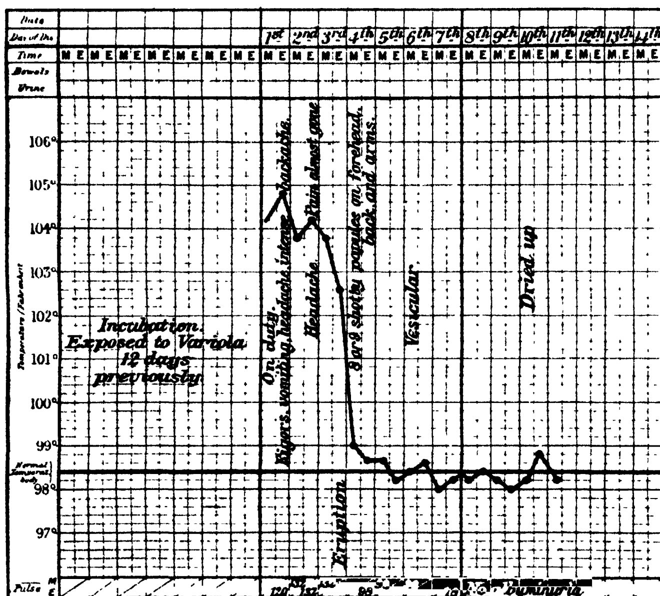


FIG. 114.—A mild case of MODIFIED VARIOLA occurring in a young woman, æt. 22, who had been vaccinated two years previously and who presented three visible cicatrices of the primary vaccination. Initial symptoms severe. No secondary fever.

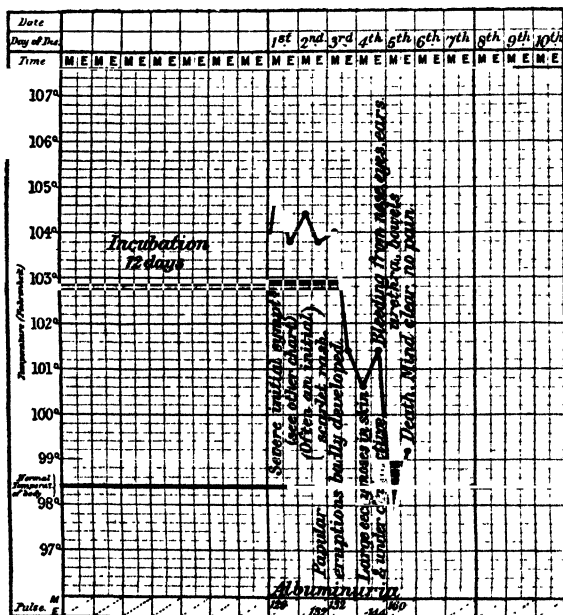


FIG. 115.—A case of MALIGNANT HÆMORRHAGIC SMALL-POX (as distinct from those cases of confluent small-pox with hæmorrhages in the pustules).—Patient unvaccinated. Death occurred on the 5th day. The various incidents are shown on the chart.

body at once, and it does not go to any further stage. *Syphilitic papules* and *pustules* are not accompanied by marked pyrexia: they remain unchanged, while in small-pox the lesions soon become vesicles and pustules (and see § 645). *Lichen urticatus* (papular urticaria) in children may be mistaken for small-pox, but is distinguished by the (1) rash being profuse on the limbs and absent or sparse on the face, (2) generally superficial situation of the lesions, (3) absence of inflammatory reaction, and (4) presence of severe itching.

van Rooyen and Illingworth have described a method of identifying microscopically the causal virus from the papules and vesicles: this method of confirmation has, in their hands, given positive confirmation in 96 per cent. of cases.

Etiology.—Guarnieri described what were first regarded as protozoa in the epithelial cells of the small-pox vesicle, but were subsequently proved by Paschen to be the elementary bodies or virus of the disease. The disease is highly infectious and is conveyed mainly from the upper respiratory passages. Infection may also be conveyed by feeding utensils, clothing and infected fingers, and via crusts from the skin of patients.

Prognosis.—Children, and especially infants, are particularly prone to the disease, and before the discovery of vaccination in 1796 (§ 480), it was a cause of considerably more than half the infant mortality in Great Britain and other countries.[1] One attack usually confers complete immunity: authenticated second attacks are extremely rare. In *V. major* the prognosis depends (1) mainly on whether there has been prophylactic vaccination. Until recently, the case mortality was about 37 per cent. among the *unvaccinated*; about 5 or 6 per cent. among all classes of the *vaccinated* taken together; and about ½ per cent. among the *properly vaccinated.* The severity of the disease seemed to depend almost entirely upon whether the patient had been recently and efficiently *vaccinated.*[2] In the healthy and recently vaccinated it was a comparatively trivial disorder, but in the unvaccinated, especially in infancy, it was one of the gravest diseases. Even so, it must be realised that vaccination is not an absolute safeguard against even virulent small-pox. (2) The second factor is the question of *age*: the official records of the outbreak in Warrington in 1773 showed that of 211 fatal cases, 166 were under three years of age. (3) Alcohol and plethora add to the gravity of the disease. As regards the *varieties*, the confluent, in which the rash may come out

[1] Warrington had an epidemic of smallpox in 1773, with a death-rate of 26·5 per 1000, all the deaths occurring in persons under nine years of age. In 1892–1893 Warrington was again visited by an epidemic, with a death-rate of 1·1 per 1000 of the inhabitants, who then had only about 1 per cent. unvaccinated persons among them.

[2] The figures from the Warrington epidemic, 1892–1893, are very striking. In the *infected* houses there were 2535 persons, and 2223 of these persons had been vaccinated in infancy. Among these latter the case-mortality was 5·2 per cent. The figures also showed that in proportion as the vaccination had been more efficient, the severity of the disease was less. Finally, among all the 667 cases which occurred in this epidemic, not one had been vaccinated or revaccinated within seven years of the attack. —Appendix to the Report of the Roy. Com. on Vaccination, 1894.

on the second day and is very abundant, is much more dangerous than the discrete form. In the former the fever does not subside on the third day, and there is a great tendency to hyperpyrexia and complications. Speaking generally, the more copious the rash, the greater the danger. True hæmorrhagic smallpox is invariably fatal, but if hæmorrhage occurs *into* the vesicular or pustular rash, there is a good chance of recovery. As regards *untoward symptoms*, the more severe the primary fever in the unvaccinated, the more severe will be the disease, but this is not necessarily so in the vaccinated; profuse salivation is a bad symptom; the case is grave if there be no swelling of the skin at about the ninth day, and still graver if the swelling goes suddenly away. The case fatality of variola minor in recent epidemics is about 0·2 per cent.

Complications.—(1) Bronchitis is common in the more severe cases. Pneumonia, empyema, and rarely œdema glottidis are often fatal. (2) A toxic myocarditis occurs in the toxic and hæmorrhagic cases, and with a severe secondary fever. Endocarditis and pericarditis are rare. (3) Nervous complications include encephalitis, with delirium and convulsions, hemiplegia or acute ataxia: post-febrile psychosis may occur. (4) Some degree of conjunctivitis is not unusual: painless corneal ulcers may produce a panophthalmia and destruction of the eye. (5) Erysipelas and cutaneous abscesses are common during the secondary fever.

Treatment.—*Prophylaxis.* It should be remembered that vaccination is capable of modifying the disease even after exposure to infection, because the incubation period of variola is twelve days and that of vaccinia only eight days. Vaccination may, therefore, be performed with efficacy during the first three or four days after exposure; and every member of an infected household should be vaccinated immediately the disease breaks out therein. For its efficiency in the prevention and modification of smallpox, see pp. 583 and 586. Treatment *of an attack* demands immediate notification and transfer to a special small-pox hospital. The patient should be nursed on a special mattress and kept as quiet as possible; the heart muscle should be carefully guarded by skilful nursing. Headache and pains in the neck and limbs in the earlier stages require aspirin and even the use of morphia or heroin: restlessness and delirium in the secondary toxic stage need full doses of sedatives and narcotics. The eyes should be examined in a good light each day. To protect the skin, the whole body may be painted daily with potassium permanganate solution (5 per cent.) from the early papular stage: a weak dettol solution is comforting and acts as a deodorant. Finsen reported that the exclusion of all except red rays from the sickroom was beneficial, but this has not proved very helpful in this country. In the control of the secondary infection and secondary fever, sulphonamides have, on the whole, been disappointing; but the use of penicillin three-hourly from the start of vesiculation may prove more efficacious. *Hygienic Treatment* is given in §§ 523 *et seq.*

disease called vaccinia, by inoculating him with the lymph taken from the udder of a cow or calf suffering from that disease. It was noticed in 1769 by a German that people engaged in the milking of cows were exempt from small-pox. Jenner, in 1796, placed the subject on a scientific basis, and ascertained that the inoculation of a human being with the lymph taken from the unbroken vesicles on the udder of a calf suffering from vaccinia protected that person from small-pox. He was also the first to inoculate this disease (vaccinia) from person to person by taking the lymph from the vesicle on the arm which had matured on the eighth day after inoculation. Vaccination was made compulsory in 1853. In 1897 this law was repealed in response to an outcry among the public that syphilis and (?) other diseases could be conveyed from person to person in this way. Syphilis certainly has, in rare instances, been conveyed by arm to arm vaccination ; but by using calf-lymph this is entirely obviated. Compulsory vaccination has been abolished by the National Health Act (1946). All public vaccinators now use lymph from the calf which has been diluted 1 in 5 with glycerol-saline, together with a preservative. Goodpasteur has perfected a method by which the virus is grown on the chorio-allantoic membrane of chick embryos.

Rules for vaccination.—The older method of four areas of vaccination has now been superseded. The area of skin to be vaccinated may be over the deltoid, on the abdomen, or over the outer side of the calf: this is washed with soap and water and allowed to dry thoroughly. Two methods of insertion may be used: (1) Three parallel lines ($\frac{1}{8}$ inch long and $\frac{1}{4}$ inch apart) are drawn by a sterile round-pointed needle, which should not draw blood: the lymph is ejected from the capillary tube over this prepared area, or alternatively the parallel strokes may be made through the lymph. (2) The multiple-pressure method is used mainly in America. A drop of lymph is placed on the skin, and pressure is applied by the side of the point of a Hagedorn needle so as to indent the skin but not to draw blood. For primary vaccination in an adult, ten pressures are made (*i.e.*, the skin is indented ten times): for primary vaccination in an infant, and for secondary vaccination, thirty pressures are made. In either case, a sterile dressing is then applied. *Primary vaccination* is safest and best performed in the third-sixth month of life. *Re-vaccination* is necessary each five to seven years, if immunity is to be maintained. *An extensive* skin rash, a poor general state of health, and recent exposure to other acute specific fevers, are the only indications to postpone vaccination.

The Phenomena of Vaccination.—There are no symptoms for the first two days. On the second or third day a slight pimple, on the fourth day a definite papule and on the fifth day a bluish-white cupped vesicle appears. On the eighth day (the same day of the week as that on which the operation was performed) the vesicle *becomes pustular* and the areola increases during the next two days: at the same time the axillary or groin glands draining the area become swollen and painful. After the tenth day the pustule dries up ; the scab falls on the fourteenth or fifteenth day, leaving a pitted

cicatrix. In re-vaccination the reaction usually appears earlier and the vesicle becomes mature sooner than in primary vaccination. No infant should be considered insusceptible to vaccination unless the operation has been repeated several times with different varieties of lymph.

The inquiries which the author made on behalf of the Royal Commission on Vaccination into the Warrington Epidemic (*loc. cit.*) went to prove (1) that efficient primary vaccination offers absolute protection against *infection* for the ensuing five or six years, and relative protection (gradually diminishing) for a considerable time ; (2) that primary vaccination lessens the *severity of the attack* of small-pox if contracted during the ensuing twenty or thirty years ; (3) that re-vaccination affords absolute *immunity from attack* during the ensuing five or six years, and relative protection for the rest of life ; and (4) that if everybody were vaccinated in infancy and again at twelve and twenty-one, small-pox would be exterminated.

Complications of Vaccination.—*Generalised vaccinia* is a rare condition almost exclusively found in children following the first vaccination. It generally occurs between the ninth and fourteenth days after vaccination and may be later still. It usually appears as a single crop of papules which mature into vesicles and pustules and later crust: occasionally there are separate crops for five to six weeks. The distribution of the lesions is not that of small-pox and there is greater variation in size of the lesions. Toxæmia is likely to be severe, but is only likely to be serious and even fatal when it supervenes on a pre-existing skin disease, especially eczema, seborrhœic dermatitis or impetigo. *Accidental vaccinia.* Persons in charge of recently vaccinated children have frequently been inoculated by lymph from the child's arm, in various parts of the body, especially on the face, lips and eyes, and occasionally on the mouth, throat and genitals.

Various transient *rashes* of a scarlatiniform or morbilliform type, urticaria, erythema multiforme, and hæmorrhagic eruptions occur. *Secondary infections* such as impetigo, furunculosis, erysipelas, cellulitis and gangrene have become rare since the introduction of calf lymph.

Post-vaccinal encephalitis, and less frequently other nervous manifestations such as meningitis, myelitis or polyneuritis develop nine to fifteen days after vaccination. *Symptoms.* There is a sudden onset with pyrexia, headache, and vomiting, which may be followed by delirium, convulsions or coma. Residual damage is rare, for cases usually end in coma and death, or in complete recovery. These sequelæ occur practically only in connection with primary vaccination, and the great majority have been found in children of school age ; only 7·2 per cent. of 509 cases of post-vaccinal encephalitis occurred in the first year of life (McNair Scott). The best *treatment* of these nervous sequelæ is by intravenous or intrathecal injection of 5–10 c.c. of the serum of a person recently successfully vaccinated with the same lymph (and see § 740).

§ 481. V. **Measles** (synonym: Morbilli) may be defined as an infectious febrile disease attended by catarrh of the ocular, nasal, and respiratory mucous membranes, and by an eruption of minute elevated papules which, as they enlarge, become aggregated into irregular and often crescentic groups.

Symptoms commence after an incubation period of seven to fourteen days, usually ten or eleven. At the commencement of the incubation period and a few hours after infection, there may be a transient febrile catarrh and a fleeting rash ; this may occur in 10 per cent. of cases and is

known as the " illness of infection." Then the typical attack commences at the end of the incubation period. (1) *Prodromal symptoms* occur in the first four days until the typical rash appears: (i.) Pyrexia comes on abruptly, though not as suddenly as in scarlet fever, rising to 102°–103° F. on the evening of the first day. During the next two days it usually declines a little (Fig. 116). (ii.) Catarrhal symptoms arise, often with some sneezing and redness of the conjunctivæ. During the next three days these increase, with profuse lacrymation, redness and œdema of the conjunctivæ, a running nose, faucial injection, and a short dry cough with catarrh of the larynx and bronchi—indeed, if the tem-

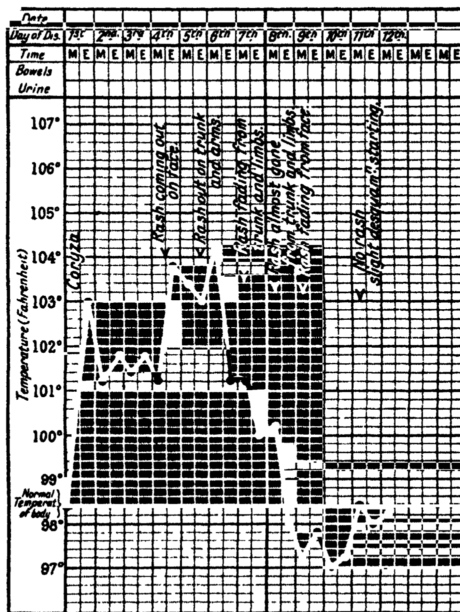


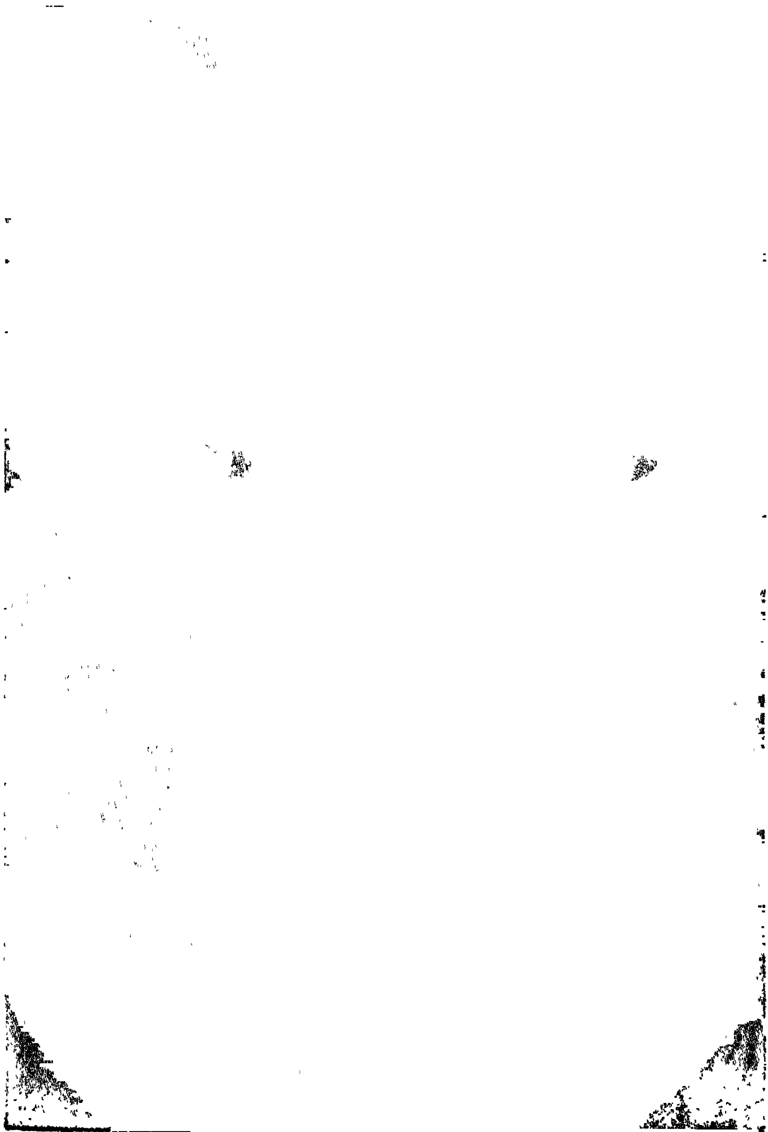
FIG. 116.—MEASLES.—Ethel H——, æt. 5 (under the author's care). Typical chart. The various incidents are shown upon the chart.

perature is not very high the case may be mistaken for coryza. The tonsils are inflamed and may present an exudation. (iii.) Koplik has described spots, which appear on the second or third day of the prodromal period, on the buccal mucous membrane opposite the bicuspid or molar teeth, and especially around and in front of the parotid duct. They are better seen in daylight than in artificial light and are often more numerous on one cheek than on the other: the typical lesions are *minute* white spots of pin-point dimensions surrounded by a red flush (§ 200). At times discrete and few in number, they may on occasion be very numerous, when they give an appearance of a white stippling on a slightly raised reddened base. They may be confused with the much larger patches of

thrush, but Koplik's spots are rubbed away only with difficulty. As they occur in at least 90 per cent. of all cases, their diagnostic significance, before the typical rash appears, cannot be over-estimated. (iv.) The tongue is at first furred, but gradually clears, so that as the rash appears the tongue may come to resemble the strawberry tongue of scarlet fever. (v.) Transient prodromal rashes of macular or scarlatiniform type are rare. (vi.) During the whole of this first phase the child looks and is " a picture of misery." Photophobia is usual. *Stage of Eruption.* (i.) The typical rash appears on the third or more usually on the fourth day (Coloured Plate II). It commences behind the ears and along the hair margin of the forehead, temples and neck. Within a few hours the whole face is involved (including the circum-oral region) and then the whole body is affected: even so the rash is always more marked on the face and trunk than on the more distant areas of the body. At first there is a reddish-brown erythema, which develops into a *macular rash* the colour of which may darken even to a maroon colour. Initially the individual lesions are discrete, but as they become more numerous they tend in the course of the next two days to coalesce into irregularly shaped blotchy patches. These are soft and velvety to the touch and the colour fades on pressure, thus differing from the early stage of small-pox papules. Soon the lesions begin to recede, and at the end of forty-eight hours to fade: within four to five days of its appearance (the ninth day of the disease) the rash has completely disappeared, except that a brownish mottling of the skin (" measles staining ") remains for some time. (ii.) Occasionally the macules become petechial. (iii.) The temperature rises somewhat as the rash appears, remains up for the two days of its development (until the sixth day), and then falls by crisis (Fig. 116). (iv.) The constitutional symptoms (malaise, headache, insomnia, etc.), and the catarrhal symptoms (especially the cough) go on increasing during the development of the rash, and they all subside together about the sixth to the eighth day of the illness, as convalescence begins. (v.) Slight desquamation of minute bran-like scales sometimes occurs over the whole body, including the hands and feet. (vi.) The blood shows a leucopenia which is replaced by leucocytosis when a complication occurs.

Varieties.—Modified, Attenuated or Abortive Measles indicates a very mild attack, either as a result of a natural immunity or of the use of prophylactic measures. Before the third month of life, measles is rare, and between the fourth and seventh months attacks are usually modified by immune bodies transmitted through the placenta. In later life, the prophylactic injection of immune bodies (*vide infra*) may result in a very mild attack: catarrhal symptoms are slight, the temperature may not be raised for more than twenty-four hours, Koplik's spots may be absent and the rash is sparse and pale coloured: complications do not arise. Occasionally the initial temperature and Koplik's spots are not followed by a rash (morbilli sine morbillis). The *hæmorrhagic* or malignant variety (toxic), fortunately now rare, is very severe, and is attended by hæmor-

PLATE I.

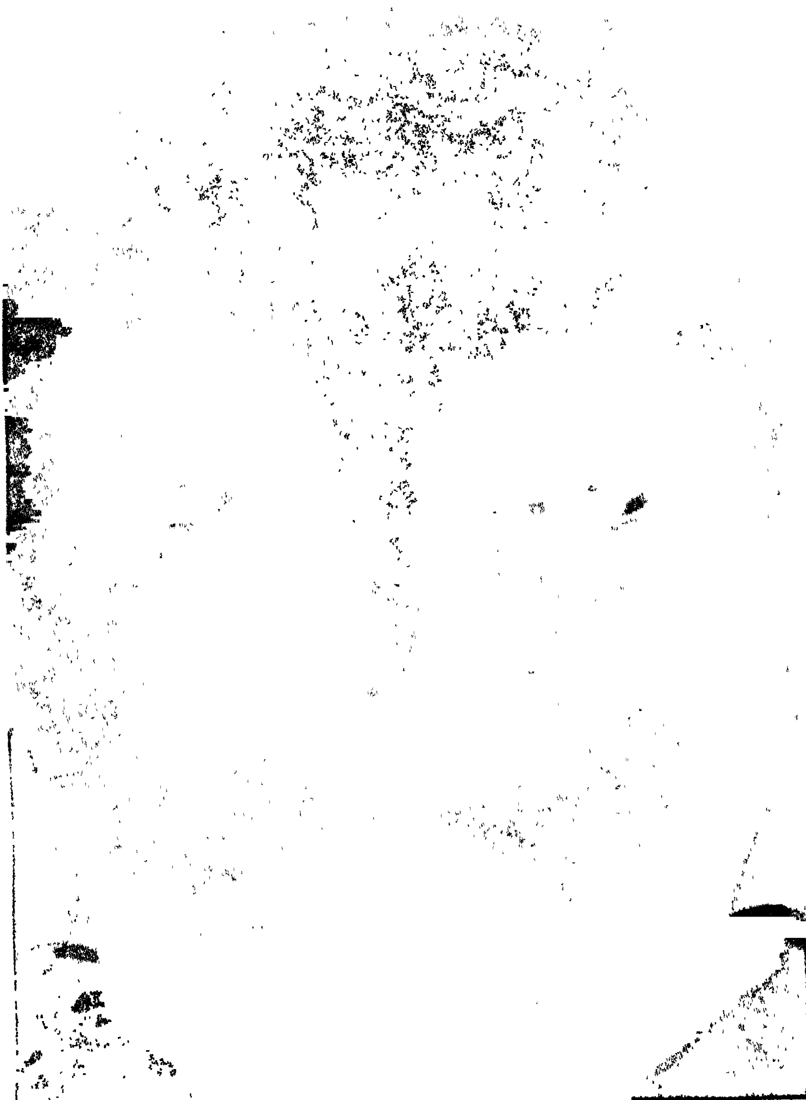


VARIOLA.

Right side of face (left of observer) represents the second day of the eruption. The other, pustular, side represents the sixth day of the eruption ; a few of the pustules, showing commencing umbilication.

Drawn from nature by Miss Mabel Green.

PLATE II.



MEASLES.

The eruption, which is very plentiful, is eighteen hours old (second day of rash). Note the evidence of coryza in the eyes and nose.

Drawn from nature by Miss Mabel Green.

rhages from many different areas of the body: it is usually fatal. Occasionally bullous lesions are seen in severe attacks (Morbilli bullosa). In the *pulmonary* variety, broncho-pneumonia commences at the beginning of an attack.

The *Diagnosis* from a severe common cold in the absence of Koplik's spots, is very difficult until the eruption appears. *Rubella* tends to occur at a later age than measles. The catarrhal symptoms and the temperature are much less marked although in adults and even in adolescents, rubella can produce severe catarrhal and constitutional symptoms. Enlarged sub-occipital glands are characteristic of rubella, while Koplik's spots are conclusive proof of measles. *Variola* often presents a difficulty; bronchial catarrh is common to both, but watering of the eyes and nose favours measles, while the presence of pain in the back and vomiting aid us considerably in diagnosing variola. The differences between the rashes are referred to on p. 581. *Erythema Multiforme* is somewhat like measles, but is recognised by the absence of catarrh, pyrexia, and of Koplik's spots. The rash due to *sulphonamide drugs* is often morbilliform. That set up by the *injection of an anti-serum* is especially suggestive, and may lead to temporary confusion. The paramount importance of recognising Koplik's spots in the early diagnosis of measles can hardly be exaggerated.

Etiology.—Measles is especially a disease of childhood, and few escape. It is endemic in England, and two-yearly epidemics occur, especially in the spring and winter. The essential cause is a filter-passing virus which has been identified in the nasal secretions, the blood and in the brain when encephalitis occurs: the organism can be cultured on the chorio-allantoic membrane of hen's eggs. It is spread by droplet infection from the nasal and bronchial secretions. Measles is as contagious before as after the eruption has appeared, and its infectivity disappears rapidly so that most cases are not infectious a week after the rash has appeared. Secondary infections by hæmolytic streptococci and by pneumococci are responsible for many of the complications. One attack confers relative immunity: the majority of so-called second attacks are probably rubella.

Prognosis.—Measles is not a serious disease in itself, except in infancy. It has become much milder in type in the last thirty to forty years, and the death-rate in England and Wales is now 1·5 to 2·5 per thousand cases. The most important determining factors are poverty and overcrowding, and the proportion of very young children. Children up to the age of 5 months are often immune and those up to the age of 9 months relatively immune (*vide supra*): after this age, and especially in the poorer classes, secondary infections are very prone to cause pneumonia, but even then much of the terror of this complication has been removed by modern chemotherapy. In middle-class children, the maximum incidence of measles is during the school age, when *complications* are fewer. (1) The most important and most common complication is broncho-pneumonia: even when recovery occurs, some residual pulmonary fibrosis, and even

bronchiectasis may ensue. (2) Catarrhal laryngitis, laryngismus or laryngeal diphtheria also occur. (3) In all cases of measles with sudden aggravation of fever for no apparent cause, acute otitis media should be suspected: this is usually caused by a hæmolytic streptococcal infection, especially before the age of 3 years, and often results in residual damage and even total deafness with deaf-mutism. (4) Conjunctivitis is a normal phenomena, but corneal ulceration which may proceed to perforation and panophthalmitis is dangerous. (5) Cancrum oris is rare nowadays; it is usually due to infection by Vincent's organisms, and begins as an ulcer on the inner surface of the cheek, surrounded by intense inflammation: soon a black slough appears, perhaps followed by perforation. (6) Acute enteritis in children before the age of 2 years may occur alone or with broncho-pneumonia. (7) Encephalitis occurs more frequently with measles than in any other acute exanthem: about the eighth day when the temperature has returned to normal, it is ushered in by drowsiness or convulsions: it tends to a rapidly fatal issue or to complete recovery. A recognised *sequela* is tuberculosis, especially of the bronchial glands: it follows measles and whooping-cough more frequently than any other febrile disease, though measles does not rouse dormant tuberculosis into activity nor aggravate active disease so frequently as was formerly supposed.

Prophylaxis.—The serum of convalescents is collected about the fourteenth day after the commencement of an attack: if injected intramuscularly in suitable doses (Table XXVI) within the first five days after exposure to infection, it will in nearly all cases either prevent an attack or render it very mild. After the fifth day, prevention is impossible, but attenuation still occurs if given before the ninth day: when an attack

TABLE XXVI.—MEASLES PROPHYLAXIS—for Children under 3 years of Age (Intramuscular Route)

Nature of Product	*Dose for*		*Days after Exposure to be given*	*Dose for Attenuation*	*Days after Exposure to be given*
	Complete Protection	*Attenuation*			
Convalescent serum .	5 c.c.	2½ c.c.	1st–5th	5 c.c.	5th–9th
Normal adult serum .	10 c.c.	5 c.c.	1st–3rd	10 c.c.	3rd–9th
Whole blood . . .	—	10 c.c.	1st–3rd	10–15 c.c.	3rd–9th
Placental extract . .	3 c.c.	—	1st–3rd	3 c.c.	3rd–9th
For children above the age of 3 years, the above doses should be doubled.					
Immune globulin . .	0·10 c.c./lb. body-weight	0·025 c.c./lb. body-weight	1st–5th		

supervenes, it is usually very mild and results in permanent immunity. Placental extract ("immune globulin") is less effective than convalescent serum, but more so than adult serum. The gamma-globulin fraction of human serum contains most of the antibodies, and this is highly concentrated and devoid of the agent which causes homologous serum jaundice (§ 332).

Treatment.—The patient is put into a shaded room, with plenty of fresh air. Admission to a fever hospital is better avoided if adequate isolation and nursing can be given at home, for cross-infection with hæmolytic streptococci and other organisms is thus avoided. Bronchitis is treated by a mixture containing ipecacuanha and liq. ammoniæ acetatis. (General treatment, see §§ 522 *et seq.*) For broncho-pneumonia and other complications, penicillin and/or the sulphonamides (§ 123) are of great value: the oxygen tent may be invaluable in small children. Particular attention must be paid to the eyes and ears.

§ **482.** VI. **Rubella**, or German Measles (synonym: Röteln), may be defined as an acute contagious disease, characterised by a polymorphous eruption, frequent enlargement of the lymphatic glands, little or no constitutional disturbance and almost invariably mild course.

The *Symptoms* vary somewhat in different epidemics. (1) After a period of incubation varying between fourteen and nineteen days, and more often seventeen or eighteen days, the temperature rises to 99°–101°. This is accompanied in adults by headache, pains in the limbs, and soon after by *slight* conjunctivitis and catarrhal symptoms. Usually the glands are swollen, the most characteristic being the upper cervical and occipital groups. Tender swelling of the posterior cervical glands is sometimes present several days before the rash appears, the patient often complaining of " stiff neck ", which he usually ascribes to having sat in a draught, or some such reasonable explanation. Occasionally the glands in other areas enlarge. Especially in children, the rash may be the first symptom of the disease, and the constitutional symptoms and primary fever are slight or absent. (2) The rash usually occurs within twenty-four hours of the first symptoms: rarely it is delayed until the third or fourth day. It consists of minute round or oval rose-red spots, varying in size from a pin's head to a pea, very slightly raised, never papular. The rash at the outset appears behind the ears and on the forehead and face, and is like that of early measles. In a day or two it becomes confluent, or nearly so, on the trunk, but on the limbs the rash is always discrete and sparse. In adults, itching may be troublesome. The rash usually lasts two to three days, and the severity of the attack is in direct relation to the severity and duration of the eruption. It is sometimes followed by slight desquamation and transient brownish staining. The blood shows a characteristic excess of Türk cells and often of plasma cells.

Diagnosis.—The characteristic features of rubella are the relative absence of catarrhal symptoms, the early appearance of the rash, and the cervical adenitis. In *measles*, the incubation period is longer, the temperature is usually higher and remains up for about six days: the child is miserable, catarrhal symptoms are marked and Koplik's spots can be identified. *Scarlet fever* shows an abrupt onset, with high fever and a high pulse-rate: the sore throat is marked with possibly a tonsillar exudate: headache and vomiting are usual. The rash appears on the *neck* and spreads downwards and is punctate in type, sparing the circum-

oral region: petechiæ occur in the flexures (Pastia's sign) and the Schultz-Charlton test is positive. *Glandular fever* is identified by the more wide-spread enlargement of the glands and the blood picture: rashes are rare in this disease, but sore throat with an exudation on the tonsils is common. In *secondary Syphilis*, the rash is not irritable and the roseola spares the face (§ 645). Some *drug* and *serum rashes* may have to be considered.

Etiology.—It is mainly a disease of late childhood, or of young adults. It is not so contagious as either measles or scarlet fever. One attack confers immunity. It is almost certainly caused by a virus.

Prognosis.—It is a much more trivial disease than measles. *Complications* are rare: polyarthritis and severe rheumatic manifestations, purpura hæmorrhagica and encephalitis have been recorded. The chief *sequelæ* are congenital defects in children whose mothers have had rubella during early pregnancy: cataract, deaf-mutism, mental changes and congenital heart disease have resulted.

Treatment is purely symptomatic. Isolation is necessary for five to six days.

VI*a*. The term "**fourth disease**" was suggested by Clement Dukes in 1900 as a provisional name for an acute exanthem which he regarded as distinct from scarlet fever, measles and rubella. The great majority of experts, however, do not recognise its autonomy, and are of opinion that most of the cases so described were examples of mild scarlet fever or rubella.

VI*b*. Erythema infectiosum, or "**fifth disease,**" is an acute exanthem appearing in epidemic or sporadic form and characterised by its typical localisation on the cheeks and extremities, and almost complete absence of constitutional disturbance, complications and sequelæ. On the face, where it first appears as rose-red macules it assumes the form of a butterfly's wings on both cheeks, which are hot and swollen; a few patches are also found on the forehead and chin. The rash next involves the extremities, especially the extensor surfaces where it develops a circinate appearance. On the trunk, which often escapes, the rash is usually morbilliform. The contagiousness is very slight; it is uncommon to find several cases in a family or institution. The *prognosis* is excellent. No *treatment* is required.

VIc. **Exanthema subitum** or "**sixth disease,**" is the name given in 1921 to an acute exanthem which occurs in children under 2 years of age. It usually runs a mild course without any symptoms beyond a three-day fever and a morbilliform rash, which appears on the fourth day, simultaneously with the fall of the temperature. It fades without leaving pigmentation or desquamation. Cases have been reported mainly in the United States, in Europe and Japan, but only a few in Great Britain.

VII. TYPHOID and some rare fevers with a rash after the fourth day are described in Group II.

§ **483.** VIII. **Dengue** (Break-bone fever: Dandy fever).—This is a specific fever lasting not more than 7 days and mainly confined to tropical climates. After an incubation period of 3 to 7 days there is sudden onset, with chilly feelings or a rigor, followed by headache, aching eyeballs and rapid rise of temperature (100° to 105° F.). Excruciating backache and joint pains follow; the joints are involved with peri-articular swelling and redness. The tongue is furred and the conjunctivæ injected. Often within 1 to 2 days the skin over the face, neck and chest becomes flushed and reddened (primary rash). Anorexia, vomiting, restlessness and insomnia may ensue. The pulse, which is at first rapid, now slows, and by the 3rd or 4th day the temperature falls to 100° F. or lower, with sweating and perhaps diarrhœa. The

patient temporarily feels better, but after a few hours to 3 days the temperature again rises, the fever lasting 2 to 3 days, and a typical saddle-back chart results. The pain returns and a measly or scarlatiniform eruption (secondary rash) appears, which implicates the limbs and perhaps the trunk and lasts a few hours to 3 days. Desquamation and itching follow. A leucopenia with relative lymphocytosis is characteristic.

Etiology.—The disease, which may occur in big epidemics, is due to a filterable virus, transmitted by aëdine mosquitoes. All ages and both sexes are susceptible and the virus is demonstrable in the blood for the first 3 days. One attack does not invariably confer immunity. The mortality rate is 0·1 to 0·5 per cent.

The *Diagnosis* is easy during an epidemic, but sporadic cases have to be differentiated from typhus, yellow fever and sand-fly fever.

Treatment.—Prevention depends on anti-mosquito measures. Medicinal therapy is similar to that outlined for sand-fly fever.

THE TYPHUS GROUP OF FEVERS consists of several related infections due to various species of rickettsias (Table XXVII). CLASSICAL or EXANTHEMATIC TYPHUS is transmitted from man to man by lice, and occurs in *epidemic* form. MURINE TYPHUS is serologically closely related, and is fundamentally a disease of rodents: it is transmitted from animal to animal and to man by fleas: in man the disease resembles classical typhus but is milder and occurs in *endemic* form. ROCKY MOUNTAIN SPOTTED FEVER and certain other forms of tick typhus form a very closely interrelated group of infections which are transmitted between the animal reservoirs of infection and man by various ticks. SCRUB TYPHUS is an epizootic (*i.e.*, endemic) disease of rodents which is transmitted from animal to animal and to man by the bites of the larvæ of various species of mites. Q FEVER is also a rickettsial disease of man, transmitted from animal reservoirs by ticks, but it differs serologically and clinically from the other tick typhuses. TRENCH FEVER is a rickettsial disease of man transmitted by lice.

The Weil-Felix reaction is of value in recognising certain of these fevers, since in some of the infections the sera of patients will agglutinate special strains of *B. proteus*. Two strains are used—*B. proteus* OX19 and *B. proteus* OXK. Strain OX19 is agglutinated by sera from patients with classical or with murine typhus, and strain OXK by sera from cases of scrub typhus. Agglutinins appear early in the second week of disease and may rise as high as 1 in 30,000 during convalescence. In the field a slide technique may be used as a rough guide. Sera from cases of tick typhus or other rickettsial diseases give negative or only weak and variable reactions with either strain. More recently specific suspensions of rickettsias have been introduced for diagnostic agglutination reactions.

§ 484. IX. **Classical or Louse Typhus** (Synonyms: *Typhus exanthematicus*, Hospital and Gaol Fever) is no longer found in England; its disappearance is a triumph of hygiene. As lice do not survive excessive heat, typhus appears in epidemics and may occur at any time of the year in Europe, but only during the cool weather in countries like Egypt and Palestine. Lice become infected some 5 to 10 days after feeding on infected human blood and remain infective for life. The causative agent, *Rickettsia prowazeki*, is found in the epithelial cells lining the gut of the louse and in its excreta; man acquires the disease by infected excreta entering through abrasions and not by the actual bite of the louse. Typhus is associated with overcrowding and personal squalor and is common in times of war, siege and famine. Both sexes are equally susceptible.

Symptoms.—(i.) The period of incubation varies from five to twenty days, but is usually about a fortnight. (ii.) There may be prodromal symptoms in the form of malaise, but the onset is usually abrupt, with shivering, rise of temperature to 103° or 104° F., and sometimes vomiting. The face and eyes are congested, the tongue coated, the breath foul, and there is persistent headache, bronchitis, and a characteristic drunken or stuporose appearance. In rare fulminating cases there may be fits and delirium. After a week of great prostration delirium develops, sometimes drowsiness

and coma. The temperature remains high for twelve to fourteen days, with slight morning remissions; then falls by rapid lysis as a rule, less frequently by crisis. (iii.) The spleen may be palpable, but the abdomen is not distended. (iv.) The rash appears on the fourth or fifth day, first on the abdomen and axillæ, and spreads to the chest, back and trunk: the face is rarely involved. It has two elements: a dusky subcuticular mottling, and purple roseolar macules, which may become petechial. (v.) There is no definite blood picture, but a leucocytosis of 12,000 to 15,000 is common

TABLE XXVII.—THE TYPHUS GROUP OF FEVERS

Disease	*Vector*	*Rickettsia Organism*	*Reservoir*	*Weil-Felix Reaction*	*Remarks*
CLASSICAL OR EPIDEMIC TYPHUS—usually severe BRILL'S DISEASE (mild late manifestation)	Lice	*R. prowazeki*	Man	OX19 +++ OX2 ++ OXK –	May be differentiated by using specific suspensions of rickettsias in agglutination and complement fixation reactions.
MURINE OR ENDEMIC TYPHUS — widespread and usually benign. Ship typhus (Toulon), Urban Shop typhus (Malaya, etc.)	Rat fleas	*R. mooseri*	Rats and mice	OX19 +++ OX2 ++ OXK –	
ROCKY MOUNTAIN SPOTTED FEVER	Ticks	*R. rickettsii*	Wild rodents and ticks	OX19 + OX2 + OXK + (Variable low titres)	
With local lesions:— FIÈVRE BOUTONNEUSE (Mediterranean), TICK TYPHUS	„	*R. conori* (*R. pijperi*)	Dog, rodents and ticks		
SCRUB TYPHUS (Tsutsugamushi fever)	Larval Trombiculid mites	*R. orientalis* (*R. tsutsugamushi*)	Field rodents and mites	OX19 – OX2 – OXK +++	
Q FEVER	? Ticks ? Dust borne	*R. burneti* (*R. diaporica*)	? Bandicoot		
TRENCH FEVER . . .	Lice	*R. quintana*	Man		
RICKETTSIAL POX . .	Mouse mites	*R. akari*	House mice		Indications of serological relationship with Rocky Mountain Spotted Fever.

Diagnosis.—The Weil-Felix reaction (*i.e.*, agglutination of *B. proteus* OX19 by the serum of typhus cases in dilutions of 1–100 to 1–2000 or higher) is a very valuable reaction, but is not obtained until the end of the first week. (1) *Typhoid fever* was originally confused with typhus, but differs in its insidious onset, type of temperature, leucopenia, rash, bacteriology and serology. (2) In *measles* the eruption resembles the typhus spots, and appears at the same date, but in typhus it does not involve the face, it is never preceded by catarrh, is never papular, and becomes petechial. (3) Some *malarial* fevers occasionally present difficulty, but they have no eruption. (4) *Uræmia* and other causes of coma may be mistaken for it. (5) Apical *pneumonia*, *meningitis*, and other causes of the *typhoid state* may be confused with typhus. The cerebrospinal fluid may be increased in pressure and contain an excess of lymphocytes in typhus. The bubonic swellings in *plague* occur earlier, during the first week.

Prognosis.—Case-mortality, 20–40 per cent.: between the age of fifteen and twenty-five, 4 per cent.; over fifty, 50 per cent or more. Thus the mortality is greatly influenced by the age of the patient, and by previous preventive inoculation. One attack usually confers immunity. Typhus is always a serious disease, especially in the plethoric and alcoholic. It terminates fatally in three ways: (i.) Degeneration of the cardiac muscle, a very common accompaniment; (ii.) coma from toxæmia; or (iii.) pneumonia. Untoward symptoms are (i.) weak, irregular, or intermittent pulse, or other indications of cardiac weakness; (ii.) an abundant rash, with high fever; (iii.) early and protracted cerebral signs or protracted hiccough; (iv.) all complications, especially pulmonary. Of the *complications* and *sequelæ*, (i.) the pulmonary are the worst, especially broncho-pneumonia and hypostatic congestion of the lungs; œdema glottidis and pleurisy are less common. Other complications are (ii.) hyperpyrexia and meningitis; (iii.) femoral and other thromboses; (iv.) gangrene of the extremities from embolism, bed-sores and pyæmic abscesses; (v.) cardiac weakness, which may remain for a long time, on account of the granular degeneration of the muscle; (vi.) post-febrile mania; and (vii.) paralysis of various parts.

Brill's disease is a mild form of typhus found in the United States of America. It occurs sporadically among immigrants from eastern Europe and is now considered to be due to exacerbations of latent louse-borne infections. *Symptoms.*—The onset is generally rapid; the fever, which is of continuous type, terminates by crisis about the fourteenth day. Frontal headache, mental apathy and profound prostration are notable features. The eruption which is maculo-papular and rarely petechial in type, appears about the fifth day. The mortality is low and does not exceed 2 per cent.

Murine Typhus is world-wide, but occurs especially in Mexico, U.S.A., Palestine, N. Africa, Egypt and Abyssinia. It is similar to but milder than louse-borne typhus; it is transmitted by fleas from infected rats. Unlike the virus of louse-borne typhus, the murine virus produces a characteristic reaction in the tunica vaginalis of male guinea-pigs. Protective measures include anti-rat campaigns, destroying fleas by dusting rat runs and floors with 5–10 per cent. D.D.T., and protective vaccination of individuals.

§ **485. X. Rocky Mountain Spotted Fever.**—*Symptoms:* During the incubation period of four to twelve days irritation and pain may be experienced in the tick-bites. The fever often commences with a slight rigor, and the temperature rapidly rises to 103°, and later to 105° or even 107° F.; the maximum is reached by the fifth to the twelfth day. About the third day the eruption appears in the form of macules on the wrists and ankles; these rapidly spread all over the body, including the face, and may become hæmorrhagic. The spleen is palpable and tender. There may be slight bronchitis and sore throat. Epistaxis and jaundice are not infrequent. Pneumonia is a common complication. Gangrene of the fingers, genitals, etc., may occur. The temperature in favourable cases falls by lysis; if it remains high the patient lapses into a typhoid state and does not recover. Early albuminuria and a leucocytosis with an increase in monocytes are found.

Etiology.—It is due to *Rickettsia rickettsii*, spread by ticks of the genus *Dermacentor* which live on certain domesticated animals and rodents harbouring the infective agent. It occurs not only in the Western States of the United States of America but also in certain Eastern States and in Columbia.

Diagnosis.—The disease resembles typhoid and louse typhus. From the former it is differentiated by the eruption, but it cannot always be distinguished from typhus. Exposure to infection by residence in an infected region may be taken into account.

The *prognosis* varies in different localities. The Western form has a mortality as high as 90 per cent.: but the Eastern form is milder and the mortality is only 5–10 per cent.

Prophylaxis consists in the avoidance of the places which are tick-infested and by destroying the ticks by the application of ammonia, turpentine, etc. The bite may be cauterised with pure phenol. Vaccination by injection of formolised suspensions of triturated infected ticks gives good protection.

Other forms of Tick Typhus occur in the Mediterranean basin (fièvre boutonneuse, transmitted from dogs by the dog tick); also in S. Africa, India, S. America and elsewhere.

§ **486 XI. Scrub Typhus** (Synonyms: Japanese River Fever, Tsutsugamushi Fever, tropical typhus, mite typhus—Sumatra, Australia, India, Burma etc., rural scrub typhus—Malaya).—A typhus-like disease occurring in scattered areas throughout the Far East, Africa, Australia and elsewhere.

Symptoms.—Some five to fourteen days after being bitten by mites the patient develops a shiver, headache, giddiness and fever lasting two to three weeks. This is at first continuous and later remittent in type. Locally there is a small ulcer or ulcers associated with a dark areola and redness, with lymphangitis and enlargement of the regional lymph glands. On the fifth to seventh day a papular and red macular eruption appears involving the face and trunk, limbs, hands and feet. The spleen may be enlarged. When the scab separates it may leave a punched-out ulcer in the second week which may take weeks to heal. The Weil-Felix reaction is strongly positive to *B. Proteus* OXK, and there is a leucopenia associated with a decrease in the neutrophils.

Etiology.—The disease is due to *R. orientalis*, transmitted by the bites of various species of larval *Trombicula* mite, the animal reservoirs of infection being various field rodents. The larva bores into the skin and causes local necrosis and ulceration followed by lymphangitis and adenitis.

Prognosis.—The mortality varies from 5 to 60 per cent.: it is better in the young and in a subsequent than in the first attack.

Prophylaxis.—Vaccination with killed suspensions of cultured rickettsias is a promising experiment. Excellent protection against mites is given by impregnating clothing and bedding with dibutylphthalate.

§ **487. XII. Q Fever** occurs in Australia, N. and Central America, India and the Mediterranean countries, but is becoming more universally recognised.

Symptoms.—It has a sudden onset and manifestations of an atypical pneumonia with fever, lasting 1–2 weeks.

Etiology.—In Australia and America the virus, *R. burneti*, has been found in various ticks and animal reservoirs: in the European outbreaks the method of transmission and source of infection have not been determined.

§ **488. XIII. Trench Fever** (Syn., Weigl's disease and similar fevers of Russia, Poland, Japan and Africa) is characterised by fever of a relapsing type and frequently but by no means invariably, by pains in the shins.

Symptoms.—In the acute type there is high fever for five to eight days, and after an afebrile period there are relapses recurring at five-day intervals. A macular rash, chiefly affecting the thorax and abdomen, is found in about 80 per cent. at the onset or during a relapse. Slight and transient nephritis is frequent which responds well to treatment. In the chronic type the onset shows only a lengthy period of increasing incapacity. In both types the febrile wave is accompanied by severe headache, tenderness of the loin and calf muscles and pains in the shins with nocturnal exacerbations. The spleen is enlarged in about one-third of the cases. *Etiology.*—The cause is probably *Rickettsia quintana*, transmitted in the excreta of lice. *Prognosis.*—Trench fever may run a protracted course, and the patient develop a neurasthenic condition. Disordered action of the heart is a frequent complication. *Treatment* is symptomatic.

§ **489. XIV. Rickettsial Pox** has recently been described in New York as being carried by a blood-sucking mouse mite, which transmits *R. akari*.

Symptoms.—Initially there is a deep-seated single papule which enlarges to form a vesicle: in 1–2 weeks a scab forms which leaves a small scar. A week after the initial lesion there is sudden fever to 103°–104°, gradually declining over the next 7 days. Frontal headache, photophobia and backache may be associated with nausea, vomiting and transient splenic enlargement. Within the first four days of fever a maculo-papular rash appears which vesiculates and gives black crusts.

The *Prognosis* is excellent.

TREATMENT OF THE TYPHUS FEVERS.—Fluids in plenty should be administered, and special care directed to the hygiene of the mouth and the avoidance of bed sores. A fluid diet, including glucose, fruit juices, broths and milk should be given. Intravenous dextrose (5 per cent.) and plasma transfusions are often helpful and cool sponging to lower the temperature is indicated. Lumbar puncture may benefit comatose patients. Hyper-immune rabbit serum or immune horse serum has been advocated: convalescent serum may also be tried. Recently promising results have been reported from administration of *p*-aminobenzoic acid, 4–8 G. initially followed by 2 G. 2-hourly by mouth: vomiting or nausea, induced by the acidity of the drug, is prevented by concurrent oral administration of sodium bicarbonate in dosage sufficient to render the urine alkaline. The new antibiotics chloromycetin and aureomycin are of the greatest value in all forms of typhus. *Prophylaxis*, especially important for doctors and nurses, depends on measures against lice and their excreta and on protective inoculation against the disease. Dichlor-diphenyl-trichlorethane (D.D.T.), 5 per cent. in an inert powder base, dusted on skin and clothes remains lethal to lice for several weeks. Overalls fitting tightly round the neck wrists and ankles may also be used. Masks and goggles may be worn to protect against infection from dried louse fæces in dust of clothes of patients. Protective vaccines are now widely used: they consist of killed suspensions of rickettsias grown in the yolk sacs of developing eggs, 2 or 3 injections each of 1 c.c. being given at intervals of a week or so followed by a maintenance dose of 1 c.c. every six months.

§ **490.** XV. **Anthrax** (Synonym: Malignant Pustule) is due to infection with the *Bacillus anthracis*. The lesion is almost always situated on exposed parts, the dorsum of the hand, arm or face; 82 per cent. of the cases show the pustule on the head or neck. It affects woolsorters, furriers, feltmakers, ragsorters, and others who come in contact with animals or their hides or fur; 40 per cent. of the cases in British leather-workers are due to handling Chinese or East Indian goods. No case has been traced to wet salted hides.

Symptoms.—The incubation period is 24 to 72 hours. First a papule forms at the seat of inoculation, which rapidly enlarges, and becomes on the second day a vesicle, with serous or hæmorrhagic contents and with considerable local œdema. On the third day this bursts, leaving a raw exuding surface, which, on the fourth day, turns to a dry black slough, surrounded by a zone of intense inflammation slightly raised above the surface. Upon this inflammatory zone there appears, also on the fourth day, a characteristic ring of small red vesicles. The œdema extends around, and the lymphatics and the glands inflame. The pain is usually very slight, and no pus forms until about the tenth day, when the slough begins to separate. The constitutional symptoms bear no proportion to the local mischief. The pyrexia may be so slight as not to interfere with the patient's ordinary avocation, and it may not come on until some days after the local signs. Usually, however, it is severe, comes on early, soon assumes a typhoid character, and there is a positive blood culture. *Intestinal* and *Pulmonary* types are also described, according to the method of infection. In the former intense vomiting and diarrhœa occur, with great prostration and cramps, with, in some cases, cyanosis and dyspnœa, and towards the end convulsions and spasms. The spleen is enlarged. In the pulmonary type, due to inhalation of diseased wool or hair (*woolsorters' disease*), there are urgent dyspnœa, and pain in the chest of sudden onset. The temperature rises to 102° to 103° F., and death may occur with profound collapse in twenty-four hours. Sometimes delirium and convulsions, or diarrhœa and vomiting, occur.

Diagnosis.—It may have to be diagnosed in the first place from the sting of an insect, from various conditions which lead to solitary vesicles or bullæ on the second day, from erysipelas (if on the face), lymphangitis, and other causes of œdema. The occupation of the patient assists, but a diagnosis may be made by examining the serum or secretion of the sore, stained by Gram's method (§ 921), under the microscope; the *Bacillus anthracis* is thus readily discovered.

Prognosis.—The mortality varies with the position of the primary lesion, being 40 per cent. when this is on the neck or face, and 12 per cent. when situated elsewhere.

Treatment.—This includes local and general rest combined with the administration of penicillin, 50,000 units 3-hourly subcutaneously, or large doses of sulphathiazole: if œdema is not controlled within two to three days, anti-anthrax serum should be given, 200–500 c.c., repeated every 12 to 24 hours until œdema is checked. If penicillin or sulphonamides are not available, neoarsphenamine may be tried. Mild antiseptics, such as a dilute solution of formalin, should be applied to the lesion.

§ 491. XVI. **Glanders** (Synonym : Equinia) may be defined as a contagious febrile disease attended by a discharge from the nostrils, and sometimes an eruption on the skin, due to the inoculation of the *Bacillus mallei*, in a person attending to HORSES affected with the disease. The eruption, which only occurs in ACUTE GLANDERS, consists of a general erythema, on which a crop of pustules of hemispherical shape appear in the course of a few days or hours. They vary in size between a lentil and a florin. There are also nodules of granulomatous material in the subcutaneous tissue and muscles, which usually suppurate, leaving large foul ulcers. The other symptoms are (i.) a copious discharge of viscid, semipurulent matter from the nostrils; (ii.) pains in the limbs and joints; and (iii.) high fever, with rigors and prostration, passing on to the typhoid state.

In CHRONIC GLANDERS (Farcy) the pyrexia and constitutional symptoms are absent, also the cutaneous eruptions (erythema, pustules, and nodules which leave ulcers and sinuses). The discharge from the nose may be the only sign.

Diagnosis.—The pustules of acute glanders resemble those of variola, but they are larger, are not umbilicated, and the temperature in glanders does not fall with the rash in those cases which present a generalised pustular eruption.[1] The pain and swelling of the joints and limbs bear some resemblance to acute rheumatism, and still more to pyæmia. The reaction to mallein may assist.

Treatment.—At present the disease is extremely fatal. Specific serum and vaccine treatment, as well as drugs, have hitherto been unsuccessful. In FARCY or CHRONIC GLANDERS the death-rate is 40 or 50 per cent. Good results have accrued from inunction by unguentum cinereum, and the use of X-rays or Finsen light.

GROUP II. CONTINUED PYREXIA

§ 492. In this group the pyrexia tends to assume a CONTINUED TYPE —*i.e.*, it runs a continuous course except for the slight normal diurnal variation (§ 471). This group is distinguished from Group I by the absence of an eruption during the first four days of the illness. It is distinguished from Group III mainly by the course of the pyrexia, though aberrant types of one group are found in the other.

Some of the fevers rare in this country have an eruption which develops usually after the fourth day. (See next page.)

TYPHOID FEVER, which may be taken as a type, may in exceptional cases present no other symptoms than *the characteristic pyrexia.* The rash, when present, may be ill-marked, and does not appear till the second week of the disease. In DIPHTHERIA there is the characteristic *throat lesion;* in INFLUENZA there are *pains in the limbs* and a more sudden advent; in PERTUSSIS the *characteristic cough;* and in MUMPS the

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[1] The author once notified a case of this kind as small-pox, and the case passed as such through the hands of two of the most experienced medical officers of the Metropolitan Asylums Board, the mistake not being cleared up until after death, and a full investigation had been made of the *circumstances under which the disease arose.* It was then ascertained that the patient was a stableman, who attended glandrous horses.

parotitis. Various Pathological Tests may aid us in the diagnosis. Cholera (§ 309) and Dysentery (§ 308) might also be included in this group, but the pyrexial disturbance is quite a subordinate feature compared with the intestinal manifestations. Dr. Cabot (*loc. cit.*) analysed 784 cases of fever lasting two weeks or longer without dropping to normal, and found that 90 per cent. were cases of typhoid fever (586), sepsis (70), or tuberculosis (54). Under "sepsis" he included all forms of septic contamination of the blood-stream, as by wounds, abscesses originating from the appendix, gall-bladder, genito-urinary tract, or alimentary canal or empyema (§§ 496 and 516).

Common.		*Rare in Britain.*	
I. The Enteric Fevers (Typhoid and Paratyphoid)	§ 493	VII. Glandular fever	§ 499
II. Diphtheria	§ 494	VIII. Plague	§ 500
III. Influenza	§ 495	IX. Undulant fever	§ 501
IV. Rheumatic fever, pneumonia, and various other inflammatory disorders, usually attended by local signs	§ 496	X. Yellow fever	§ 502
V. Whooping cough	§ 497	XI. Cerebro-spinal fever	§ 503
VI. Mumps	§ 498	XII. Relapsing fever	§ 504
		XIII. Other fevers, rare or unknown in this country transmitted by ticks, sandflies, etc.: Tularæmia, Kala-azar, Phlebotomus fever, Rat-bite fever	§ 505
		XIV. Psittacosis	§ 506
		XV. Bornholm Disease	§ 507
		XVI. Heat stroke	§ 508

The Enteric Fevers include Typhoid and the Paratyphoid Fevers. In young children the clinical picture differs from that in adults, and commonly manifests itself as gastro-enteritis or broncho-pneumonia.

§ 493. I. **Typhoid Fever** is an acute specific fever of about four or five weeks' duration, and is due to the ingestion of typhoid bacilli. In contrast to the fevers in Group I, the onset is insidious but profound toxæmia develops, often attended by successive crops of rose-coloured spots and characteristic ulceration of the Peyer's patches of the small intestine.

Symptoms.—The period of incubation is ten to fourteen days, but may be shorter or longer. There is a stage of increasing illness and bacteriæmia (first week), followed by a continued high temperature and profound prostration (second and third weeks): in cases that recover there follows a slow decline in fever (fourth and fifth weeks) before convalescence is established.

First week.—(i.) The most important early symptom is severe frontal headache: otherwise there are simple malaise and lassitude, some degree of bronchial cough, epistaxis, and disturbed nights. (ii.) Anorexia and nausea are associated with abdominal discomfort, flatulence, and indefinite pain in the right iliac fossa. The bowels are irregular with constipation or temporary looseness of action. (iii.) The tongue is always heavily coated with a white fur. (iv.) The temperature is characteristic (Fig. 117), tending to rise in step-ladder fashion, being higher in the evening than in the morning. Yet the pulse rate is often slowed in proportion to the temperature, and rather soft. (v.) The bacteriæmic

nature of the symptoms in this first week is demonstrated by the frequency with which a positive blood culture can be obtained. A polymorph leucopenia is almost invariable.

During the *second and third weeks* the condition of the patient deteriorates. The *three characteristic features* in this stage are: (i.) The temperature remains up (continued pyrexia) at 103° to 105° F., the diurnal remissions often being no more than are met with in health (Fig. 117). The pulse is slow in proportion to the temperature, is soft and often dicrotic in character and the blood pressure lowered. (ii.) The rash generally comes out about the seventh to twelfth day (average, tenth) in successive crops [1] of small rose-coloured lenticular spots, slightly elevated, soft, and disappearing on pressure. Each spot lasts about three or four days. They are never petechial. They are chiefly met with on the abdomen, sometimes on the rest of the trunk, very rarely on the face or limbs. The number of these spots varies considerably, but they are seldom abundant. They may be very small, and thus be overlooked or mistaken for flea-bites. (iii.) The spleen is generally enlarged during this period. It is seldom large, it may be tender and the lower edge is rather soft, which makes it more difficult to feel. Otherwise, (iv.) Lethargy becomes very marked and gives rise to an aspect which is fairly characteristic (*facies typhosa*): the drowsiness deepens to semi-coma and in severe cases the typhoid state eventually supervenes. (v.) Some diarrhœa is usually present after the first week—at least in cases of moderate severity—and the stools are of a characteristic pea-soup or yellow ochre colour—this feature is of less value as a means of diagnosis if the patient is wholly on a milk diet. The number of stools passed in twenty-four hours is very variable, but tends to decrease in the third week. In more than half the cases there is no diarrhœa throughout, but these include the large proportion of mild attacks: complete absence of diarrhœa is exceptional in cases of any severity. (vi.) Tympanitic distension of the abdomen (meteorism) is common (especially if the patient be injudiciously fed), and there is pain and gurgling in the right iliac fossa, though great care should be used to elicit this symptom, as the intestinal wall is thinned by disease. (vii.) The mouth becomes dry and sordes collect on the teeth and lips. The tongue at first develops a brown fur, but in the second week this clears, and the tongue becomes glazed and dry or red and smooth: shallow transverse fissures are often seen on it. (viii.) A toxic albuminuria is usual.

The *fourth and fifth weeks* are characterised by a gradual improvement in the patient's condition. (i.) The temperature gradually falls in a step-ladder fashion, the reverse of the initial rise (Fig. 117). (ii.) The extreme mental and physical apathy give place to a slowly renewed interest. With increasing appetite weight is gradually regained. (iii.) The stools become more formed and constipation often follows. Prolonged *convalescence* is necessary, for energy is slow in returning. After an apyrexial

[1] This fact may be revealed by enclosing each of the spots which appear on one day by a circle, next day by a triangle, and so on, by a skin pencil or aniline ink.

period, relapse may occur, with a recrudescence of the symptomatology, although such is rarely as severe as the original attack. It is particularly during the third and fourth weeks that the dreaded complications of perforation or hæmorrhage of the ulcerated Peyer's patches are most liable to occur.

The *varieties* of typhoid fever are legion. It is a safe rule to remember that continued fever of any kind may be due to typhoid, whatever symptoms are presented. The predominant symptoms may be those of broncho-pneumonia or of meningitis. In the *septicæmic variety*, the disease commences suddenly with a rapid rise of temperature, vomiting and rigors: intestinal symptoms are usually absent. In the *ambulatory form* the patient keeps about, and perforative peritonitis or intestinal hæmorrhage may be the first manifestation.

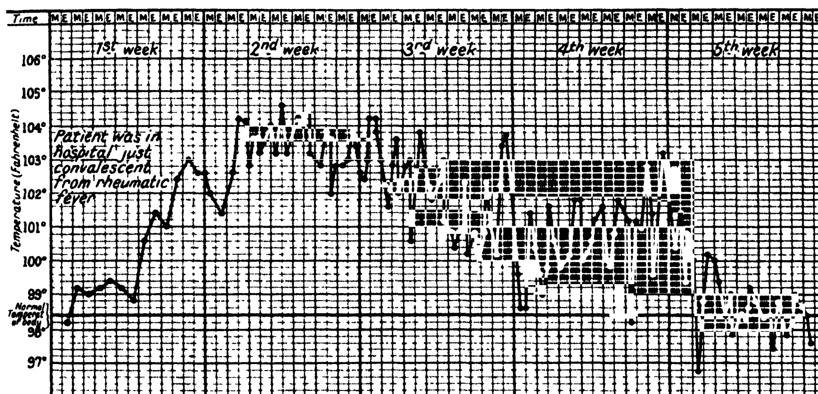


FIG. 117.—TYPHOID FEVER (typical chart), Henry H——, æt. 22, was in hospital when he developed typhoid fever. There was an apathetic mental condition, feeling of profound illness and headache, watery pea-soup stools, and bronchial catarrh. The chart shows the continued character of the pyrexia in the second and third weeks, with gradually increasing remissions in the fourth and fifth weeks.

Diagnosis.—The chief clinical features are the insidious onset and prolonged course of the illness, the profound prostration, the temperature chart and slowed pulse rate, the rash and the enlarged spleen. By cultural methods the organism may be found in the blood (especially in the first week), in the stools (first to third weeks) or in the urine (third week). A blood count which shows a polymorph leucopenia in the presence of a high temperature is highly suggestive of uncomplicated typhoid. The diazo reaction is positive between the fifth and tenth days and in all but severe attacks becomes negative in the second or third week: by itself it is not diagnostic. The Widal reaction is rarely positive before the tenth to fourteenth days, and may be negative throughout: however, a positive reaction is diagnostic. Difficulty may arise in previously inoculated persons: even so in the acute stage the " O " (somatic) antigen titre rises, while the " H " (flagellar) antigen titre remains low throughout.

Undoubtedly many slight cases of typhoid are overlooked and regarded as *Febricula*. Slight cases may also be mistaken for *influenza*, which except for the more sudden advent and brief duration, much resembles mild typhoid. The other *specific fevers* in this group may also have to be excluded. In most cases of typhoid a mild *bronchitis* and *hypostatic congestion* of the lungs occurs, and nothing is commoner than to confuse this with the early stages of typhoid fever. Early headache and delirium may suggest *meningitis*, but the latter is recognised (apart from examination of the cerebro-spinal fluid) by (i.) the retracted abdomen: (ii.) the headache persists longer, and may concur instead of alternating with delirium: (iii.) signs of increased intracranial pressure and local cranial palsies supervene. (It must be remembered that true meningitis and also pneumonia occur due to typhoid bacilli.) *Typhus Fever* has a sudden onset with high fever, a greater tendency to delirium in the early stages, conjunctival injection, a rash on the fourth day which is dark red in colour and does not invade the face, and the Weil-Felix reaction is positive. *Acute Pulmonary* or *Miliary Tuberculosis* sometimes closely resembles typhoid. The positive signs of typhoid are wanting, and the presence of tuberculosis is suggested by (i.) the intermittent character of the temperature and its prolonged course; (ii.) the lung symptoms are much more marked; (iii.) the rapidity of breathing is out of proportion to the other signs of illness: and (iv.) the result of a chest X-ray. *Malignant endocarditis* is recognised by (i.) the intermittent character of the temperature (usually), often with rigors, (ii.) the cardiac signs, and (iii.) the positive blood culture. Pyæmia is differentiated by the wide range and irregularity of the pyrexia.

Undulant fever (§ 501) resembles typhoid in its insidious onset, high fever and enlargement of the spleen, but is distinguished by the patient's serum agglutinating *Brucella abortus* and having no effect on the organisms of the typhoid group. Tularæmia, which, like undulant fever, may attack laboratory workers and cause a prolonged fever, is also distinguished by an agglutination test.

Etiology.—Typhoid fever is due to the typhoid bacillus (the Eberth-Gaffky bacillus). Most epidemics are due to contamination of the water supply by sewage: especially in rural areas this is more common after a dry summer when leakage from cesspools and drains permits contamination of shallow wells. Infection has also been traced from sewage-contaminated waters to oysters and other shell-fish, to ice-creams and to the milk supply. In a patient, *all discharges from the stomach, bowels, bladder and lungs* are infective: thus nurses, and friends of patients contract the disease by handling bed-pans, sheets, and other articles contaminated by these excreta: the discharges become more virulent after standing for twelve to twenty-four hours. The urine and fæces may contain typhoid bacilli long after restoration to health: "typhoid carriers" [1] are persons whose stools have been shown to carry bacilli many years after an attack—the

[1] The best example was "Typhoid Mary," who during her employment as cook in different households and institutions initiated 10 outbreaks with 51 cases.

original nature may not have been recognised. The gall-bladder harbours the organisms which are periodically discharged into the bowel and thus the stools are periodically rendered infective. Second attacks are rare but an attack of typhoid does not protect against a subsequent attack of paratyphoid fever. The greatly diminished incidence of typhoid fever in recent years has been attributed partly to the immunisation of a large proportion of the susceptible population during the two Great Wars, partly to the more careful supervision of water and milk supplies, and to chlorination of any water which may be suspect. The toxins of typhoid seem particularly prone to produce weakness of muscular action: hence the extreme asthenia of voluntary muscle, the myocardial weakness, and the hypotonia of involuntary muscle.

Prognosis.—The case mortality varies in different epidemics from 5 to 20 per cent. It is always a serious disease on account of the numerous complications, prolonged course, and its exhausting nature. The usual duration is four or five weeks, though it can vary from ten days to six weeks even without relapses which are by no means infrequent. Many fatal issues would be avoided if it were remembered that slight attacks require just as much care as severe ones, being liable to hæmorrhage or perforation if the patient does not remain at rest. The prognosis is more favourable between 5 and 10 years of age. It is more serious (i.) in children under 3 and persons over 60: (ii.) when the fever is severe and continued, especially when it remains above 104° F. throughout the second week and especially if the diurnal remissions do not increase, as they should do, in the third week: (iii.) when there are vomiting (except at an early stage), urgent diarrhœa at any time, severe tympanites or hæmorrhage, or marked delirium. A sudden fall in temperature suggests hæmorrhage, or perforation with peritonitis. The most common *complications* are: (1) Pneumonia and pleurisy. Especially towards the end of the third week (2) Hæmorrhage due to the separation of the sloughs from Peyer's patches, occurs in 8–10 per cent. of cases, and for the same reason (3) Perforation with local or general peritonitis is a still more serious complication. Peritonitis arising in typhoid fever is often unattended by the pain so characteristic of that disorder. Its occurrence can then only be recognised by (i.) vomiting; (ii.) great aggravation of the already existing prostration; (iii.) a small rapid pulse (120 to 140); (iv.) immobility followed by distension of the abdominal wall; (v.) sudden frequency of micturition; (vi.) a sudden fall, usually followed by a rise, of the temperature; (vii.) a rising leucocytosis; (viii.) the *facies Hippocratica.* (4) Myocarditis is present to some extent in every case: it can be severe and associated with a general circulatory failure of toxæmic origin, which may prove fatal. (5) Other complications are thrombosis of the femoral or popliteal vein, local suppurations and inflammations, such as parotitis, periostitis, cholecystitis, cancrum oris, and laryngeal ulceration; and, rarely, arthritis leading to dislocation, typhoid spine due to spondylitis (§ 457, 15) and rupture of the rectus abdominis, simulating intestinal perforation. As

sequelæ multiple abscesses, various psychoses, polyneuritis, phthisis, and miliary tuberculosis may occur.

The temperature may rise again after convalescence has begun. Such *recrudescence* may be due to too liberal a diet, excitement, or constipation; or it may be due to a *relapse*, which occurs in about 10 to 15 per cent. of all cases. There is usually an apyrexial interval of about five to ten days, but sometimes the temperature has never dropped satisfactorily. The second attack is usually less severe and shorter than the first, but there may be fatal relapses. As many as five relapses may occur, though more than two are rare in this country.

Treatment.—There are five indications: (*a*) to conserve the energy of the patient, and in the third and fourth weeks to take every care to prevent hæmorrhage or perforation by skilled nursing; (*b*) to give a suitable diet, and avoid meteorism; (*c*) to use such drugs as will support the strength of the patient and reduce diarrhœa and flatulence; (*d*) the use of serum-therapy; and (*e*) to use barrier nursing in its strictest sense. (*a*) *Bed rest* is of the highest importance, and as the patient will probably be in bed for at least six weeks, a sorbo type of mattress is most comfortable. During the third and fourth weeks when the dangers of hæmorrhage and perforation are greatest, the patient should not be allowed to turn himself in bed: perforation may occur if a patient is allowed to raise himself as in changing a draw-sheet. It is a great mistake, however, to keep the patient continually on the flat of his back, as this tends to induce congestion of the lung bases and also the formation of bed sores—he should be carefully turned every two hours by day. Especial attention should be paid to the care of the skin and of the mouth. (*b*) The *diet* now given is of higher nutritive value than used to be the custom, and this undoubtedly supports the patient's strength. Milk in quantities of 2–3 pints a day is the staple diet: it should be sufficiently diluted and barley water, lime-water or sodium citrate (gr. 2 to 1 fl. oz.) added to prevent the formation of curds. Custards, junkets, jellies, eggs, chicken broths, clear soup, beef tea, chocolate and cocoa are nutritious and non-putrefactive: toast, cereals, plain biscuits, and boiled or steamed pounded fish are added if the patient can digest them. Predigested foods such as Benger's are an aid to promote assimilation, especially if the tongue is heavily furred, and pepsin is said to be of service. (*c*) Chemotherapy with chloromycetin may prove effective—give G. 4 initially, then G. 3 daily and when the patient is afebrile follow with decreasing doses. Cardio-vascular weakness is dealt with by injections of nikethamide, and prostration with a very feeble pulse is aided by cortin (10 c.c. twice daily intramuscularly), with an initial intravenous dose of ascorbic acid 1 G. If profuse, the diarrhœa must be checked by enemata of starch and opium (♏ 30 of tinct. opii to fl. oz. 3 of mucilage of starch); or liq. morphinæ ♏ 20, with dilute sulphuric acid ♏ 10, every three or four hours. If these fail, give acetate of lead, bismuth carbonate, or bismuth salicylate. Constipation should never be treated by purgatives, but by glycerin suppositories, liquid paraffin

by mouth, aided by cautious small enemata. If the abdomen is tympanitic, reduce the amount of food and of milk and give it peptonised or more diluted: a flatus tube or a small turpentine enema may help. Hæmorrhage should be checked by the administration of opium, absolute rest must be enjoined, and the amount of the diet temporarily reduced. For perforation, immediate laparotomy and suture of the bowel is usually necessary. (*d*) Felix has recently introduced a serum which has given good results; for adults the dose is at least 50 c.c., followed by two doses of 25 c.c. intramuscularly, daily. (*e*) Typhoid patients may be treated in a general hospital ward, but great care taken by everyone to prevent spreading the infection: the doctors and the nurses must wear gowns, and when handling bedpans and urinals, rubber gloves. The stools must be immersed in and stirred with liq. cresol sap. (lysol) 10 per cent., which is left in contact for 12 hours: the urine and all other excreta, all utensils, bed linen, and the separate thermometer must be disinfected for several hours with 5 per cent. phenol.

Typhoid carriers arise in those persons who harbour the bacilli and excrete them at regular or at irregular intervals. There may previously have been a recognised attack of typhoid, or such may not have occurred, the disease having been mistaken for influenza, etc. Such persons are most dangerous when they handle food, milk or water supplies. The carrier state may be temporary or permanent: for its detection bacteriological testing of the stools and urine is necessary. If the Vi- (Virulence) agglutinins are present, and particularly if with tests at three-monthly intervals the titre is rising, a carrier state must be strongly suspected, and a considerable number of specimens of stool and urine examined. Urinary carriers respond well to sulphonamides, but fæcal carriers may be incurable; cholecystectomy cures 75 per cent. of cases.

Prophylactic Treatment is based on a knowledge of the origin of the disease and its mode of introduction into the system via the mouth. The incidence of typhoid in a community is a fair index of the purity of its water supply: when any doubt arises, and especially in rural areas, the water should be boiled or chlorinated. The carrier state must be carefully searched for, and preventive measures taken to ensure that such persons do not handle food, water or milk. Preventive vaccination, originally introduced by the late Sir Almoth Wright, has proved an established success, as was well exemplified in the fighting forces during the last two Great Wars (for the method, see § 521). The vaccine is usually given hypodermically, but several observers have claimed that oral administration is equally effective. Felix has prepared a particularly efficacious vaccine containing Vi and O antigens. In any case vaccine therapy is not effective in the treatment of typhoid fever.

Paratyphoid Fever is due to infection by B. paratyphosus A, B or C. Paratyphoid A is almost unknown in England and Holland, and is uncommon in Germany, but is common in France, Italy, the Balkan countries, Soviet Russia and the tropics. Paratyphoid B is now commoner than

typhoid fever in this country. Paratyphoid C, which is much less frequent, is prevalent in the Middle East and Mediterranean. Paratyphoid has assumed such prominence that inoculation against the A and B paratyphoid fevers has to be carried out as carefully as against typhoid fever. A mixed vaccine (T.A.B.) is now usually employed (§ 521). It is best distinguished from typhoid fever by cultural examinations and the Widal test. On clinical grounds, differentiation in any individual case is at the best uncertain. However, in paratyphoid infection, the disease tends to be rather less severe, and to run a shorter course : in fact, the temperature may return to normal within 2–3 weeks. It has often been remarked that paratyphoid infection tends to give a much greater profusion of the eruption on the trunk, and that the spots tend to be more obvious as they are darker in colour : but often no rash is visible at any stage of an attack. Intestinal symptoms and complications are not so frequent, in consequence of which the mortality rate in some epidemics is only 1–2 per cent. Even so, it is most unwise to presume on this, and treatment to guard against intestinal hæmorrhage and perforation must be just as strict as in typhoid fever. Mixed infections of two or more of the varieties of enteric fever are not uncommon. *Treatment* is as for typhoid fever.

Enteric Fever in Infants and Young Children usually does not conform to the clinical picture presented above. Whether the cases occur sporadically or in epidemics, it is often necessary at first to make a tentative diagnosis of "pyrexia of uncertain origin." The onset is often sudden, and in infants the presenting symptom is that of gastro-enteritis: in slightly older children, the symptoms and signs are those of bronchopneumonia, of meningitis, or of appendicitis, the origin of which is only established by careful bacteriological study. Even in children dying of the disease, intestinal hæmorrhage and perforation are rare. Relapses are more frequent than in adults.

§ **494.** II. **Diphtheria** is a contagious fever due to the Klebs-Loeffler, or diphtheria bacillus (§ 921). It most commonly involves the throat, but may start on or spread to the nose or larynx, and more rarely the ear, the conjunctiva, the vagina, or a wound of the skin. The organisms rarely penetrate the surface, but multiply and cause the formation of a surface coagulum ("the membrane"). Dangers arise chiefly from the absorbed exotoxins which attack especially the myocardium and the peripheral nerves: in the narrow laryngeal passages of children, obstruction to respiration is serious.

Symptoms of Faucial Diphtheria.—The incubation period is variable, but it is often two to four days. (1) The onset is usually gradual (extending over a day or two), but in some cases is more sudden. (2) There is general listlessness, *pallor*, and often headache and vomiting. A trace of albumen in the urine is common. (3) The temperature is low in proportion to the appearance of illness, and temperatures above 100° F. are unusual: in many of the worst cases the patient is apyrexial. The pulse is soft and rapid. (4) Sore throat and dysphagia, though usual,

are not always complained of by young children. For the first few hours the throat may only be congested. Within twenty-four hours one or both tonsils shows a characteristic patch of creamy-white, wash-leather-like membrane situated on an obviously congested surface, and if forcibly removed this leaves bleeding-points. The patches tend to run together, and to spread beyond the tonsils on to the fauces, soft palate, uvula and pharyngeal wall. The presence of membrane on these parts is a diagnostic feature of great value from simple tonsillitis. The size and rapidity of spread of the membrane, and the amount of œdema present, are an index of the severity of the case. (5) The membrane spreads to

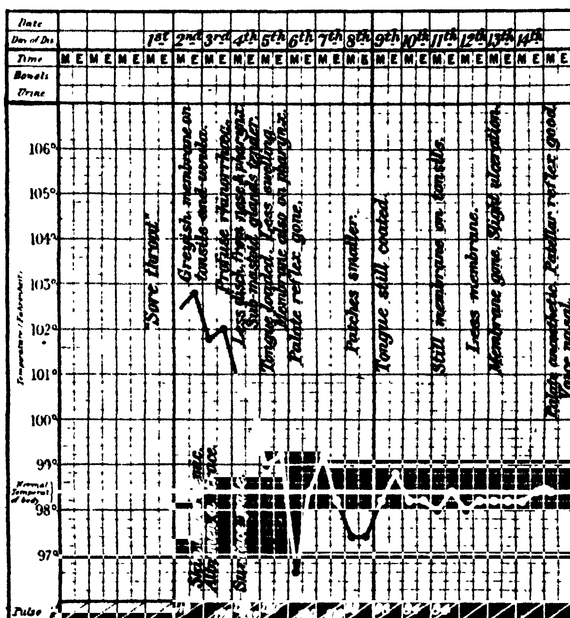


FIG. 118.—DIPHTHERIA.—Male, æt. 9. An ordinary case of faucial diphtheria without implication of larynx. The palate was still anæsthetic one month later. Not followed by paralysis. The different events are indicated on the chart.

the larynx and bronchi in certain cases, and it may spread upwards to the nose (especially in children). An ichorous discharge from the nostrils in a child lying prostrate and fretful in bed is very characteristic of severe diphtheria. (6) The glands at the angles of the jaws are enlarged even in mild cases, and the patient may complain that the neck feels stiff. When the membrane inside the throat is extensive, the glands become much more swollen, and these and the surrounding œdema may give an appearance of "a bull-neck." (7) There is often a diagnostic odour to the breath: recognition of this is most valuable, but depends a great deal on the observer's acuity of smell. In mild cases and particularly in those energetically treated by antitoxin at a very early stage, the

disease aborts in a few days and the membrane separates. In moderate and in severe cases, and especially in those who are not given antitoxin until later, the toxin may have become fixed in the tissues, and the patient's condition then undergoes a change for the worse. Even when the case appears to be mild, and is treated early, these toxic effects may arise, and so every case must be treated by strict bed rest. The three major specific effects of the toxæmia are: (8) In the first week, and in virulent cases, general toxæmia gives rise to death by the seventh day. (9) From the end of the first week and progressively during the second week, myocardial damage and failure occur. Slight cases show a rise of pulse rate, a fall in systemic blood pressure and some enlargement of the heart to the left: in more severe cases, tachycardia is marked (up to 140 per minute), the heart sounds become feeble, the blood pressure may fall to very low levels: and the resulting failure of the cerebral circulation leads to restlessness, drowsiness and coma, while failure of the right heart causes symptoms or signs of this disease (§ 55): vomiting, progressive liver engorgement and suppression of urine occur in fatal cases. Disorders of rhythm, bradycardia, heart block and electro-cardiographic changes have all been recorded. There is a tendency to recovery in a period of weeks, and the heart muscle appears to return to normal. (10) Diphtheritic paralysis is due to polyneuritis. It comes on usually in the third or fourth week, sometimes later. In order of its appearance we find: (i.) it starts in the palate, and therefore a nasal voice or dysphagia is the earliest symptom, and fluids are returned through the nose. (ii.) Next we may get loss of accommodation, with blurred vision on attempting to read. A squint and other cranial nerve pareses are unusual. At this stage loss of the knee and ankle jerks may be seen. (iii.) About the seventh to eighth weeks polyneuritis of the limbs is seen (§ 791). The attitude and gait in children may be characteristic, the little patient walking with a shambling gait, drooping head and shoulders (from weakness of the neck and trunk muscles) and marked foot-drop. (iv.) Especially in severe cases, there may be paralysis of the diaphragm, pharynx and intercostals: the abdominal reflexes are lost only in the severest cases. Temporary involvement of the pyramidal tract, with a positive Babinski, and various forms of motor and sensory impairment occur, such as paraplegia, ataxia, numbness, formication, astereognosis and loss of vibration sense.

Other Varieties of Diphtheria occur in conjunction with faucial diphtheria or alone. The varieties are: (1) Laryngeal diphtheria (membranous croup). *Symptoms.*—The patient is usually under 5 years of age and (i.) there is a croupy cough with a hoarse cry: (ii.) there is inspiratory stridor, followed soon by (iii.) symptoms of laryngeal obstruction. These may be continuous, or intermittent, with inspiratory recession of the ribs, and of the suprasternal notch accentuated by the use of the accessory muscles of respiration, and some cyanosis. (iv.) The child is pale and ill, with a low grade temperature and other signs of diphtheritic toxæmia. (2) Nasal diphtheria, when it occurs alone, is a mild disease of young

children (§ 179. V). (3) The auditory meatus, the conjunctiva, the vagina, and wounds of the surface may harbour diphtheria bacilli. Care must be taken to prove these organisms are pathogenic, and not diphtheroids which are normal inhabitants of mucous membranes and of the skin.

The *Diagnosis* of diphtheria is made by finding the Klebs-Loeffler bacillus in swabbings from a characteristic membranous lesion. In cases of doubt a virulence test must be performed. The diagnosis of the sore throat caused by tonsillitis, scarlet fever, and diphtheria presents certain difficulties, and is given in Table X, § 156. *Follicular tonsillitis* is distinguished by the absence of the definite wash-leather-like patches on the fauces, nasal, or laryngeal passages, and usually the presence of higher fever. There may also be a history of previous attacks, though an inference based on this may be very misleading. Albuminuria, too, is much less common. *Scarlet fever* is distinguished by its abrupt onset, higher fever, rash, strawberry tongue, and generally the absence of membrane from the throat. Simple "*croup*" (catarrhal laryngitis) is distinguished by the history of previous attacks and the absence of patches in the throat, but this is often the case in true diphtheria, in which case an appeal must be made to bacteriology. *Membranous croup* is always diphtheritic. *Vincent's Angina* is distinguished by the bacteriological examination (§ 155*d*), and the fact that the patch is usually depressed instead of being raised above the surface. For differentiating *agranulocytic angina* and the anginose variety of *glandular fever*, see § 155*e*, *f*.

Etiology.—The disease is due to the diphtheria bacillus (*Corynebacterium diphtheriæ*), of which three strains are now recognised, *gravis*, *intermedius* and *mitis*: generally speaking, the gravis strains are the most virulent and produce most toxin, whereas some mitis strains are avirulent to man. The infection is spread by droplet infection and by formites—cups, spoons, etc.: occasionally it is conveyed by milk. Diphtheria carriers may be the cause of local epidemics.

Prognosis.—The case-mortality varied widely in different epidemics, and used to be 25 to 50 per cent.; since the introduction of serum therapy it has fallen to 3 or 4 per cent. Faucial cases in adults are usually mild. During the first week, the disease in little children is often fatal, by the spread of membrane to the larynx. The prognosis is greatly improved when adequate doses of serum are given in the first twenty-four hours: every few hours' delay, especially in virulent cases, increases the risk of complications and lessens the recovery rate. *Untoward Symptoms*.—The prognosis is unfavourable in severe cases, especially when (i.) fœtor of the breath is marked; (ii.) periglandular œdema forming a "bull-neck" is present; (iii.) in the presence of hæmorrhage or epistaxis, purpuric cases being almost invariably fatal; (iv.) marked albuminuria is a bad sign. Other unfavourable symptoms are (v.) a low temperature with severe local lesions; (vi.) when the membrane is extensive, thick and persistent, especially in young patients; (vii.) rapid extension of the membrane to the larynx, leading to croupy cough, dyspnœa and cyanosis. The appear-

ance of a well-marked serum rash within a week of injecting antitoxin is a favourable sign (J. D. Rolleston). *Complications.*—(1) Bronchopneumonia, formerly so frequent in laryngeal cases, now attacks only 4 per cent. since modern treatment is available. (2) Nephritis and dropsy during convalescence are very infrequent, and permanent lesions of the kidney are rare. (3) Otitis media is not uncommon. (4) Embolism secondary to cardiac thrombosis may occur and give rise to hemiplegia or gangrene of a limb from blocking of a main artery.

Treatment.—The indications are (*a*) to neutralise the toxin ; (*b*) to keep the patient at rest in order to diminish the effects of myocarditis and polyneuritis ; (*c*) to inhibit the local process and (*d*) to treat complications. (1) *Antitoxin* will neutralise diphtheria toxin only if it has not become fixed in the tissues. It is therefore vitally important to administer a sufficient dose immediately the condition is suspected or diagnosed, without waiting for bacteriological confirmation. Doses and methods are given in §§ 521 *et seq.* (2) Heart failure is liable to occur about the tenth to the fourteenth day in severe cases. From the commencement of the disease, therefore, the patient must be kept lying down and at *strict rest in bed*—one pillow at the most being allowed for comfort : during this time he must be fed and washed. This position is maintained even in mild cases for two weeks, and for longer in more severe cases, and any subsequent activity will be curtailed immediately evidence of myocarditis shows itself. In a severe case, when myocarditis is likely to be followed by polyneuritis, it is not unusual for the patient to have to remain in bed for 2–3 months. Extra activity is only allowed very gradually. Glucose should be freely given by mouth ; in severe or malignant cases, and in the presence of vomiting, 50 c.c. of 50 per cent. dextrose should be given intravenously once or twice daily. Restlessness must be combated by sedatives and even by small doses of morphia. (3) Antitoxin has rendered *local* treatment by syringing, spraying or swabbing unnecessary in the great majority of cases, especially in young children. Older patients, however, may derive comfort by having their throat syringed by some pleasant lotion such as one containing tinct. lavandulæ or tinct. myrrhæ. Disinfectants are not required. (4) When the larynx is involved steam inhalations and hot applications to the neck give much relief. Tracheotomy or intubation will have to be considered : the results are more satisfactory when *done early*, and all laryngeal cases should be closely watched for the epigastric retraction during inspiration which indicates severe inspiratory obstruction. The instruments must always be at hand and sterile for immediate use, and oxygen may be helpful. In the United States good results are reported from the use of laryngeal suction, by means of a catheter attached to a suction machine and passed through a laryngoscope. The Drinker or Paul-Bragg apparatus is of great use in replacing or augmenting the failing respiratory mechanism in diaphragmatic paralysis. *Freedom from infection* is proved by three negative cultures for C. diphtheriæ, taken from the nose and the throat at not less than two-day intervals. *Prophylaxis.*—A

carrier of virulent C. diphtheriæ may be (1) a person who has just recovered from an attack (convalescent carrier), or (2) one who is innocently harbouring the infection. Especially in the latter the virulence of the organism must be proved. When a carrier state is persistent, any local contributory cause should be treated: it will often respond to systemic penicillin, or in faucial cases to tonsillectomy.

The question of susceptibility to diphtheria is settled by the Schick test. This is described, together with the method of prophylactic immunisation, in § 521, p. 655. Although diphtheria may sometimes occur in the immunised, the attack in the great majority of such cases is mild and fatalities are very rare.

§ **495.** III. **Influenza** is an acute fever which, although endemic in the winter and spring months, is liable to break out in epidemics. It has been known for at least five centuries, and certain of the great pandemics (as in 1918–19) have been attended by a considerable mortality.

Symptoms.—(1) After an incubation period of one to three days, the patient's temperature rises in a few hours to 102°–104°. The onset is frequently attended by severe headache, pain behind the eyes, shivering, anorexia, and pains in the limbs and back which form such a characteristic feature of influenza. (2) The pulse rate is often relatively slowed in proportion to the temperature, and a true bradycardia may occur. (3) The constitutional symptoms, malaise and prostration are out of all proportion to the pyrexia and to the local signs. (4) Catarrh usually accompanies the fever—*i.e.*, there is some redness and watering of the eyes, nasal catarrh, sore throat and a dry cough. (5) The face is flushed and the tongue heavily coated. (6) *Eruptions* of erythematous or urticarial type occur. (7) Some cases present only the above symptoms and signs: but *types of the disease* occur in which different systems of the body are attacked. Some of the symptoms thus presented are of the nature of complications: (i.) The *respiratory tract* is very frequently involved, and laryngitis, tracheitis, bronchitis and pneumonia may arise. (ii.) The *heart* may be affected by myocarditis. (iii.) Involvement of the *alimentary* tract may be evidenced by gastro-enteritis, diarrhœa, vomiting, etc. ("gastric influenza"). (iv.) The *nervous system* may possibly be attacked, and encephalomyelitis occur. Cases of disseminated sclerosis and encephalitis lethargica are attributed to this disease. Influenzal meningitis is due to Pfeiffer's bacillus and not to true influenza.

Diagnosis.—The term "influenza" is often improperly applied to what is really febrile catarrh. In addition to the absence of the influenza virus in febrile catarrh, this clinical distinction is drawn by C. H. Stuart Harris: (1) Premonitory symptoms, such as coryza, sore throat or cough, are uncommon in influenza, in which the onset is sudden, whereas febrile catarrh starts insidiously with a "cold" and fever. (2) The first symptoms of influenza are constitutional rather than respiratory. (3) The cough in influenza is short and dry; in febrile catarrh it is paroxysmal, painful and often productive. (4) Sore throat is constant in febrile catarrh, but is not a feature of influenza. (5) Laryngitis is rarely severe in

influenza, but a very hoarse voice is common in febrile catarrh. (6) Bronchiolitis and pneumonia are the characteristic complications of influenza, and basal bronchitis and broncho-pneumonia of febrile catarrh. Leucopenia is usual in uncomplicated cases.

Etiology.—The agent responsible is a virus, of which two varieties, A and B, have so far been identified. Wilson-Smith, Andrewes and Laidlaw have reproduced influenza in ferrets by intranasal installation of filtrates of throat and nose washings containing the virus from influenza patients, and have found that mice are susceptible to the virus of human and swine influenza, and that this virus can be retransmitted to man. The virus can be grown on the developing chick embryo, and the serum of human convalescents contains specific virus-neutralising antibodies. Pfeiffer's bacillus is not causal, but this organism, hæmolytic streptococci or staph-aureus are responsible for many of the complications. One attack confers no immunity: old and young, rich and poor are attacked alike.

Prognosis.—The case-mortality is about 1 per cent. among the old and young together. In middle-aged and elderly people the respiratory type is very apt to end fatally with pneumonia, and undoubtedly many cases presumed to be primary pneumonia are really secondary to influenza. It is fatal only through its complications. The disease itself is usually trivial, and the patient soon recovers. Relapses are not infrequent.

Complications are chiefly respiratory, and include sinusitis, otitis media and mastoiditis, bronchitis and broncho-pneumonia. These are caused almost entirely by the associated secondary infections. Relapses are common. The *sequelæ* are often more troublesome than the disease itself: (i.) There is a neuro-vascular asthenia, causing weakness in the legs, tachycardia or bradycardia, palpitation, flushings, faintings, perspiration, dyspnœa, and the like. (ii.) Anxiety states, depression, neurasthenia, neuritis and neuralgia may be very persistent. Insomnia can be very troublesome.

Treatment.—There is no specific treatment. During the attack, and for several days after the temperature has become normal, the patient should be kept in bed in view of the sequelæ: aspirin, sodium salicylate and codein will reduce the fever and lessen the pains in the limbs. For the complicating infections, the sulphonamides and/or penicillin are most useful. *Prophylaxis.*—It is well to keep elderly people away from infection during an epidemic. A patient is not infectious to others forty-eight hours from the onset, unless pneumonia ensues, when infection can be transmitted up to 6 days. The prophylactic value of vaccines has not yet been substantiated.

§ 496. IV. Rheumatic Fever, Pneumonia, and other Inflammatory Disorders, which usually present well-marked **local manifestations.**—The three groups of fevers just described are those commonly met in England, in which the pyrexia may run a continued course, and which have no eruption during the first four days. But it must not be forgotten that

certain inflammatory disorders may give rise to pyrexia of a continuous type, and that the usual local signs of these disorders may be absent, at the time when the patient is first seen. It will be well, therefore, to mention those which might be mistaken for an acute specific fever.

(*a*) Obscure (so-called) Local [1] Inflammatory Diseases are mostly met with as complications secondary to fevers. They can usually be detected by a thorough examination of all the organs in the body (§ 473). Nevertheless, certain cases of (1) *pericarditis* or *malignant endocarditis*, or (2) *pneumonia*, *pleurisy*, or *empyema*, may be latent—*i.e.*, the usual physical signs may occasionally be wanting or overlooked. (3) Various affections in or around the *throat, nose and ear;* (4) some *abdominal* disorders, such as cholecystitis, pyelitis, deep-seated abscesses (hepatic, subphrenic, perinephric, tubal), inflammation of the mesenteric glands or pancreas, etc.; (5) certain rare cases of *sarcoma* and *carcinoma*; or (6) inflammation of the *meninges*, tuberculous or epidemic, may also give rise to an elevation of temperature sometimes unattended by marked local symptoms; (7) *parasitic infections*, trichinosis, actinomycosis. In obscure cases of long-continued fever the causes to be suspected are pulmonary tuberculosis, typhoid and undulant fever, deep-seated abdominal abscesses, endocarditis, Hodgkin's disease and syphilis (cp. §§ 516 and 517).

(*b*) Certain General Inflammatory Disorders are attended by pyrexia, which may similarly give rise to difficulties in diagnosis. (1) In *rheumatic fever* and *acute gout* the pyrexia is nearly always continuous. The joint lesions are the cardinal feature in these cases; but it must not be forgotten that acute rheumatism may commence with inflammation of the pericardium (the structure of which very much resembles that of a joint), and that the joint lesions may not be apparent for several days. (2) There are several conditions special to infancy and childhood which are attended by continued pyrexia: (i.) *Infantile paralysis* (acute anterior poliomyelitis) is attended at its outset by a considerable rise in temperature, which may last for several days, and be accompanied by restlessness, peevishness, etc.; (ii.) *meningitis*, tuberculous or epidemic. (3) Septicæmia, and see § 515. (4) Certain blood diseases, especially acute leukæmia and pernicious anæmia, may for a time be overlooked. (5) Examination of the urine may reveal bacilluria, an unsuspected cause of pyrexia. (6) *Constipation* also may cause fever. (7) *A nervous or hysterical pyrexia* has been described, and I have seen the temperature go up in an erratic manner, at odd times, in nervous subjects. But while admitting that the nervous system plays a very important part in the production of fever it is difficult to prove that there is not a compound cause in operation in such cases. Only a thorough *post-mortem* and bacteriological examination would enable us to be certain that none of the many obscure foci of inflammation above mentioned were present.

[1] The word "local" is here used in a qualified sense. Many of these diseases with local manifestations are now known to be due to a general infection.

§ 497. V. **Whooping Cough** (Pertussis) is an acute specific infectious disease characterised by an initial catarrh, and usually followed by paroxysmal attacks of coughing, succeeded by a long noisy inspiration (or whoop) and usually vomiting. The disease is most common in those under five years of age, but adults are not exempt.

Symptoms.—Following an incubation period of 7–14 days there is (1) a preliminary *Catarrhal Stage* which is apt to be overlooked unless enquired for. Running from the nose and sometimes from the eyes is attended by malaise and a low-grade temperature. Soon a short dry cough develops and becomes more persistent, and the individual coughs become grouped together. This catarrhal state lasts up to a week or more, and is followed by (2) *Paroxysms of Coughing.* (i.) These are more noticeable at night and vary considerably in severity. In milder cases there are a series of explosive coughs in rapid succession, followed by a long-drawn inspiration. In typical cases the explosive coughs follow one another until the child has largely emptied the lungs of expired air, and this is succeeded by a loud inspiratory crow or *whoop,* through the narrowed chink of the half-closed glottis. One attack may succeed another, punctuated by a series of whoops, until the child manages to dislodge and cough up a small piece of tenacious mucus, often with *vomiting.* In the process, the face and eyes become more and more congested, and the lips cyanosed, and when the attack is over there is temporary exhaustion. (ii.) The onset of an attack is often recognised by the child who runs to his mother for comfort: attacks are made more frequent by food and by any excitement. (iii.) As a result of the straining cough, the face remains somewhat swollen between the attacks; and subconjunctival hæmorrhages, epistaxis, and a blood-streaked sputum may occur. (iv.) The number and severity of the paroxysms increases to a maximum which is maintained for a week or more, and then starts to decrease. The whoop gradually disappears, but the paroxysmal cough persists for weeks or months after the acute phase has passed. (v.) The temperature is lower than in the catarrhal phase, unless complications ensue, but usually there is some tachycardia: in milder cases the child is apparently quite well between the attacks of coughing, although a puffiness of the face may persist. (vi.) There are no characteristic physical signs in the lungs, and bronchitic signs are generally not as numerous as the severity of the cough would suggest. (vii.) The disturbed sleep and the difficulties of feeding cause considerable exhaustion even for weeks or months. (viii.) There is a tendency for the whoop to return on taking a fresh cold, for months or years, without any return of the original infection.

The clinical *varieties* are: (1) The disease passes through the catarrhal stage to that of the paroxysmal cough, but whooping never eventuates: even in the absence of whooping, repeated paroxysms of coughing succeeded by vomiting, render the diagnosis almost certain. (2) In some adults, and when broncho-pneumonia supervenes in the catarrhal stage in children, the cough may be spasmodic without being paroxysmal.

(3) In one atypical form in infants, attacks of sneezing or hiccough replace the paroxysmal cough.

The *Diagnosis* is usually simple during an epidemic, but otherwise the early catarrhal symptoms may be mistaken for *coryza.* Tuberculosis or other causes of *enlargement of the tracheo-bronchial glands* gives rise to a paroxysmal cough, but the whoop is absent. In the first stage of the disease there is a leucopenia, but during the second stage there is a leucocytosis usually ranging from 15,000 to 27,000, with 70–80 per cent. of lymphocytes. The erythrocyte sedimentation rate is slightly retarded or normal in uncomplicated cases, but rises with any complication. Isolation of *H. pertussis,* either by the cough plate method, or by inoculating a penicillin plate of Bordet-Gengou medium from a post-nasal swab, is the most certain means of diagnosis early in the disease, and is of particular value in atypical and abortive cases.

Etiology.—Bordet and Gengou found that the causal organism is a cocco-bacillus (*Hæmophilus pertussis*) which is most abundant in the respiratory mucus in the catarrhal stage.

Prognosis.—With the decrease of virulence of diphtheria, scarlet fever and measles, whooping cough has become one of the most serious of the specific diseases of childhood, and as such is now compulsorily notifiable in England and Wales. The immediate prognosis depends particularly on the age of the child, as it is more likely to be fatal under one year: otherwise on the severity of the attack, and especially on the occurrence of secondary infections with a hæmolytic streptococcus, pneumococcus or H. influenzæ: then a serious *complication* is broncho-pneumonia, the importance of which has lessened with the advent of chemotherapy. Convulsions in infancy are more liable to occur if there is a tendency to infantile tetany. Spasm of the glottis may be the cause of sudden death. Other complications include cerebral or retro-bulbar hæmorrhages, otitis media, right-sided cardiac dilatation; an ulcer under the frenum of the tongue is due to the forced protrusion against the teeth in the act of coughing. Among the *sequelæ* there is a particular tendency for broncho-pneumonia to be followed by fibroid lung and bronchiectasis; whooping cough may reactivate a dormant tuberculous infection in the chest: and herniæ or prolapse of the rectum are seen.

Treatment.—The child should be nursed in an airy room, with considerable bed spacing from other children to prevent secondary infections being spread from the one to the other. Food should be in small quantities at short intervals, and if a feed is vomited, another immediately after is less likely to set up a paroxysm of coughing. Belladonna is the most useful drug, and aureomycin (given early in an attack) is on trial: children will stand ♏ 10–15 of tinct. belladonnæ if the dose is increased gradually: it may be usefully combined with small doses of chloral and of bromide. Antipyrin, ephedrin and phenobarbitone (gr. $\frac{1}{12}$ at 3 months, gr. $\frac{1}{6}$ at one year t.i.d.) have been advocated. Broncho-pneumonia calls for the use of the sulphonamides and/or penicillin to combat secondary infections:

unfortunately these drugs have no effect on the causal organism. For convulsions, a hot mustard bath or lumbar puncture is most successful: chloroform inhalation may be needed. A period of *isolation* of four weeks from the onset of the whoop is sufficient, but infectivity does not necessarily last as long as this, and many cases can be proved to be no longer infectious after three weeks—by exposing three successive cough plates at intervals of one to two days. The value of vaccines in treatment is not proven, but 10–20 c.c. of convalescent serum is of distinct value in the initial catarrhal stages, or in *prophylaxis*. For this, a vaccine of Phase I organisms is also of value in doses of 5, 7 and 10 billion organisms at weekly intervals above the age of 10 years, the dose being one-fifth of this under one year.

§ **498.** VI. **Mumps (Acute Epidemic Parotitis)** is an acute febrile infectious disorder characterised by inflammatory swelling of one or both parotid glands. The period of incubation is usually 17–18 days, and in exceptional cases up to 4 weeks.

The *Symptoms* usually commence with (1) moderate fever (102° F.). This usually commences insidiously, but may start with a rigor. The pulse is often slowed. (2) There are constitutional symptoms with headache, malaise, anorexia and constipation. (3) Attention may be drawn to the neck by a complaint of sore throat and stiffness in the neck. (4) When looked for, and especially in an epidemic, there is at an early stage, redness around the mouth of the parotid duct on one or both sides. *Characteristic symptoms* appear on the first to the fourth day with (5) pain and swelling of one or both parotid glands. Usually one side is first affected, followed by the other in a day or two: both may be affected together, or one side may escape altogether. The glands are acutely tender, the skin over them is stretched, and trismus may be so marked as to prevent the mouth being opened more than a quarter of an inch. (6) The secretion of saliva is usually suppressed, the mouth becomes dry and the tongue remains furred. (7) The local pain and the deficiency of saliva make swallowing a very painful and difficult process. (8) The blood shows a leucocytosis chiefly due to an increase of lymphocytes. The temperature subsides in three or four days to a week, constipation becomes less troublesome, and the glandular swellings slowly subside, unless complications ensue.

Varieties.—Particularly in epidemics, the submaxillary and even the sublingual glands are also affected. Sometimes the parotid glands escape, and one or more of the other salivary glands are alone involved.

Diagnosis.—Enlargement of a *pre-auricular lymph gland* is unilateral due to some local source of sepsis, and does not involve the deep part of the gland. *Simple and suppurative parotitis* (§ 9) are associated with oral sepsis such as occurs in typhoid and typhus fever, and in abdominal and cachectic states; mumps is almost always bilateral and very rarely suppurates. Care must always be taken to exclude " the bull-neck " of *toxic diphtheria*; *Mikulicz' Syndrome* is usually mistaken for mumps

(§ 9). In the *uveo-parotid syndrome* the parotitis is harder and less obvious, and only the pre-auricular part of the gland is involved.

Etiology.—It is almost entirely confined to children and the young between 5 and 25. It is rare in the very young and very old, but is often epidemic and runs through a school. The infective agent is a filterable virus spread by droplet-infection from the nasal secretions and saliva of patients: the disease can be reproduced from these sources in monkeys, and is most infectious at the end of the incubation period. Death is very rare. The chief *complications* are (1) orchitis, and much less often, oophoritis. Orchitis is very rare before puberty and is most frequent in young adults who are sexually active. It usually follows about 7–10 days from the commencement, when the parotitis and fever have settled. There is sudden pyrexia, with enlargement of one testicle, which becomes very tender and possibly fluctuant; the inflammation settles slowly in a week or ten days, and as usually only one testicle is involved, the incidence of subsequent sterility is low. In some epidemics there may be a swelling of a mammary gland or of a testicle, preceding or accompanying that of the parotids, and cases occur in which there is no parotitis. (2) Meningo-encephalitis is not uncommon in the presence of orchitis: most cases recover. Other complications are (3) albuminuria, most liable to occur in severe attacks and in adults; (4) pancreatitis (§ 256); (5) meningitis, usually ill-developed, but sometimes typical; (6) encephalitis; (7) neuritis; (8) otitis interna, which usually causes permanent deafness; (9) œdema of the larynx secondary to submaxillary localisation of mumps; (10) joint symptoms, usually arthralgia, but sometimes serous or suppurative arthritis. Diabetes mellitus is an occasional sequel. *Treatment.*—Rest in bed is essential until the glands have subsided and the temperature is normal: and the patient is isolated for at least a fortnight. A kaolin poultice to the neck is comforting. Feeding may of necessity be through a straw, and rarely nutrient enemata are required. Orchitis and the other complications are less likely to occur if the patient is kept in bed and the bowels freely opened: sexual stimulation of any kind should be avoided. Infectious precautions with all feeding utensils and handkerchiefs are necessary. *Prophylaxis* by injection of human convalescent serum is of doubtful advantage and has resulted in infective hepatitis.

§ **499.** VII. **Glandular Fever** (Infectious mononucleosis) is an infectious fever occurring in sporadic or epidemic form, in children and adults, probably due to a virus. After an incubation period of five to twelve days the disease shows itself in one of three fairly well-defined clinical syndromes. The GLANDULAR TYPE is the commonest, and occurs chiefly in children and young adults. *Symptoms* are: (i.) Sudden onset with fever of 101° to 103° F., often with vomiting; (ii.) transient sore throat or mild tonsillitis; (iii.) fairly severe frontal headache and limb pains; (iv.) sweating may be profuse; (v.) on the second or third day painful enlargement of the upper cervical glands, which remain discrete and tender. Sometimes they reach a considerable size, and are followed by enlargement of the axillary, inguinal, and epitrochlear glands. (vi.) Abdominal pain and tenderness, with pyrexia and vomiting indicate enlargement of abdominal glands, and may precede cervical adenitis—then appendicitis is often diagnosed. (vii.) Some enlargement of the liver

and spleen is common; (viii.) a painful cough may indicate enlarged mediastinal glands. In the ANGINOSE VARIETY a membrane is present on the tonsils and surrounding œdema is severe—simulating diphtheria. Vincent's bacilli and spirochætes are often present in the membrane. The FEBRILE FORM is most common in adults. *Symptoms.*—(i.) There is a sudden onset, with sore throat, headache and even a rigor. (ii.) Macular, papular or urticarial rashes appear particularly on the trunk, towards the end of the first week. (iii.) Glandular enlargement is relatively late—even in the third week. (iv.) Fever may be prolonged for three or even four weeks; at first remittent, it later becomes intermittent. (v.) Splenomegaly is rare. In all three clinical forms, the course of the disease may be prolonged. In the glandular variety the glands begin to decrease in 5–7 days without suppuration, but may still be palpable months afterwards: the fever often takes two to three weeks to settle and leaves considerable exhaustion. The Wassermann reaction may be completely or incompletely positive. Shortly after the commencement, the blood shows a leucocytosis between 6,000 and 20,000 per cu.mm.: the differential count reveals a large number of mature and immature mononuclear cells, which may constitute 60–75 per cent. of the total: sometimes cells of the lymphocytic variety predominate.

Diagnosis.—Owing to the difference in prognosis the diagnosis from *leukæmia* is very important. In glandular fever the onset is usually sudden, sweating is marked, the cervical glands are usually first involved, and purpura is very rare. In *acute leukæmia* there is often previous malaise, the glands in different areas enlarge simultaneously, anæmia and purpura are common, and the patient progressively deteriorates. An agglutination test (*Paul-Bunnell test*) shows the presence of heterophile agglutinins for sheep cells in the serum: it is positive in many cases of glandular fever towards the end of the first week, in a dilution of 1 in 64 (§ 924).

The *prognosis* is excellent. *Complications.*—A relapse, hæmorrhagic nephritis, or jaundice may occur.

Treatment is symptomatic and convalescence should be reasonably prolonged. The severe anginose form may respond very rapidly to sulpharsphenamine or neoarsphenamine, giving 0·30 G. to an adult on two successive days, even when Vincent's organisms have not been demonstrated: a transient acute laryngeal œdema has been reported following intravenous medication. Sulphonamides should not be used.

The remaining fevers in this group are PLAGUE, UNDULANT FEVER, YELLOW FEVER, *which are met with abroad;* CEREBRO-SPINAL FEVER, *which until recent years has for a long time been rare in this country; and* RELAPSING FEVER, *met with in epidemic form only in times of famine. In* HAY FEVER, DYSENTERY, *and* CHOLERA, *there is some disturbance of the temperature.* WEIL'S DISEASE is described in § 334.

§ 500. VIII. Plague (Bubonic Plague, Typhus Bubonicus, Oriental Plague, the Black Death) may be defined as a highly infectious and fatal fever, characterised by inflammatory glandular and periglandular swellings, hæmorrhages beneath the skin and from the mucous membranes. The last great epidemic in London was in 1666. Its chief epidemic centres in the present day are Northern India, China, Mongolia, and Uganda. Since 1894 there has been a pandemic over most of the civilised world, and our present knowledge of the disease has therefore greatly increased.

Symptoms.—(1) The incubation period is from two to ten days. (2) There is often a prodromal stage, with depression and pains, but usually the onset is sudden, with shivering, and fever rising to 103° or even 107° F. Mental aberration is not uncommon. Prostration is marked, and may be accompanied by vertigo, staggering gait, and lethargy, soon passing into the typhoid state. The spleen and liver may be enlarged. In some cases the speech is halting and staccato, the expression vacant, and the eyes congested; the condition is sometimes mistaken for acute alcoholism. A small vesicle, corresponding to a flea bite, is occasionally observed in the early stages of the disease; and examination of the fluid contents may reveal plague bacilli. (3) On the second or third day a tender swelling of the lymph glands (bubo) appears, the affected group, dependent on the site of the infecting flea bite, being inguinal and femoral in 70 per cent., axillary in 20 per cent. and cervical and submaxillary in

10 per cent. of the patients. The glands rapidly enlarge, pain is intense and suppuration generally supervenes from the seventh to the twelfth day, if the patient survives. (4) Petechiæ and subcutaneous hæmorrhages are not uncommon. A distinctive rash is rare, but when present it resembles typhus. There are six principal *varieties*, which prevail in different epidemics: (i.) The *bubonic* variety is the commonest, glandular swellings occurring in quite 70 per cent. of all the cases. The causal organism is frequently recovered on blood culture. (ii.) The *septicæmic* type is very fatal: the glands enlarge slightly, but they do not suppurate; (iii.) a *fulminant* form, with high fever, little glandular enlargement, vomiting of blood, and death within a few hours; (iv.) a *pneumonic* form, which may be mistaken for bronchitis, influenzal pneumonia, or broncho-pneumonia, attended by intense prostration, no glandular enlargement, and death usually on the third to the fifth day; herpes is absent and the pulse-respiration ratio not so much altered as in true pneumonia; (v.) an *abortive* form, in which there are buboes without much fever, subsiding in fourteen days; and (vi.) an *ambulant* or mild form, with chronic glandular enlargement, great anæmia, and weakness. Intestinal, cerebral and cellulo-cutaneous types are also encountered.

Diagnosis.—Early in an epidemic bubonic plague may have to be distinguished from climatic bubo, soft sore or syphilitic bubo, tularæmia and rat-bite fever. Gland puncture reveals plague bacilli in both smears and culture of the gland juice, and in most of the severe cases of bubonic plague *Pasteurella pestis* can be cultured from the peripheral blood at some stage of the disease. In pneumonic plague the sputum is watery and sanguineous, never viscid and rusty as in pneumonia; the bipolar plague bacilli are present in great numbers in smears of the sputum and this particular form is directly transmitted from individual to individual by droplet infection. Septicæmic plague is diagnosed by positive blood cultures.

Etiology.—Plague is due to the *Pasteurella pestis.* Outbreaks of plague were often preceded by a large mortality among rats and other rodents, and it is now known that the bubonic form of the disease is spread by them. The fleas infesting rats convey the infection to man. The alimentary tract of the flea becomes blocked by a mass of bacilli, the result of growth from infected blood previously imbibed; some of these bacilli are voided during attempts to suck blood and so pass to a fresh victim, being enabled to enter through the puncture made by the flea. Filth and overcrowding predispose to plague. The pneumonic form is directly conveyed from man to man by droplet infection. Age and sex have little influence.

Prognosis.—The case-mortality in the early periods of epidemics is generally 50 per cent. In well-cared-for white patients the mortality varies from 20 to 40 per cent. In the usual course of bubonic plague death occurs before the sixth day; or, if the patient is to recover, convalescence starts between the sixth and tenth day. The pneumonic variety is so fatal that of 43,000 cases in Manchuria only three recovered. Prolonged suppuration of the glands may delay convalescence considerably. The course of the disease is very difficult to forecast. Hæmorrhages usually herald death. The *sequelæ* include boils, pneumonia, dropsy, partial paralysis, and mental disorder.

Treatment.—Careful nursing and fluid diet are essential. Treatment consists of large doses of anti-plague serum intravenously; sulphonamides in full doses have given promising results in bubonic and septicæmic plague, but have so far failed in the pneumonic type. Buboes should be treated with hot fomentations, a kaolin poultice or belladonna and glycerin applications; when suppuration occurs incision should not be delayed. Morphia may be necessary for the pain. Prophylactic treatment consists of the extirpation of rats and the flea vector; dusting floors and rat runs with 5–10 per cent. D.D.T. powder has proved very useful. Prophylactic vaccines are also of value.

§ 501. IX. Undulant Fever. There are two main types: (1) Malta Fever, found particularly in those countries which border on the Mediterranean, in S. Africa, in the southern portions of the U.S.A., and the Punjab, due to *Brucella melitensis.* All ages and both sexes are liable to contract the disease. It is conveyed to man by the milk of infected goats which need not show any signs of ill health. (2) Abortus fever,

contracted from cattle or swine suffering from contagious abortion due to *Brucella abortus* (an organism closely related to *Brucella melitensis*). It is prevalent on the continent of Europe, especially Denmark, in North Africa and the United States. In England many cases are being reported and the disease is by no means uncommon. The disease is conveyed by drinking raw milk, handling the carcases or hides, removing the animal's placenta or slaughtering pigs. Porcine strains of *Brucella* (*B. suis*) are also known.

Symptoms.—The incubation period is fourteen days, though exceptionally it appears to be very much longer; the prodromata include malaise, muscular pains, and dyspepsia. Soon increasing headache, fever, and muscular pains cause the patient to seek advice. The temperature keeps high (102° to 104° F.) for about fourteen days, and may then drop for a few days, only to rise again. After several such undulations the temperature becomes intermittent, with a marked rise at night. The general health of the patient suffers in many ways, the chief symptoms being gastro-intestinal. There are muscular and joint pains, which may be accompanied by considerable swelling, sore throat, sweating, anæmia, enlarged painful spleen, and bronchitis. There are three varieties of the disease. The *malignant* is of acute onset, and runs a rapid course to a fatal termination, preceded by the typhoid state and hyperpyrexia. The *intermittent* variety is of very slow onset, and runs a long course, with sudden elevation of the temperature each evening often with a rigor. The patient does not as a rule make any complaint of specific symptoms until his general health begins to be affected. The *ambulatory* type includes the not infrequent cases in which the *Brucella melitensis* is found in the blood of persons who are in no respect ill.

The *Diagnosis* is arrived at from the clinical signs, by the recovery on culture of the specific organism from the blood or urine (abortus strains grow better with an atmosphere containing CO_2); and by the agglutinin reaction of the blood. An intradermal test, using a killed culture, is frequently positive. In doubtful cases blood should be inoculated into the peritoneal cavity of a guinea pig, as positive cultures are more readily obtained from this source.

Prognosis.—In the common type the mortality is about 3 per cent. Complications are arthritis, neuritis, orchitis, parotitis, mammitis, bronchitis, pneumonia, cardiac failure, and hyperpyrexia, the latter being the usual cause of death. Menorrhagia, abortion and premature labour may result, especially in *B. abortus* infection. The disease may last two years or longer; the average is 3 to 6 months. *Brucella melitensis* infections are usually much more severe than infections caused by *Brucella abortus.*

Treatment.—Prophylaxis consists in avoiding the milk and cheese of infected goats and cows. The milk is rendered safe by boiling. It is important not to handle infected carcases; laboratory workers must be especially careful with Brucella cultures. Careful nursing and a nourishing diet adequate in vitamins is important in so prolonged a disease. Vaccines are of doubtful value; specific anti-serum holds promise, though not yet obtainable commercially. Protein shock in the form of intravenous injection of T.A.B. vaccine, commencing with 50 millions and working up to 250 millions at three-day intervals, and sulphonamide therapy are worth trying. Penicillin is of no value. Where the temperature exceeds 103° F. cold sponging should be instituted. Joint involvement is treated on general lines.

§ 502. X. **Yellow Fever** is an acute infectious disease endemic in a large part of tropical Africa and South America; in severe cases it is accompanied by jaundice, black vomitus and other evidences of hæmorrhage into the mucous membranes or skin.

Symptoms.—The incubation period in man is from 3 to 5 days in mosquito-transmitted infections, and up to 10 days in laboratory workers who have contracted infection from contact with blood containing the virus. Mild, ordinary and fulminating clinical types of the disease are encountered. (*a*) In the *mild* or larval infections there is headache, vomiting and fever of short duration, lasting a few days. Albuminuria is generally demonstrable, but jaundice, if it develops, is mild.

(*b*) In the *ordinary* type three stages are recognised, the *sthenic*, the *remission*, and the *asthenic* stage. (i.) The onset is generally sudden with chilly sensations or a rigor, the temperature rising to 103° or 104° F. on the first day. Frontal headache, backache, pains in the extremities and photophobia are characteristic, the face is flushed, the conjunctivæ injected and the tongue furred with bright red edges. Albuminuria appears about the second day and rapidly increases and the urine soon contains casts; bile salts and pigments are found later. At first the pulse is rapid and bounding, but later it slows and by the third day equals only 60 to 70 per minute despite the elevated temperature. (ii.) About the third or fourth day the temperature drops considerably or falls to normal. (iii.) After the short remission the fever usually returns for 2 or 3 days. In this stage the liver is enlarged and tender, epigastric discomfort is marked, and hiccough and jaundice with hyperbilirubinæmia and a biphasic van den Bergh reaction are common. There is no splenic enlargement. Petechiæ, melæna and black vomit may appear, while oliguria and anuria with nitrogenous retention and acidosis are the rule in fatal cases. Hypotension and bradycardia, due to involvement of the A-V bundle, are characteristic. Death may occur from the 5th to the 12th day. Leucocytes vary from 5,000 to 15,000 per cu.mm. (*c*) *Fulminating* cases develop high fever, purpuric skin rashes, oozing from the gums, early black vomitus and melæna. Jaundice is intense. Hiccough, tremor, delirium and coma due to cholæmia, and uræmia with anuria develop. Death occurs on the 3rd or 4th day.

Etiology.—Yellow fever is due to a filterable virus. Although clinically indistinguishable, three types of yellow fever are recognised, namely, urban, rural and jungle. The first two are transmitted normally by the tiger-banded mosquito, *Aëdes ægypti* (*Stegomyia fasciata*); the third is transmitted by various forest mosquitoes from various animal reservoirs. The blood of man is infective during the first 3 days of fever, but never the excreta. One attack confers immunity and convalescent human serum protects the susceptible monkey, *Macacus rhesus*, when exposed to the virus. The virus may traverse the intact skin in either man or monkey; and in this way many research workers have contracted fatal infections.

Diagnosis.—Important points in diagnosis from other tropical fevers are the severe prostration, early albuminuria, slow pulse, jaundice and the absence of splenic enlargement. In mild cases, intracerebral inoculation into mice of the patient's blood in the early days of the disease produces an encephalitis. Later, and in recovered cases, immune bodies in the patient's blood may be demonstrated by the mouse protection test: mice whose brains have been previously traumatised by injected starch solution are inoculated intraperitoneally with mixtures of virus and suspected serum. If the serum contains no immune bodies, encephalitis results: whereas if immune bodies are present (due to previous infection of the patient), the mice remain well. *Leptospirosis* closely simulates yellow fever, but there is extreme muscular tenderness, neutrophil leucocytosis and a history of a recent immersion accident or an occupational relationship to rats; see § 334. *Malaria* complicated by jaundice, is recognised by the splenomegaly and parasites in the blood, and *blackwater fever* by the hæmoglobinuria. *Infective hepatitis*, *toxic hepatitis* and *acute yellow atrophy* all have a more gradual onset and no stage of remission. In *relapsing fever* with jaundice there is enlargement of the spleen and spirochætes are readily demonstrable in the blood.

Prognosis.—In the average case the mortality is about 20 per cent. Intense and early jaundice, severe nervous disturbances and intractable hiccough, widespread hæmorrhages into the skin and from mucous membranes and anuria are of bad omen.

Treatment.—(*a*) *Prophylactic* treatment includes all measures for the destruction of the mosquito vector, *Aëdes ægypti*, a domestic mosquito which may bite during both day and night. Prophylactic inoculation with chick-embryo cultures of low virulence pantropic virus is protective for four years; the absence of human serum in preparing the vaccine has now removed the risk of homologous serum jaundice

which was previously a serious complication. Rubber gloves should be worn when collecting blood for laboratory purposes, and infected patients should be screened during the first 4 days of fever. (*b*) *Curative* treatment is unsatisfactory. Convalescent serum is of doubtful value once symptoms develop. Careful nursing and abundant fluids during the acute phases of the illness are desirable. Glucose and sodium bicarbonate should be added to all drinks, and 1 to 2 pints of 5 per cent. dextrose may be given intravenously each 24 hours. Calcium lactate gr. 40 daily is useful. Champagne may relieve the vomiting and a mustard plaster over the epigastrium sometimes diminishes hiccough; cold sponging and sedatives are good for the insomnia. Gradual increase of food is allowed after the temperature has been normal several days.

§ 503. XI. **Cerebro-Spinal Fever** (Syn.: Epidemic Cerebro-Spinal Meningitis, Meningococcal Meningitis, Spotted Fever) is due to the meningococcus invading the blood stream from the naso-pharynx, and later reaching the meninges. In some cases two separate stages are evident, with symptoms of septicæmia followed by those of meningitis, but the stages often overlap. *Principal Symptoms:* (i.) In the initial stages there may be nasopharyngeal catarrh: (ii.) fever, accompanied by fleeting joint pains, headache, vomiting and later, skin rashes. The temperature is often irregular at the onset and even subsides to normal for a day or so, before rising again. It is rarely over 102°–104° F. except for a terminal hyperpyrexia. These septicæmic symptoms are accompanied or succeeded by (iii.) symptoms of irritative intracranial inflammation, such as very severe headache, vomiting, photophobia, restlessness, drowsiness and delirium. There is always retraction of the head, and sometimes opisthotonus, owing to the rigidity of the muscles of the back. Hyperæsthesia, especially along the spine, neck stiffness, and severe pain in the back, may be so great that all movement is intolerable. Kernig's sign is usually present. Compression symptoms may supervene later. (iv.) A prominent feature is the presence of an eruption, often symmetrical. Herpes simplex is frequent except in infants, and may have unusual localisation. Urticaria and erythema may occur. On the second day or later a purpuric rash sometimes appears, and may cover the body ("spotted fever"); its frequency varies considerably in different epidemics; in some it has been rare. (v.) Polymorphonuclear leucocytosis appears early. Unusual forms are: (i.) The fulminating type is associated with the Waterhouse-Friderichsen Syndrome (§ 244); in this, septicæmia and often hyperpyrexia are associated with acute circulatory collapse and anuria, cyanosis, purpura and often death within 24 hours. With this (ii.) an acute encephalitis may co-exist or the latter may occur alone. The brain involvement gives deep coma and stertorous breathing, sometimes with convulsions: meningitis is not necessarily present. (iii.) Abortive forms occur with moderately severe headache, fever, and neck stiffness; the C.S.F. shows polymorph cells, but meningococci may be very difficult to demonstrate. (iv.) Chronic septicæmic cases show moderate fever, occasional rigors, muscle and joint pains, headache and rose-red spots, sometimes resembling erythema nodosum: a positive blood culture is obtained, but the meninges may or may not be involved later. (iv.) Posterior basic meningitis of infants.

Posterior Basic Meningitis is a subacute form of this disease occurring in infants from three to twelve months old, characterised by (i.) an acute onset with convulsions and gastro-enteritis, or a gradual onset with drowsiness, vomiting and a meningitic cry. (ii.) A few days later there is the gradual onset of the retraction of the head which may amount to opisthotonos with flexor and extensor spasms in the limbs; (iii.) staring of the eyes, with blindness, appearing quite early in the disease, unassociated with changes in the optic nerves, and due to involvement of the occipital cortex; strabismus is common; (iv.) rigidity of the limbs, which may be localised or confined to one extremity; (v.) paroxysms of high fever lasting a day or two at a time. The onset of *Hydrocephalus* is heralded by *vomiting* and *wasting*, with enlargement of the skull in infants, bulging of the fontanelles and opening of the sutures between the bones. A resonant or "cracked pot" note is present on per-

cussion over the anterior horn of the lateral ventricle in infants in whom the fontanelles are closed.

Diagnosis.—In the septicæmic stage a post-nasal swab shows meningococci. In the meningitic stage, tuberculous meningitis may be differentiated by its more insidious onset and absence of eruption. From tuberculous and other forms of meningitis the best method of diagnosis is by lumbar puncture, when the fluid is found to be turbid, and to contain the specific diplococcus either free or in the polymorphonuclear leucocytes. Care should be taken to exclude anterior poliomyelitis with acute onset, in which a stage of cerebral irritation lasting even as long as seven to ten days is not uncommon. When an epidemic is present the diagnosis is simple.

Etiology.—It occurs sporadically and in epidemics, usually in persons under twenty; some epidemics have occurred chiefly among infants, and males more than females. It is most frequent in winter and spring. It is undoubtedly contagious, although much less so than the acute exanthemata. "Carriers" play the chief part in its spread, overcrowding, especially of sleeping quarters, greatly increasing the danger of transmission which occurs as the result of the droplets of secretion being sprayed around during coughing and sneezing. It is due to the *Neisseria meningitidis* (Diplococcus intracellularis meningitidis of Weichselbaum), which is Gram-negative and best grown on tryp-agar or ascitic fluid.

Prognosis.—The prospect of recovery is not good when the disease attacks infants or old people: even with modern chemotherapy the death rate is 20–25 per cent. Amongst the unfavourable signs are the occurrence of hyperpyrexia, purpura, bronchopneumonia, or circulatory collapse or encephalitis (see above): or an unduly prolonged period of illness. The other common complications are acute arthritis, acute sinusitis and optic neuritis. Amongst the sequelæ may be mentioned deafness, iridochoroiditis, panophthalmitis, subacute arthritis, orchitis, chronic hydrocephalus, and transient paralysis of the limbs, aphasia and dementia.

Treatment consists in isolating the patient and nursing him in a darkened room: all attendants must wear masks and gowns. The results have been revolutionised by the use of sulphathiazole and sulphadiazine: sulphamezathine is not quite so effective. For dosage see Table XXVIII; injections of the soluble salts are used till vomiting ceases, and a copious fluid intake insisted on. When speed is essential, intravenous medication four-hourly, or a continuous drip into a vein is resorted to. Penicillin is not so effective as the sulphonamides, but may be given in addition. Fulminant cases with circulatory collapse may respond to adrenal cortical extract or desoxycorticosterone acetate (5 mgm. injected six-hourly), together with 600 c.c. of human plasma and then 600 c.c. 5 per cent. dextrose in half-normal saline. Apart from the initial diagnostic puncture, lumbar puncture is not usually required. Frequent doses of chloral and potassium bromide āā 20 gr., and occasional injections of morphia may be given with advantage. *Prophylaxis.*—Even as small a dose of sulphadiazine as G. 2 has cleared meningococci from the nose of carriers.

§ **504.** XII. **Relapsing Fever** (Synonyms: Famine Fever, Spirillum Fever) embraces a group of infectious fevers due to *Treponema recurrentis* found in the blood, spread either by lice (widespread form) or by ticks (Central African, Peruvian and American forms). The incubation period varies from five to nine days. The primary fever lasts generally from five to seven days, and short febrile relapses are not uncommon.

Louse-borne Relapsing Fever. *Symptoms.*—(1) The fever has a sudden onset, with rigor, headache, backache, and pains in the limbs. The face is flushed, the eyes injected and photophobia is common. Often there is an initial erythematous rash and later roseolar macules or petechiæ. The temperature rapidly rises and after remaining elevated for six or seven days, returns to normal by crisis. The fall is preceded and attended by profuse perspiration or diarrhœa, or both. This is followed by an interval of about a week, during which the patient feels exhausted, and the pulse and temperature are subnormal. At the end of this time a relapse occurs which is similar to the first attack, but shorter, lasting three or four days. In rare cases there is a second and even a third relapse. (2) Abdominal pain and

tenderness, and definite enlargement of the spleen and liver, are present in most cases. Jaundice and epistaxis are not uncommon in severe cases; sometimes there is vomiting of blood. Delirium is very rare, but if present is of the noisy kind, and occurs at the crisis. Convalescence is slow. (3) Treponemata are found in the blood during the pyrexial period. A neutrophil leucocytosis accompanies the fever and a leucopenia the afebrile period.

TICK-BORNE RELAPSING FEVER is met with in Central Africa, Persia, America and Spain, and never assumes the epidemic proportions of the lice-borne fevers. It differs from the louse-borne form in the shorter duration of the initial fever, in the greater number of relapses and the paucity of treponemata in the peripheral blood. Epistaxis, hæmaturia and jaundice may occur and the central nervous system may be involved with paresis of the cranial nerves and coma; the C.S.F. may show increased pressure and lymphocytosis. Complications are pneumonia, parotitis and iritis. It is often more resistant to treatment, *e.g.* with organic arsenic, than the louse-borne form.

The *Diagnosis of Relapsing Fever* depends on demonstration of treponemata in the blood. Intraperitoneal injection of patient's blood into young white mice or rats may help diagnosis, as treponemata may appear in the animal's blood in 24–48 hours.

Prognosis.—The case-mortality averages about 5 per cent., but may be very high in the African form. Age has not much influence, but dissipation and debility are unfavourable. One attack does not confer immunity from a second. Death, which occurs generally at the height of the first attack, is usually due to syncope, from hæmorrhage or from myocardial degeneration. When occurring later, it may be due to complications. Untoward symptoms include hæmorrhage, suppression of urine, the typhoid state, cerebral symptoms, or indications of a weak heart. A rapid pulse, a high temperature, and even jaundice, are not necessarily unfavourable.

Treatment.—Arsenical compounds (*e.g.*, neoarsphenamine), gold compounds (*e.g.*, solganol), or penicillin should be injected as early as possible and preferably when the temperature is rising: if given when a natural crisis is imminent grave reactions may occur due to rapid destruction of large numbers of organisms. Digitalis, pituitary extract and strychnine may be required to treat collapse occasioned by the crisis. Paraldehyde is useful for the sleeplessness, and lumbar puncture when nervous manifestations are severe.

§ 505. XIII. Apart from the relapsing fever above described, there are several forms of fever transmitted by ticks, sand-flies, etc. The best known of these are: Tularæmia, Kala-azar, Phlebotomus and Rat-bite Fevers.

Tularæmia (Synonyms: Deer-fly Fever, Pahvant Valley Fever, Ohara's Disease). A rodent disease, due to *Pasteurella tularense*, transmissible to man and prevalent in the United States of America, Russia, Europe and Japan; many accidental infections have occurred among laboratory workers.

Symptoms.—Two principal forms of the disease have been described—glandular and typhoid. The glandular form is characterised by fevers, rigors, generalised pain, headache, the formation of a papule which ulcerates, and enlargement of the regional lymphatic glands. It is the type common in butchers, poultry-men and trappers. In the typhoid type there is a fever of varying degree which lasts a considerable time. There are no localising symptoms; it is the type generally found in laboratory workers. The *diagnosis* is made by agglutination of *P. tularense* by the patient's serum or by culture of the organism from the local lesions or glands.

Etiology.—(1) Bite of horse-fly or wood-tick infected with *P. tularense.* (2) Contamination of hands or conjunctival sac with internal organs or body fluids of rabbits, hares, squirrels, water rats or other animals infected with *P. tularense.*

Prognosis.—Convalescence is slow, but recovery usually occurs without sequelæ. The chief complications are bronchopneumonia, pleural effusion, abscesses in the lungs, liver and spleen, peritonitis and meningitis. The mortality rate is very low.

Treatment.—A serum prepared from a goat or horse inoculated with formaldehyde

suspensions of *P. tularense* has been used with remarkably good results. The dose is 30 c.c. given on two successive days. Streptomycin is also proving effective.

Kala-azar.—A disease found in China, India, Assam and the Sudan, associated with enlargement of the spleen and liver, anæmia and leucopenia, some wasting and irregular fever of long duration. It is caused by *Leishmania donovani*, which are found in monocytes in the peripheral blood and in the reticulo-endothelium of the viscera and bone marrow. An infantile form of the disease occurs throughout the Mediterranean littoral. Transmission is by sand-flies.

Symptoms.—The incubation period varies from one to twelve months and the onset is generally sudden with fever; sometimes it is more insidious. When established there are (1) irregular remittent or intermittent pyrexia, the temperature charts sometimes showing a double daily rise in the afternoon and evening; (2) increased pigmentation of the skin; (3) anæmia of secondary type associated with marked leucopenia (1000–5000 cells per cubic millimetre): there is a relative increase in lymphocytes and monocytes, a decrease in neutrophils with disappearance of the eosinophils; (4) loss of weight, and cachexia; (5) splenomegaly, the spleen being first soft and doughy but not tender, and later enlarging and becoming very hard. Diarrhœa, enlargement of the liver, night sweats, asthenia and low blood-pressure may develop. Cancrum oris associated with agranulocytosis, otitis media, hæmorrhage from mucous membranes, purpura and secondary infections like influenza, pneumonia and tuberculosis may cause death. Hepatic cirrhosis, jaundice and post kala-azar dermal leishmaniasis may follow the disease.

Diagnosis.—Kala-azar has to be distinguished from leukæmia, Banti's disease, schistosomiasis, chronic malaria, undulant fever, relapsing fever and typhoid fever. Diagnosis is made by finding the parasites in juice aspirated from the spleen, liver, bone marrow or lymph gland. The formol-gel test is usually positive after two to five months: the serum added to a drop of commercial formalin becomes opalescent within one to two minutes and coagulates solid like boiled egg white in twenty minutes A positive reaction, associated with leucopenia, is valuable evidence of kala-azar. Sometimes the parasites are demonstrable in blood smears and they may be cultured on rabbit blood agar medium at 22° C.

Treatment.—Antimony compounds are generally curative, although refractory cases are encountered especially in the Mediterranean. Sodium antimony tartrate may be used as described for schistosomal dysentery (see p. 385). Sodium stibogluconate, 0·6 G. in 6 c.c. fluid intravenously daily for 7 days, the course being repeated after a week's rest, gives very good results. Other pentavalent antimonial compounds are also used. Ascites and nephritis are indications for care in the use of antimony, and if pneumonia or jaundice supervene the injections may have to be suspended until these complications are cured. Children tolerate a relatively larger dose than adults. In antimony-resistant cases, propamidine or pentamidine (0·1 to 0·3 G. daily for 7 to 14 days) may be used although toxic effects, such as immediate collapse controllable by adrenalin, may occur. Stilbamidine is now little used because of its immediate, general and delayed neural toxic effects which occur especially with old solutions. Cure is indicated by decrease in the size of the spleen, an absence of pyrexia and clinical symptoms extending over a period of six months, a negative formol-gel test, and a permanent disappearance of parasites. Where agranulocytosis with cancrum oris supervenes, injections of pentnucleotide should be given without delay.

Phlebotomus Fever (Synonyms: Papataci Fever, Three Days' Fever) is a fever affecting new-comers in the summer months in Herzegovina, Dalmatia, Malta, Crete, Mesopotamia, Egypt, India, and other parts of the tropics and subtropics.

Symptoms.—After an incubation period of two to seven days the patient has a rigor, followed by severe headache, fever, and severe pain in the eyeballs and brow, back, and calves of the legs. The eyes are congested, the face flushed, the tongue foul. The fever lasts from one to five days, most often seventy-two hours. Vomiting, bradycardia and leucopenia with relative lymphocytosis may occur. The disease is never fatal. One attack confers immunity.

Etiology.—The disease is due to a filterable virus transmitted to man by the bite of a sand-fly (*Phlebotomus* species). White races are specially affected and the virus is present in the peripheral blood during the first two days and can be transmitted by direct inoculation. The *diagnosis* lies between influenza, malaria and dengue, the latter showing secondary rises of temperature and a rash not observed in sand-fly fever.

Treatment.—The disease is best prevented by the destruction of sand-flies which bite at night, and are so small that they can pass through the meshes of an ordinary mosquito net. Repellents, such as dimethylphthalate, are useful. Medical treatment consists of aspirin, phenacetin and caffein citrate or even opium for the pain; cold sponging is beneficial when the fever is high.

Rat-bite Fever (Synonym: Sodoku) has long been described in Japan as occurring after the bites of rats and cats. Cases are also met in Europe and America. There are two varieties: (1) The first is due to *Spirillum minus*, and the *symptoms* are: (i.) There is a history of a rat-bite which is followed by local pain, swelling and a purple-red discoloration; (ii.) this develops into a chancre-like ulcer 1–3 weeks after the bite, with lymphangitis and lymphadenitis; (iii.) there is fever even to 105° F., which recurs at intervals of about six days. It may last a day or a week and assume an intermittent type; (iv.) the fever is accompanied in most cases by a large macular or papular rash; (v.) the blood shows a moderate leucocytosis, but blood cultures are negative: the Kahn reaction may be positive but the Wassermann reaction is negative. (2) The second is caused by *Actinomyces muris* (*Streptobacillus moniliformis*). *Symptoms:* (i.) After the rat-bite the wound heals quickly: but (ii.) within 2–5 days there is high fever, severe arthritis, and sometimes painful nodules in the muscles: (iii.) there is often secondary anæmia and polymorph leucocytosis.

Treatment.—The disease caused by *Spirillum minus* reacts to salvarsan and to penicillin (given for 7–10 days): that due to *Actinomyces* only to penicillin.

§ 506. XIV. **Psittacosis** is a disease of parrots due to a filterable virus. The infection is contracted by inhalation of excreta from infected birds, including the green Amazonian parrot, grey parrots and budgerigars.

Symptoms.—The incubation period varies from seven to twelve days and the onset is acute or gradual, usually acute. Headache is marked, the patient becomes dull and apathetic and a typhoid-like condition may develop; occasionally epistaxis occurs. The spleen is not generally palpable; small rose spots may appear, somewhat resembling those of typhoid fever. Pulmonary symptoms are frequent and may be present from the onset, or develop some days later. There is not much expectoration as a rule, but cough is troublesome. The pulse respiration ratio is low. Physical signs vary, but are not infrequently those of massive consolidation with woody dulness on percussion. The disease may terminate in recovery after two or three weeks. The death rate is from 16–35 per cent. Recrudescence during convalescence is sometimes observed.

Diagnosis.—The diagnosis is made from a history of contact with a sick parrot by the patient affected with an obscure fever resembling typhoid or pneumonia. Elementary bodies may be demonstrated in impression smears from the liver and spleen of mice which have been injected intraperitoneally with infected sputum. Agglutination and complement fixation tests are helpful in diagnosis.

Treatment.—Prophylaxis consists in forbidding the importation of infected birds and in strict quarantine. Sick parrots should be immediately destroyed and cages treated with antiseptics. *Treatment* is symptomatic.

§ 507. XV. **Bornholm Disease.** This disease, also known as "epidemic pleurodynia," "epidemic myalgia" or "devil's grip," is manifested by severe pain in the chest or abdomen with rise of temperature to 102° or 104° F.: the affected muscles are locally tender. The symptoms usually disappear in about twenty-four hours, but recur in nearly a quarter of the cases. The *complications* are pneumonia, pericarditis and orchitis. The disease is very rarely fatal. *Treatment* is purely symptomatic.

§ 508. XVI. **Heat-stroke and Allied Maladies.**—In the tropics or where atmospheric temperature and humidity become unduly high, physiological breakdown may occur as (1) heat-stroke (heat hyperpyrexia, sun-stroke, heat traumatism, heat apoplexy, thermal fever, etc.); (2) heat exhaustion; or (3) heat cramp. Stokers and miners outside the tropics and subtropics may be affected.

Symptoms.—(1) The onset of **heat-stroke** is often sudden, with fever, fits and coma, or there may be premonitory symptoms such as restlessness, giddiness, dyspnœa or gastro-intestinal symptoms. Sweating decreases or stops, and coma with rapid pulse and stertorous breathing or Cheyne-Stokes' respiration follow. Fibrillary muscular twitchings develop, the skin feels burning hot and dry as parchment, the face and conjunctivæ are congested and cyanosis appears. The urine contains albumin, indican and perhaps ketone bodies. Violent convulsions with incontinence of urine and fæces ensue. The knee-jerks are absent. The rectal temperature may be 108° to 112° F., and unless appropriate treatment be rapidly instituted the patient dies with a weakening pulse and respiratory failure. Gastro-intestinal features may be so severe that cholera is suspected. (2) **Heat exhaustion** comes on suddenly with weakness, giddiness and faintness. The temperature is 102° to 103° F., the pulse rapid and cardiac failure may supervene. Constipation is common. Unless properly treated heat-stroke may supervene. (3) **Heat cramp** is due to loss of chloride through excessive sweating, and affects stokers and engineers doing hard muscular work in hot atmospheres. The spasms are most painful, implicate the abdominal muscles and those of the extremities, and may last twenty-four hours or longer.

Diagnosis.—Absence of axillary sweating and diminution in urinary volume and chloride are important warning signals. *Cerebral malaria* with coma may be mistaken for heat-stroke, and vice versa. Splenomegaly and the presence of parasites in the blood films should prevent confusion, though in any case of doubt 10 grains of quinine dihydrochloride in 10 c.c. of distilled water should be injected slowly intravenously. *Cerebral hæmorrhage* into the pons is associated with coma and hyperpyrexia, but the pupils are pin-point in size and lumbar puncture may reveal blood-stained cerebrospinal fluid. The coma of *uræmia, diabetes, alcoholism* and *drugs* is not accompanied by high fever.

Etiology.—All ages and both sexes may suffer. Strenuous work in conventional European clothing under tropical conditions, insufficient intake of water and salt, diseases of the skin affecting the sweat glands, direct exposure to the heat of a tropical sun, a shade temperature exceeding 110° F., lack of air movement and a high humidity, are the factors leading to heat exhaustion and heat hyperpyrexia. Debilitated and intemperate people and patients suffering from malaria and other fevers are especially susceptible. Failure of the mechanism governing heat production and heat loss, circulatory failure secondary to dilatation of peripheral vessels, dehydration from fluid loss, and chloride depletion resulting in cramps, are the means by which a thermal breakdown occurs.

Prognosis.—The case mortality is about 25 per cent. for heat hyperpyrexia. The prognosis largely depends on the rapidity with which treatment is instituted. In the choleraic type and in comatose patients with temperatures over 109° F. the outlook is grave. Even after the temperature has been reduced, debilitated patients may die from cardiac failure and collapse.

Treatment.—In heat exhaustion the patient is put to bed, if possible in a cool room. Citrates, sodium bicarbonate, sodium chloride and glucose are administered with drinks, and, if vomiting occurs, 1 to 1½ pints of normal saline may be injected intravenously. In heat hyperpyrexia hydrotherapy is essential. The patient is placed on a rush or wire mattress under a fan and sprayed with cold water. Ice may also be applied to the head and nape of the neck. As soon as the rectal temperature has reached 102° F. hydrotherapy is stopped. Venesection of 1 pint of blood is performed if cyanosis or convulsions be present. Iced saline enemata and intravenous injections of saline are also helpful, while collapse is treated with nikethamide, strychnine,

pituitary extract and digitalis. Heat cramp responds to hypertonic saline injections intravenously, and the daily consumption of salt should be markedly increased.

HAY FEVER (Hay Asthma), especially the constitutional variety, DYSENTERY, and CHOLERA, give rise to a certain amount of pyrexia of a continued type.

HAY FEVER (§ 179) is recognised by the violent attacks of sneezing.

DYSENTERY (§ 308).—Acute dysenteries may be attended at the onset by some degree of pyrexia, but much the most important symptom is diarrhœa.

In CHOLERA (§ 309) the abdominal cramps, collapse, and diarrhœa are the leading symptoms. During the collapse stage the temperature may be as high as 105° F. in the rectum, although in the axilla and mouth it is subnormal. In the reaction stage, if the patient lives, there is usually a degree or so of pyrexia lasting from a week to a fortnight.

Finally, there are several diseases which in their typical forms belong to Group III or, belonging to Group I, are seen perhaps before or after the eruption comes out, which may present pyrexia of a continued type. It is well in all cases of difficulty or doubt to remember this, and to pass in review the members of all three groups.

GROUP III. INTERMITTENT PYREXIA

§ 509. In this group of diseases the pyrexia is of an INTERMITTENT (or remittent) type—*i.e.*, the temperature drops at regular or irregular intervals to normal (or nearly to normal). This group is distinguished from Group I by the complete absence of eruption. It is distinguished from Group II mainly by the wide variations of the temperature.

Common.		*Rare.*	
I. Malaria	§ 510	Amœbiasis	§§ 308, 336
II. Latent tuberculosis ..	§ 512	Malignant endocarditis ..	§ 50
III. Visceral syphilis	§ 514	Lymphadenoma	§ 572
IV. Acute septicæmia	§ 515	Pernicious anæmia	§ 539
V. Subacute septic conditions	§ 516	Leukæmia	§ 543
VI. Typhoid and paratyphoid fever (some cases) and occasionally influenza ..	§ 493	Opium habit	§ 900
		Trypanosomiasis	§ 518
		Trichiniasis	§ 593

The clinical investigation of these diseases is often attended by considerable difficulty. MALARIA, which may be regarded as the type of this group, is essentially a *paroxysmal pyrexia*, each paroxysm having three stages (cold, hot, and sweating), and each paroxysm being typically *separated by one or more days' interval of apyrexia*, except in certain subtertian fevers. TUBERCULOSIS and SYPHILIS have a daily rise and fall, and are good examples of *regular diurnally* intermitting pyrexia. ACUTE SEPTICÆMIA, on the other hand, is noted for the *irregular* character and wide range of its temperature and the severity of the rigors. CHRONIC SEPTIC CONDITIONS occupy a position midway between these two types—regular and irregular intermitting pyrexia. In a given case of intermitting pyrexia which has arisen in a tropical or subtropical climate, malaria, undulant fever, amœbiasis or tropical liver abscess are probable, but in England the commonest cause is probably latent tubercle. Tubercle as a cause of this type of fever is nearly as common in the tropics as elsewhere. The SERUM REACTIONS aid us to some extent in the diagnosis of this group.

Turning to the rarer diseases, which must always be kept in mind, MALIGNANT ENDOCARDITIS is chiefly remarkable for the *long course* it may run. In LYMPHADENOMA we usually find the enlarged *glands*; and in PERNICIOUS ANÆMIA the skin is very sallow, and the blood picture is characteristic.

It follows therefore that if we have a patient's temperature chart before us, and it shows definite intermissions or remissions, the disease will belong to one of three sub-groups:

A. Regular Intermittent Pyrexia, with one or two days' interval, which contains only one disease—Malaria .. .. .. § 510

B. Regular Intermittent Pyrexia occurring daily, such as Tuberculosis, and Visceral Syphilis .. .. §§ 512 *et seq.*

C. Irregular Intermittent Pyrexia, such as Septicæmia, and other pyogenic processes .. .. §§ 515 *et seq.*

§ **510. Malaria** (Synonyms: Ague, Intermittent Fever, Remittent Fever, Jungle Fever). Malaria is a non-contagious disease caused by at least four different parasites which infect the red blood corpuscles of man and give rise to periodic paroxysms of fever, enlargement of the spleen and anæmia: transmission is by anopheline mosquitoes.

Symptoms.—The incubation period varies from ten to twenty days as a rule, but may be delayed for months. At onset the initial fever may be continuous or remittent in type. Some time elapses before the typical periodic fever, commencing frequently about mid-day with headache and aches and pains in the limbs and joints, develops. The ague paroxysm has three characteristic phases. First, the *cold* stage, lasting ½ to 2 hours, in which the patient, who lies curled up in bed covered with blankets, feels and looks cold, and shivers or has a rigor despite the fact that the internal temperature is rising; the skin may be livid and the nails blue. This is followed by a *hot* stage in which blankets are discarded. The face is flushed, the skin dry and hot, and nausea, a high temperature (103° to 106°) and perhaps vomiting and delirium may ensue; this generally lasts four to five hours. Then begins the *sweating* stage, which lasts one to two hours and is accompanied by a critical fall in temperature and profuse perspiration which soaks the bedclothes. An apyrexial interval follows, its duration being determined by the species of infecting parasite. In *malignant tertian* the cold, hot and sweating stages are less pronounced and the temperature is rarely so high, but the fever generally lasts at least twelve hours and may continue for days. Examination of the patient during the fever generally reveals a tender and palpable enlargement of the spleen, parasites are to be found in the blood and secondary anæmia is frequent. Herpes is commonly seen on the lips.

Varieties of Malaria.—There are three common species of malarial parasites, the so-called benign tertian (*Plasmodium vivax*), the quartan (*Plasmodium malariæ*) and the malignant tertian (*Plasmodium falciparum*). A fourth species, *Plasmodium ovale*, is found in some districts: its effects are similar to those of *P. vivax*. There are several types of periodicity (Fig. 119): (1) Tertian fever with febrile attacks on alternate days (*P. vivax* or *P. ovale* infection): (2) Quartan fever, with attacks every fourth day, and a two-day apyrexial interval (*P. malariæ*): (3) an Irregular or Continuous fever with a tendency to tertian periodicity

(*P. falciparum*): (4) DAILY OR OTHER PHASIC FEVERS are due to double infection by one or more species. Uncomplicated cases of the benign forms of malaria (*i.e.*, *P. vivax*, *P. malariæ* and *P. ovale*) are rarely fatal.

Malignant Tertian malaria (*P. falciparum*) carries a worse prognosis and is much more difficult to diagnose: the infected corpuscles adhere to one another and to the walls of the capillaries producing local tissue anoxia, and the symptoms vary according to the organs chiefly involved. (i.) *Cerebral malaria* causes delirium, stupor and coma, and may give rise to an epileptiform attack, various pareses and hemiplegia. Meningitis may be simulated. (ii.) "*Pernicious malaria*" is a term applied to the

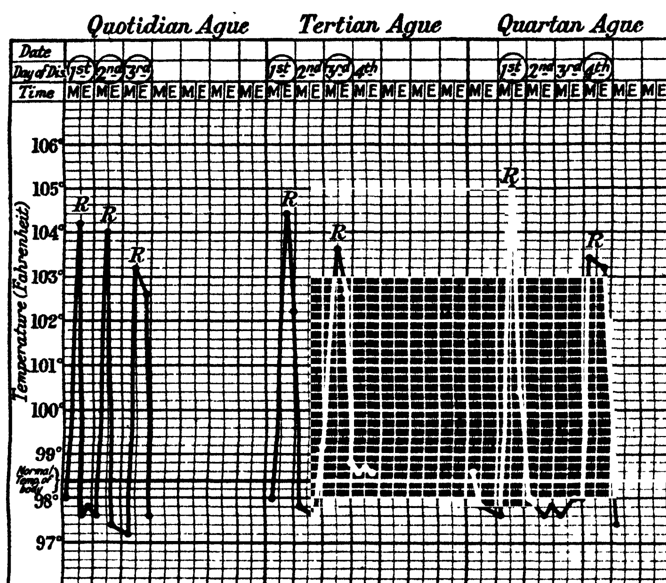


FIG. 119.—TYPES OF MALARIA.—Quotidian (daily) due to two cycles of vivax parasites; Tertian (every other day); and Quartan (every third day). "R" indicates the rigor which ushers in the cold stage.

severe and even grave manifestations of *P. falciparum* infections, which may end fatally unless promptly treated. (iii.) *Hyperpyrexia* causes an internal temperature of 107°–111° F., but the skin may be cold. (iv.) *Abdominal malaria*, by affecting the blood supply to the alimentary tract, may produce epigastric pain, vomiting even of blood-stained material, purging, and even the passage of blood and mucus (choleraic malaria). (v.) "*Bilious*" *remittent fever* may show merely hæmolytic jaundice associated with hyperbilirubinæmia, or toxic jaundice with a biphasic van den Bergh reaction and bile salts and bile pigments in the urine. Bilious vomiting, dark fæces and urobilinuria occur in both varieties, and in some cases very severe hæmolytic anæmia may ensue.

Diagnosis.—The spleen is always enlarged in malaria, though occasionally, especially at the onset or when a patient has been taking quinine,

it may not be palpable at the costal margin. In chronic cases in hyperendemic areas great enlargement ensues and the spleen often extends below the umbilicus or even into the pelvis. Not infrequently the liver is enlarged and tender. Malaria, especially during the primary fever, may be mistaken for other tropical febrile diseases such as typhoid, paratyphoid, relapsing fever and kala-azar: later, periodic fever commencing about midday, splenomegaly, anæmia and the response to specific remedies suggest the diagnosis. In all cases of tropical fever, every effort should be made to demonstrate parasites in the blood (Fig. 128) by taking blood films before treatment is commenced. After full doses of quinine dihydrochloride or of mepacrine B.P. (atebrin), the fever should fall within 96 hours; if it remains up, or rises, provided the drug is being absorbed, the disease is almost certainly not malaria. In the apyrexial periods, urobilinuria, leucopenia and a monocytosis of 12 to 15 per cent. are suggestive of malaria, while hyperbilirubinæmia is not infrequent. Where secondary anæmia is present, polychromasia, anisocytosis and poikilocytosis are frequent findings.

Etiology.—Malaria has a widespread geographical distribution. In the tropics malignant tertian preponderates, giving rise to fatal epidemics; in colder climates benign tertian manifesting a definite seasonal prevalence is met with. In Europe malaria does not occur above the 3000-feet level. All races and both sexes are susceptible and children surviving in hyperendemic areas gradually acquire a relative immunity or tolerance; there is a progressive decrease in the parasitic and spleen rate as age advances.

Life Cycle of Malarial Parasites (see § 532).—(1) Sporozoites are inoculated with the saliva of infected anopheline mosquitoes during the act of biting. (2) On analogy with avian malaria, it is now believed that soon after inoculation into human beings, the parasites first enter cells of the reticulo-endothelial system and the cubical cells of the liver, where they undergo a non-pigmented cycle of asexual development (the exo-erythrocytic cycle). From this source, the red cells later become infected and relapses occur. During this initial cycle, the organisms are relatively insusceptible to anti-malarial drugs, which are so effective in clearing the asexual forms from the blood. In *P. falciparum* infections the exo-erythrocytic stage is short lived, numbering two or three cycles occupying a total of 6–7 days: consequently continuous treatment for 3–4 weeks not only controls the acute attack but, by persistent action in the blood, completely eliminates infection as the reservoir becomes exhausted. In *P. vivax* infections, the exo-erythrocytic cycles persist for several months in indefinite number: consequently no short course of treatment can be guaranteed to eliminate it completely. (3) After the parasites enter the red cells they become vacuolated, develop pigment and enlarge into amœboid-like schizonts. The pigment collects centrally, the chromatin divides and becomes distributed peripherally with its surrounding protoplasm, forming spores (merozoites); these ultimately rupture the corpuscle, escape and re-enter other corpuscles, so continuing the asexual or schizogonous cycle in man. (It is possible that the reticulo-endothelial system may be re-invaded by these blood forms.) (4) From time to time sexual forms (gametocytes) appear in the peripheral blood and when these are sucked up by a suitable anopheline mosquito fertilisation ensues in the stomach of the mosquito. The stomach wall is penetrated, and after a series of local developmental changes the mature oöcyst ruptures and sporozoites are liberated into the body cavity and reach the salivary gland and saliva.

Under satisfactory temperature conditions the mosquito phase of the life cycle takes about ten days.

Prognosis.—In the tropics malaria is a major cause of death, and cases of malignant tertian with pernicious manifestations frequently succumb rapidly if untreated. The benign infections are not so often fatal in the absence of complications. The chronic, repeatedly infected malaria case is liable to develop cachexia, pigmented skin, anæmia and "ague-cake" spleen and may die of intercurrent diseases like sepsis, pleurisy, pneumonia and dysentery, or in the presence of certain food deficiencies develop a hæmolytic nutritional macrocytic anæmia which is often fatal, especially in pregnancy. Blackwater fever may supervene in chronic infections with *P. falciparum.* Rupture of the spleen may follow slight trauma. Nephritis with œdema is not infrequent in quartan malaria. Other complications include neuralgia, iritis, corneal ulceration and retinal hæmorrhages, amnesia, while certain psychoses may follow cerebral malaria. Abortion often occurs. After a patient has left an endemic area, relapses can occur up to two years with malignant tertian fever; up to three years, and exceptionally considerably longer, with benign tertian; and up to seven years with quartan fever.

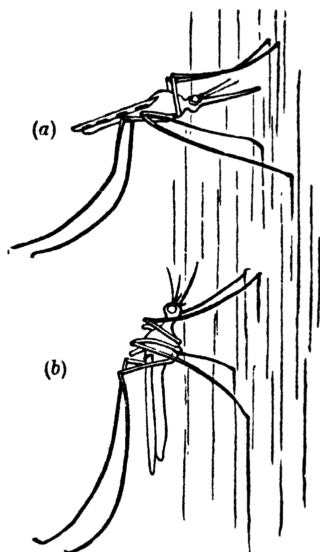


FIG. 120.—MOSQUITOES settling on a wall. There are two chief types of mosquitoes—Anopheline and Culicine—easily differentiated by their attitudes when resting upon a wall. Anopheles (*a*) is the more dangerous one, and is recognised by its spotted wings and its tilted attitude; Culex (*b*) rests parallel to the surface. Anopheline larvæ lie flat on the surface of puddles, whereas Culicine larvæ lie more perpendicularly, and if disturbed rush to the bottom of the pool. Anopheline larvæ are found in puddles which contain algæ and which are too large to be dried up in a week (time needed for the mature insect to be hatched). They are not found in pools which contain minnows, nor in shallow rain pools that are easily dried up. In certain districts they may be found even in rapid streams. 5 per cent. D D.T. in kerosene oil (about ♏ 60 to a pool of 1 square yard) kills all larvæ in six hours.

Prophylaxis.—In order to get rid of the larvæ of the mosquito, marshy tracts and swamps must be drained, cisterns and wells screened, and 5 per cent. D.D.T: in kerosene applied to stagnant or sluggishly flowing water. Bungalow gardens need special attention and, where possible, houses should be made mosquito-proof. Protection against *mosquitoes* is provided by mosquito nets at night, suitable clothing and mosquito boots by day: and the application of repellents, such as dimethylphthalate to exposed parts. *Individual protection* can be ensured by a dose on alternate days of paludrine 0·10 G. In malignant tertian (*P. falciparum*) malaria, this acts as a true causal prophylactic, and kills the parasite in the pre-erythrocytic stage: in benign tertian (*P. vivax*) malaria it is only a

partial causal prophylactic but completely suppresses clinical manifestations. Paludrine probably acts in the same way with *P. malariæ* and *P. ovale* infections. Instead of paludrine, mepacrine 0·10 G. daily can be used: chloroquine as a prophylactic is on trial.

Treatment.—The most efficient anti-malarial drug is the new compound *paludrine* which is remarkable also in the rarity of its toxic effects. Malignant tertian (*P. falciparum*) infections are usually eradicated by 0·10 G. t.i.d. orally for ten days. Benign tertian (*P. vivax*) infections, owing to their long exo-erythrocytic stage, often relapse after a short course of the drug; consequently 0·10 G. t.i.d. is given until the acute attack is controlled: this normally takes only a few days, but after this a maintenance dose of 0·10 G. weekly should be given for six months. Parasites still entering the blood from the exo-erythrocytic reservoir during this period are destroyed by the drug so that clinical manifestations of malaria do not appear, and after six months it is hoped that the reservoir of infection will have become exhausted. The effect of the drug on quartan and ovale infections has not yet been fully assessed. *Mepacrine* B.P. (atebrin) 0·10 G. t.i.d. for five days followed by 0·10 G. daily for three weeks will also eradicate malignant tertian (*P. falciparum*) infections, but stains the skin yellow and sometimes gives rise to gastro-intestinal or other disturbances. *Quinine* gr. 10 t.i.d. is also effective in controlling acute attacks of malaria, but none of the drugs will prevent benign tertian (*P. vivax*) from relapsing. *Pamaquin* B.P. (plasmoquine) 0·01 G. t.i.d., or the much less toxic *pentaquine* 0·02 G. t.i.d., act on the sexual forms of the parasites: in addition they lessen the incidence of relapse but must be combined with a short course of quinine, mepacrine or paludrine. Unfortunately pamaquin is somewhat toxic, and produces gastric symptoms and blueness of the skin (methæmoglobinæmia): at present it would seem satisfactory to rely on a weekly maintenance dose of paludrine to prevent clinical relapses.

Paludrine, mepacrine and quinine control acute attacks of malaria by a schizonticidal effect on the asexual parasites in the blood. Consequently their action is dependent on adequate absorption. In cases of great urgency, such as in "pernicious" or cerebral attacks of malignant tertian (*P. falciparum*) malaria, or where some factor such as persistent vomiting, collapse or coma, interferes with administration by mouth, then intravenous or intramuscular therapy is indicated. A suitable form of paludrine has been prepared for intravenous use. Alternatively mepacrine methanesulphonate B.P. (0·3 G. intramuscularly) may be injected: possibly the most rapid response is obtained with intravenous quinine dihydrochloride (gr. 10), the solution being given very slowly. In grave infections prompt treatment is essential as otherwise irreversible changes may occur in vital organs: one or two parenteral injections usually suffice to control the serious symptoms after which oral therapy can be instituted. In comatose cases removal of cerebro-spinal fluid by lumbar puncture may assist recovery. Intravenous dextrose solution may also be of value in dehydrated and unconscious patients.

§ 511. **"Blackwater Fever"** (Synonym: Hæmoglobinuric Fever), so named from the colour of the urine, is an acute illness developing in patients infected with latent or demonstrable malignant tertian malaria: clinically, it is characterised by the rapid destruction of red blood corpuscles, resulting in hæmoglobinæmia, hæmoglobinuria, fever, vomiting, jaundice and anæmia.

Symptoms.—The onset cannot be foretold. It generally comes on suddenly with chill, fever and loin pain, followed by epigastric discomfort, bilious vomiting and the passage of red urine which in severe cases soon becomes porter-coloured, due to the presence of the blood pigments, oxyhæmoglobin and methæmoglobin. Hæmolytic jaundice follows a few hours after onset and anæmia rapidly develops; 50 per cent. of the corpuscles may be destroyed overnight. Low blood-pressure, pallor, restlessness and cold extremities are characteristic of the early stage. Hiccough and Cheyne-Stokes' breathing often develop in severe cases. The spleen and liver are enlarged and tender and the urine shows albumen, blood pigments, urobilin and a characteristic brown granular sediment containing granular casts; red corpuscles are scanty or absent. Blood chemistry shows a hyperbilirubinæmia and increased blood urea; the plasma contains oxyhæmoglobin and a pigment, methæmalbumin, which had previously been regarded as methæmoglobin. In severe cases anuria and acidosis due to renal failure may supervene. Malarial parasites are sometimes found before and during the first few hours of an attack, but generally soon disappear. The fever generally declines in three to four days, the vomiting lessens and the urine clears; a post-hæmoglobinuric fever sometimes persists. Different clinical types include (1) Transient mild hæmoglobinuria. (2) Fulminating cases, often dying in forty-eight hours. (3) Anuric cases in which oliguria and anuria culminate in death some seven to ten days later with uræmia. (4) Intermittent hæmoglobinuria lasting eight days or longer. (5) Hyperpyrexia followed by death.

Mechanism of Hæmolysis and Anuria. The hæmolytic agent acts intravascularly on the corpuscles, liberating oxyhæmoglobin, and possibly originates from the reticulo-endothelium hypertrophied as a result of chronic malaria. The liberated blood pigment is dealt with by the liver and kidneys. Hyperbilirubinæmia, hæmolytic jaundice and pleocholia with bilious vomiting and dark brown stools result, and absorption of the excess of stercobilin produces urobilinuria. Some of the circulating hæmoglobin is secreted by the kidney as oxyhæmoglobin, and if the urine be acid is converted into methæmoglobin and acid hæmatin which, along with other debris, may block the tubules, but it is now considered that intra-renal vascular changes producing anoxæmia of the kidney are chiefly responsible for the failure of renal function and anuria.

Etiology.—It occurs in various hyperendemic areas of malignant tertian malaria in Africa, India, America and the Balkan peninsula, and generally affects the European, who has been taking quinine irregularly, after from one to five years' residence. The exact cause of the hæmolysis is not known, but it generally follows the administration of quinine or pamaquin. With efficient prophylaxis of malignant tertian (*P. falciparum*) malaria, as by the use of paludrine, blackwater fever should become exceedingly rare.

Diagnosis.—The disease must be distinguished from relapsing fever, leptospirosis and yellow fever by the spectroscopic demonstration of oxyhæmoglobin and methæmoglobin in the urine. The history of residence in malarial countries and of malarial infection is important.

Prognosis.—The case mortality is about 25 per cent. and death results from sudden heart failure, anæmia or anuria. One attack predisposes to another; even if the patient returns to Europe attacks may recur unless the malarial infection be eradicated.

Treatment.—Blackwater fever is best prevented by the eradication of malignant tertian malaria. The patient must be carefully nursed in a recumbent position. Large amounts of fluid containing glucose should be consumed and sodium bicarbonate and citrate given, since the tubules are less likely to be blocked if the urine be kept

dilute and alkaline. The diet must be restricted at first, and proteins only gradually introduced, in view of the renal damage. Intravenous injections of 1 to 2 pints of 5 per cent. dextrose assist the heart's action and help to combat toxæmia and suppression; the latter is treated by hot fomentations, cupping the loins, caffeine citrate and hot colonic lavage (120° F.). Blood transfusion is a life-saving measure in severe anæmia, and should be repeated whenever the red cell count falls below 1,500,000 per cu. mm. Cheyne-Stokes' breathing also suggests the necessity for transfusion. When parasites persist, paludrine or mepacrine should be administered. Iron and liver therapy may be instituted during convalescence.

§ 512. **Latent Tuberculosis.**—Tuberculosis is often said to be latent in the absence of the obvious signs or local manifestations. In all cases of unexplained pyrexia in this country, one of the possible causes to be suspected is tuberculosis in some part of the body. Although in some circumstances the extension of a tuberculous lesion in the lung may be entirely symptomless, and unattended by any appreciable rise of temperature, it is a good clinical axiom that active tuberculosis in any part of the body is usually associated with a daily intermitting pyrexia; and the degree of fever is a fair indication of the degree of activity of the process. Fig. 121 is a chart recorded from a case with active tuberculosis: the temperature drops each morning to (about) normal, and rises each evening one, two, or more degrees, occasionally *vice versa* (§ 513). The patient may seek advice on account of weakness, dyspepsia, loss of weight and other vague symptoms. Such a condition may go on for weeks without any local manifestations, as in the cases referred to under Tuberculous Meningitis (§ 727). The lungs, kidneys, peritoneum, and various other organs may be affected. (1) The commonest locality in adult life is the *lungs*. In this case X-ray evidence of disease often precedes physical signs: when these appear they may resemble bronchitis or simple pulmonary congestion (§§ 117, 131). (2) Apart from the lungs, the *meninges*, *peritoneum*, and other *serous membranes* are perhaps the commonest positions in childhood in which tuberculosis may be present without definite signs. (3) In the *kidney*, tuberculous pyelitis may be readily overlooked, and in suspicious cases the urine should be carefully examined for pus and tubercle bacilli (§ 412). (4) Tuberculosis may also be latent in other situations, such as the ear, spine, intestines, and other viscera; and, finally, the tuberculous process may be generalised, and give rise to *Acute Miliary Tuberculosis*.

§ 513. **Acute Miliary Tuberculosis** may be of the meningeal type, usually known as tuberculous meningitis (§ 727); of the pulmonary type (§ 117); or of the typhoid type, with which we are now concerned. It is characterised by intermitting pyrexia, prostration, and a tendency to the typhoid state—due to a generalised infection of the body by the tubercle bacilli.

Symptoms.—(1) The onset is insidious and for some time there are no localising symptoms or signs. The patient is often a child or young adult who complains of lethargy, which is found to be associated with an evening rise of temperature. The inverse type—*i.e.*, a lower temperature in the evening than in the morning—is said to be more common in this than in any other form of tuberculosis. (2) The lassitude

increases, the daily rise of temperature becomes more marked (Fig. 121), and with this increase of toxæmia there is headache, loss of appetite, often a dry tongue and insomnia. In the course of a few weeks the typhoid state develops, with wandering of the mind, a muttering delirium (especially at night) and progressive cachexia. (3) By now, localising signs have often shown themselves in one or more organs, with an especial tendency to signs of tuberculous meningitis. In others, there are signs of generalised bronchitis, with numerous fine râles scattered over the lungs: sometimes the involvement of the peritoneum is shown by general tumidity, with occasionally a palpable spleen. (4) Investigation of the cerebro-spinal fluid may be valuable, and X-ray examination of the lungs may reveal the typical "snow-storm" appearance of the lung fields.

Diagnosis.—(1) Cases of acute miliary tuberculosis in the early stage are sometimes admitted to hospital as bronchitis, in the later stages as typhoid fever. The

FIG. 121.—ACUTE MILIARY TUBERCULOSIS.—Geo. W——, æt. forty-nine. Seven weeks' history of vague illness before admission, during which time there was profuse hæmoptysis on one occasion. The signs in the chest were very indefinite during life. After death the lungs were sparsely studded with miliary tuberculosis. The liver and peritoneum were also dotted with tiny tubercles hardly visible to the naked eye.

possibility of miliary tuberculosis should be remembered in all cases of "*bronchitis*" attended by an intermitting pyrexia, especially in young adults. The presence of tubercle bacilli in the sputum is unusual; the sputum is usually small in amount, and indeed cough and sputum may be absent. (2) The course of the disease may closely resemble *typhoid fever*: the slowed pulse, rose-red spots and a palpable spleen favour typhoid. The Ehrlich-Diazo reaction occurs in both typhoid and acute tuberculosis, but not the Widal reaction. Choroidal tubercles are sometimes visible on ophthalmoscopic examination, and settle the diagnosis.

Etiology.—The disease is due to a general dissemination via the blood stream (bacteræmia) of tubercle bacilli throughout the body. The usual cause is rupture into a vein of a subacute or chronic focus (often a caseating gland), in a person with a low resistance.

Prognosis.—The disease is almost uniformly fatal in the course of four to eight or more weeks (see § 117). Death occurs by coma, sometimes by pulmonary or other complications. The height and range of the temperature is a fair measure of the virulence and activity of the morbid process.

Treatment.—In such widespread mischief treatment has in the past been uniformly unavailing: now streptomycin is on trial and successes with this have been recorded. As regards prevention, it should always be remembered that convalescence from pulmonary tuberculosis should be very thoroughly re-established before treatment is stopped.

§ **514. Visceral Syphilis.**—There are two different stages of syphilis in which intermitting pyrexia may occur. (*a*) At the first development of the primary roseolous eruption there may be some fever. This is generally overlooked, but at other times it may be accompanied by thirst, loss of appetite, and shivering. It always occurs within sixty-five days of the date of the infection, and is only present if no early treatment be given. (*b*) In the later secondary and tertiary stages of the disease intermitting pyrexia may occur in connection with syphilitic

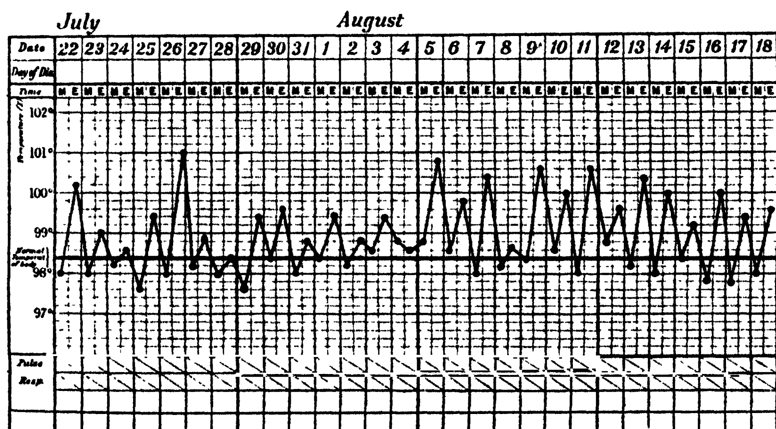


FIG. 122.—VISCERAL SYPHILIS.—Annie L——, æt. sixty-six. The temperature subsided under iodide in large doses, but she ultimately died of exhaustion and hypostatic pneumonia. P.M. —Gummata of liver and bones, hypertrophic cirrhosis, widespread fibrosis of organs.

periostitis, or gummata of the internal organs. Syphilitic lesions of this kind should be considered in cases of prolonged intermitting pyrexia, especially when attended by anæmia. The morning temperature is normal, but in the evening it goes up one, two, or more degrees (Fig. 122). There may also be rigors, nocturnal sweating, and paroxysms of pain in the joints; these symptoms speedily subside when iodide is given. In obscure cases careful investigation should be made of the eyes, liver, ribs, clavicles, and other bones; the Wassermann reaction should be tested, and iodide of potassium tried. In rare instances the fever may be continued and simulate typhoid.

§ 515. **Acute Pyæmia, or Septicæmia,**[1] is a disease characterised by a wide range of temperature, accompanied by rigors and sweating, due to the direct infection of the blood by a micro-organism, usually through some breach of surface in skin or mucous membrane.

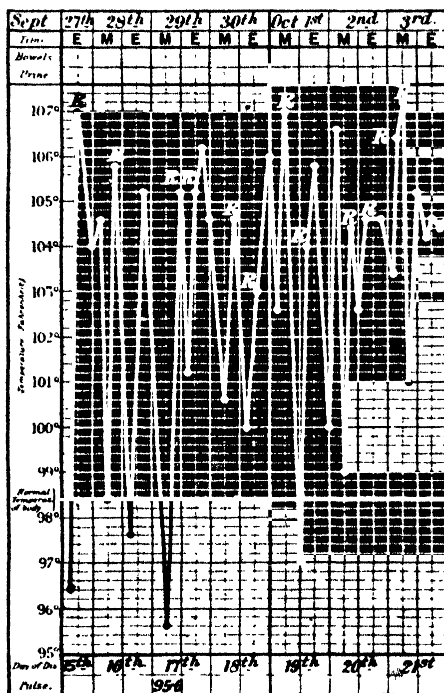


FIG. 123.—ACUTE PYÆMIA (typical of an irregularly intermitting pyrexia).—Catherine W——æt. six, admitted to hospital. She was taken ill somewhat suddenly fourteen days previously with shivering and vomiting. On admission she was in a condition of prostration. There were no physical signs excepting a systolic bruit over the whole cardiac area, and slight enlargement of the spleen. Three days later, there was rusty sputum with streaks of blood: dulness and crepitations over the right back. She was delirious from time to time, and died six days later. At the autopsy pus was found in the mastoid cells and sinus thrombosis secondary to long-standing middle ear disease (of which a history was now obtained), infarcts in the kidney, and pyo-pneumothorax secondary to rupture of one of the gangrenous-looking abscesses of the lung.

The *Symptoms* are (1) pyrexia, which runs a very characteristic course, and is distinguished from all other diseases not of septic origin by the *wide* and *very irregular* range of the temperature (Fig. 123). The remissions may occur several times a day, and have not the diurnal regularity which marks the two preceding classes of disease (§§ 512 and 514). There may be as much as 6° or 7° difference between the temperature in the

[1] In *Bacteræmia*, living organisms are present in the blood stream but do not produce clinical symptoms and signs. *Septicæmia* is a clinical rather than a pathological conception. A bacteræmia is present, and in addition the patient exhibits signs and symptoms of infection of the blood stream. *Pyæmia* is a condition of metastatic abscess formation due to the presence of infected thrombi in the blood stream.

course of a few hours: when it rises suddenly the temperature is often accompanied by a rigor, followed by very profuse perspiration and a rapid fall. The pulse is rapid and compressible. (2) Toxic Symptoms include prostration, headache, anorexia, nausea, a dry furred tongue, often constipation, and aches and pains in the muscles: the skin is sallow, and anæmia may develop. The mind is clear at first, and remains so for a considerable time, but towards the end there is a tendency to the typhoid state. (3) Later on in the disease emboli may occur in different parts of the body: in the lungs they give rise to a generalised congestion and patches of pneumonic consolidation or abscess (as in the case given in Fig. 123): the spleen may become palpable: and deposits of pus may occur in or around the joints or in other parts of the body. The serous cavities may contain pus, constituting empyema or pyo-pericarditis. The leucocytosis and other changes in the blood may aid diagnosis; a positive blood culture clinches the diagnosis, and should always be performed when a *local* cause for pyrexia cannot be found.

Portal Pyæmia is a condition where the primary focus is in the alimentary tract, the veins of which drain into the portal vein. Appendicitis is the commonest cause and the abscesses are usually confined to the liver. A high leucocytosis with a negative blood culture aids the diagnosis of portal pyæmia.

Acute Osteomyelitis (Acute Periostitis) is a pyæmic process which may set in very suddenly, usually after an injury to one of the superficial bones, generally the tibia. In children there may be no history of injury. The diagnosis is easy when the tissues round the diseased bone are swollen, but during the first day or two pain is often complained of near a joint, and may lead one to diagnose rheumatic fever.

The *Diagnosis* of septicæmia is easy when there is an external wound or abrasion, and should never be difficult when there is the wide variation of the temperature, coupled with the rigors and the sweats. The chart of a typical acute case is like nothing else. When due to some internal cause, it may resemble malignant endocarditis, typhoid fever, coli bacilluria, pneumonia, malaria, remittent fever and acute rheumatism. But when carefully recorded temperatures of several days are available, and a thorough examination of the organs is made, the diagnosis should not be difficult. *Streptococcal* septicæmia is marked by high fever, rapid pulse (over 120), joint pains, skin rashes, rapidly developing anæmia, hæmaturia and often diarrhœa. *Staphylococcal* septicæmia is often associated with a previous history of boils or carbuncles, an initial slow pulse and a tendency to form abscesses in the renal cortex, bones and joints. With *pneumococcal* infections there is no primary focus, a hot dry skin, herpes labialis and a considerable rise in the respiration rate even in the absence of lung signs.

Etiology.—The principal cause is the introduction of a virulent organism either through the *skin* or through a *mucous membrane*. In addition there is often a particular susceptibility of the patient. (1) A mere prick or scratch of the skin may permit the entrance of micro-organisms: cases have been caused by doctors and nurses becoming infected by an accidental prick or cut during a surgical operation or dressing an infected

wound. In hospital practice there may be a spread into open wounds of virulent organisms (especially hæmolytic streptococci) from the nose or throat of a carrier, from soiled fingers and instruments, and even with dust particles. Similarly organisms can pass through mucous membranes, even without a visible abrasion: in this manner a variety of bacteria and viruses can enter the blood stream through the tonsils, nasopharynx, mastoid cells, bronchial mucosa, and the intestinal biliary and urinary passages. Special attention should be directed to the uterus: *recent abortion, perhaps criminally induced, should always be borne in mind when a young woman is admitted with septicæmia.* After recent parturition, the surface of the uterus resembles an open wound, and offers a large surface for the passage of organisms directly into the venous sinuses, by introduction of an infected glove or instrument: here again the original source may be a droplet infection from the nose or throat of a doctor or midwife. The disease is then called PUERPERAL FEVER, or Puerperal septicæmia: when the infection is derived from a previous case of puerperal septicæmia it is especially virulent due to the phenomenon of *passage*. (2) A lowered resistance is due to hygienic causes—overcrowding, lack of fresh air and sunlight, and to poverty: or to special *predisposing causes*—diabetes mellitus, alcoholism, hyperthyroidism, chronic nephritis, agranulocytosis, and ill health due to bad teeth or infected tonsils.

The *Prognosis* has been remarkably altered by modern chemotherapy. Whereas cases of intense septic infection from a wound or from parturition used to run a rapid and fatal course in ten to twelve days, nowadays the majority respond to a sulpha-drug, penicillin or streptomycin when given early and in adequate doses. There still remains a small proportion of cases in which the disease is so fulminant that treatment has no time to be effective; or the responsible organism may be resistant to all chemotherapeutic agents at present available. Not all cases are of this extreme virulence, and there are a number in which small quantities of septic matter are constantly leaking into the general circulation from some *internal* source over many weeks or months, especially when there is an untreated or undrained primary focus (as in the patient referred to in Fig. 123). There is, in fact, no hard-and-fast line to be drawn between the *acute* septicæmia now under consideration and the *subacute* and *chronic* septicæmia due to pent-up pus or ulceration described below (§ 516). Acute pyæmia is most serious and, if untreated, often fatal. Death may occur either by the intensity of the infection, asthenia, or complications. The *untoward symptoms* are a very high temperature, frequent rigors, or cerebral symptoms. The most frequent *complications* are (1) bronchopneumonia, which invariably occurs in severe cases; (2) pericarditis, peritonitis or pleurisy, which usually becomes purulent; and (3) suppurative inflammation of the spleen, liver, brain and other organs, consequent on the infected emboli; (4) malignant endocarditis. Among the sequelæ in certain less acute cases which recover may be mentioned a destructive form of arthritis.

Treatment.—The indications are (1) to find the cause and to identify the invading organism; (2) whenever possible to administer chemotherapeutic drugs which will lead to the destruction of the causal bacteria; (3) to relieve the symptoms and support the strength of the patient. (1) The *source of the infection* must first be identified. Subsequently drainage of the infected material will be instituted: this may necessitate surgical drainage, *e.g.*, of an abscess cavity, osteomyelitis, etc. (2) *Chemotherapy* indicates the use of drugs which exert a bactericidal or bacteriostatic effect on the invading organism, but without serious detriment to the patient. Salvarsan was the first of these substances discovered by Ehrlich. Modern chemotherapy has led to the use of sulphanilamide and its derivatives, the *sulphonamides* or "sulpha-compounds". These drugs act by inhibiting the multiplication of susceptible organisms (bacteriostasis), thus allowing the defensive mechanisms of the body to destroy them. Sulphanilamide and sulphapyridine have now been mainly replaced by sulphathiazole, sulphadiazine, etc. The drugs most in use are shown in Tables XXVIII and XXIX. They are administered by mouth, after crushing the half-gramme tablets, or intravenously as the sodium salts and well diluted: for surface wounds sulphanilamide powder can be locally applied. It is usual to give a large initial dose and to maintain an adequate blood concentration by four-hourly doses subsequently. During their administration plenty of fluid and an alkaline mixture must be prescribed, as otherwise the drugs or their acetyl derivatives may crystallise out and produce blockage of the renal tubules. Other toxic effects include nausea, mental depression, skin rashes, and drug fever: it is advisable to discontinue administration after a week, unless the blood is watched for leucopenia and agranulocytosis. In certain cases it is helpful to determine the sensitivity of the organism to the particular sulphonamide beforehand, to ensure an adequate therapeutic effect. *Penicillin* is an antibiotic and chemotherapeutic substance: originally derived from the mould *P. notatum* and now mainly from *P. chrysogenum*, it has revolutionised medical treatment. Penicillin acts chiefly as a bacteriostatic and bactericidal agent on a wide variety of susceptible organisms (Table XXIX) and the penicillin sensitivity of organisms can be determined beforehand in the laboratory. It is usually administered by three-hourly subcutaneous or intramuscular injection, its dose being measured in Oxford Standard units (Table XXX). In its more purified forms, it is almost free of undesirable side-effects and even considerable over-dosage produces no toxic effects on the patient: drug fever and urticaria are more common with the less-purified products. It can also be used locally by injection into abscesses, empyemata, and into the cerebro-spinal canal, as well as locally into wounds, as pastilles in the mouth, and by inhalation as an ærosol. *Streptomycin* is derived from the soil organism *Actinomyces griseus*, and differs from penicillin in being effective against such gram-negative organisms as those of the coli-typhoid group, and against the tubercle bacillus (Table XXIX). As it is less rapidly

Table XXVIII.—Average Doses of some of the Principal Sulphonamide Preparations as at Present Recommended.

Disease	*Drugs Recommended with Initial and Subsequent Doses in Grammes*	*Remarks*
Pneumococcal Pneumonia.	*Sulphadiazine, Sulphamezathine, Sulphamerazine* or *Sulphathiazole.*	Especially useful against types 1, 7 and 10: less useful against types 2, 3 and 5 (Whitby). Blood concentration should be 5–10 mgm. per cent.
Adult	2 : 2 : then 1 four-hourly for 36 hours	
Child 1– 3 months.	¼ : Then ⅛ four-hourly for 36 hours	
6–24 „ .	½ : Then ¼ four-hourly for 36 hours	
3 years . .	¾ : Then ⅜ four-hourly for 36 hours	
5 „ . .	1 : Then ½ four-hourly for 36 hours	
Hæmolytic Streptococcal Infection (including Puerperal Fever).	*Sulphadiazine, Sulphamezathine, Sulphamerazine, Sulphathiazole* or *Sulphanilamide.*	
Severe	1st 24 hours, 1 : 1 : 1 : 1 : two-hourly; then 1 : 1 : 1 : 1 : six-hourly. 2nd 24 hours, 1 four-hourly. If vomiting: Sodium Salts of drugs 2 G., then 1 G. four-hourly intramusc., or well diluted with N-saline intravenously.	Doses decreased as temperature falls, but continued at about 2½–3 G. daily for at least 5–6 days after temperature is normal (Colebrook).
Moderate and mild	1st 24 hours, ¾–1 four-hourly, reducing as temperature drops to 1 t.d.s. for several days after temperature is normal.	
Meningococcal Meningitis.	*Sulphadiazine, Sulphamezathine, Sulphamerazine* or *Sulphathiazole.*	Blood concentration should be 5–10 mgm. per cent. within 12 hours of starting treatment. Can use 20 c.c. saturated solution (0·5 per cent.) Sulphanilamide in N. saline intrathecally, for initial dose. Can combine with penicillin therapy.
Child 0– 2 years .	1 : Then ½ four-hourly.	
2– 5 „ . .	1½ : Then ¾ four-hourly	
5–10 „ . .	1½–2 : Then ¾–1 four-hourly	
10–15 „ . .	2–2½ : Then 1 four-hourly for 3 days. Then gradually reducing doses for further 6 days.	
Adult 15–40 years .	2 : 2 Then 1 four-hourly for 3 days: then 1 eight-hourly for 6 days.	
Over 40 „ . .	Slightly smaller doses. If vomiting, give sodium salt intramusc. or well diluted with N-saline intravenously, 1 G. four-hourly, till vomiting ceases, then continue with oral doses.	
Gonorrhœa.	*Sulphathiazole* or *Sulphadiazine.*	Penicillin therapy is more effective.
Acute or chronic .	1 four-hourly for 5 days.	
Staphylococcal pyæmia.	*Sulphathiazole.* 4 four-hourly for 24 hours: then 3 four-hourly.	Combine with penicillin therapy and blood transfusion.
B. Coli in urine.	*Sulphamezathine* or *Sulphacetamide.* 1 six-hourly for 5 days.	Combine with alkali.
B. Proteus and Strept. fæcalis in urine.	*Sulphathiazole.* 1 six-hourly for 5 days.	Combine with alkali if urine is not already alkaline.
B. Dysenteriæ (Flexner or Shiga).	*Sulphaguanidine, Sulphasuccidine* or *Phthalylsulphathiazole.* 6 : 3 four-hourly: later 3 t.i.d.	Not effective against Sonne dysentery.
Lymphogranuloma Inguinale.	*Sulphathiazole* or *Sulphadiazine.* ½ four-hourly for 5 days: after interval of 3–5 days, give two further courses of ½ six-hourly for 5 days.	Combine with Fouadin.

TABLE XXIX.—THE EFFECT OF SULPHONAMIDES, PENICILLIN, STREPTOMYCIN, AUREOMYCIN AND CHLOROMYCETIN ON ORGANISMS.

		Remarks.
Sulphanilamide	*Effective against:* B. coli, Cl. welchii, Lymph. inguinale, Strep. hæmolyticus. *Less effective against:* Br. abortus, Prot. vulgaris, N. gonorrhœa, N. meningitidis, Pneumococci.	
Sulphathiazole	*Effective against:* B. anthracis, B. coli, Proteus vulgaris, N. meningitidis, Pneumococci, Staph. aureus, Strep. hæmolyticus. *Less effective against:* N. gonorrhœa, Strep. fæcalis.	Readily excreted. Well tolerated by most. Liable to produce anuria and hæmaturia.
Sulphadiazine and derivs., viz., Sulphamerazine and Sulphamezathine (Syn. Sulphamethazine)	*Effective against:* B. anthracis, B. coli, B. dysenteria (Flexner and Shiga), B. friedlander, Cl. welchii, Lymph. inguinale, N. meningitidis, Pneumococci, Staph. aureus, Strep. hæmolyticus. *Less effective against:* N. gonorrhœa, Proteus vulgaris.	Very well tolerated, and slowly excreted. Toxic effects on liver and kidneys (especially sulphadiazine).
Sulphaguanidine, Sulphasuccidine (Syn. Succinylsulphathiazole), and Phthalyl-sulphathiazole	*Effective against:* B. dysenteria (Flexner and Shiga).	Largely insoluble in gut.
Sulphapyridine	Rarely prescribed on account of toxic side-effects.	
Penicillin	*Effective against:* B. anthracis, Cl. œdematiens, Cl. welchii, Coryn. diphtheriæ, Leptosp. icterohæmorrhagica, M. catarrhalis, N. gonorrhœa, N. meningitidis, Pneumococci, Spirillum minus, Staph. aureus, Strep. hæmolyticus, Trep. pallidum, Trep. pertenue (yaws), Vincent's organisms. *Less effective against:* Actinomyces, B. proteus and Strep. fæcalis (in urine), Strep. viridans.	
Streptomycin	*Effective against:* A. ærogenes, B. coli, B. dysenteriæ, B. friedlander, B. proteus and B. pyocyaneus, Bruc. tularense, H. influenzæ, Salmonella, Streptococci, Staphylococci. *Less effective against:* B. typhosus, M. tuberculosis.	
Aureomycin	*Effective against:* B. coli, Pneumococci, Staphylococci, Streptococci, Strep. fæcalis, Rickettsia organisms, virus of Lymphogranuloma Inguinale and of Primary Atypical Pneumonia. *Less effective against:* Brucella infections.	Administered orally: passes freely into C.S.F.
Chloromycetin	*Effective against:* B. coli, B. proteus, Ps. pyocyanea, Rickettsia organisms, virus of Primary Atypical Pneumonia. *Less effective against:* Brucella infections, B. typhosus and B. paratyphosus.	Administered orally.

TABLE XXX.—COMMON USES AND AVERAGE DOSES OF PENICILLIN—AFTER FLEMING. (Doses in Oxford Units by deep subcutaneous injections unless otherwise stated.)

Actinomycosis. 60,000 3-hourly for 21 days. Also local injections into abscesses of 1,000–6,000 8-hourly via capillary tubes.
Anthrax. 15,000 3-hourly for 5–6 days.
Arthritis, pyogenic. 20,000 3-hourly: plus 30,000–50,000 in 3 c.c. into joint on alternate days.
Bacterial Endocarditis. 120,000 3-hourly for 42 days.
Boils. 15,000–20,000 3-hourly for 5–6 days, with local cream (B.P.) to prevent spreading.
Bronchiectasis. 100,000 in 3 c.c. b.d. with special inhaler.
Bronchitis, acute. 50,000–100,000 in 3 c.c. b.d. or t.i.d. with special inhaler: or as for pneumonia.
Children. 1,500–2,000 per lb. body weight per day, by 4-hourly injection (neonatals 6 hourly).
Cystitis and pyelitis. 15,000 3-hourly gives high urine concentration and often effective against Strep. fæcalis and B. proteus.
Diphtheria and carriers. 20,000–30,000 3-hourly.
Empyema, acute. 120,000 in 10 c.c. into cavity daily, or 240,000 in 10 c.c. into cavity each 2 days: gives adequate blood concentration also.
If for local effect only, 20,000–40,000 in 20–40 c.c. into cavity daily.
Gonorrhœa, acute. 30,000 3-hourly for 5 doses or 100,000 and repeat once in 8 hours.
Ludwig's Angina. 40,000 3-hourly for 3 days.
Meningitis, acute. 30,000 3-hourly for 5–7 days. Also 10,000 each 24 hours intrathecally. Also sulphonamides.
Ophthalmia. Drops of 2,500 per c.c. plus ointment 400–800 per G.
Osteomyelitis, acute. 60,000 3-hourly for 12–21 days.
Peritonitis, acute. 30,000 3-hourly—diffuses freely from blood.
Pneumonia. 15,000–30,000 3-hourly for 5–7 days.
Rat-bite fever. 20,000–30,000 3-hourly for 7–10 days.
Sinusitis, early. 1,000 per c.c. with ½ per cent. ephedrine in N. saline as spray.
Late: wash out with 1,000 per c.c.
Syphilis, early. 40,000 3-hourly for 60 doses, with bismuth and arsenicals.
Late: 6–10 doses bismuth each 4 days with Pot. iod.; then as for early disease; repeat penicillin after 3 weeks.
Vincent's Angina. 15,000–20,000 3-hourly with penicillin lozenges.
Weil's disease. 40,000 3-hourly for 4 days.

excreted than penicillin, it is injected each 6–8 hours: its present state of impurity makes reactions common, and organisms rather rapidly develop resistance to its use. *Chloromycetin* (chloramphenicol) was originally obtained from *Streptomyces venezuelæ*, and is now prepared synthetically: its chief value is against all forms of typhus, the enteric fevers and virus pneumonia. *Aureomycin*, obtained from the mould *Streptomyces aureofaciens*, is effective against a wide range of organisms, including a number of viruses. These last two antibiotics are usually given orally. (3) The patient's general resistance is encouraged by a free supply of fresh air, at least 5 pints of fluid daily, with sugar as the principal food, restful sleep and especially in anæmic cases by blood transfusion.

§ 516. **Subacute and Chronic Septic Conditions** (*e.g.*, **Abscess, Ulceration etc.**) **also give rise to intermitting pyrexia.** The various clinical conditions met under this heading are due to the absorption of some septic or toxic material into the circulation. The possible sources of the sepsis are numerous, and may be grouped into two divisions—(*a*) ABSCESS

and (*b*) Simple Inflammation often with Ulceration (internal or external). Clinically, the former is more acute than the latter, and indeed, the former might be called subacute, the latter chronic, septicæmia.

(*a*) Abscess (Pent-up Pus).—Pus never forms in any part of the body—*e.g.*, in the pleura (empyema), in the liver (hepatic abscess), or elsewhere—without an intermitting or remitting pyrexia: this may be accompanied by "chills," "shivers" or "rigors." Before the clinical thermometer was invented, these shiverings (sometimes followed by sweatings) were the chief symptoms by which the formation of pus was identified. It must however be remembered that chemotherapy, and especially the use of the sulpha-drugs, may allow pus to develop—sometimes in considerable amounts—while the patient remains apyrexial: it is thus easy to mask the presence of a dangerous infection in a hidden area, *e.g.*, in the mastoid air-cells, or in a subphrenic abscess. In the presence

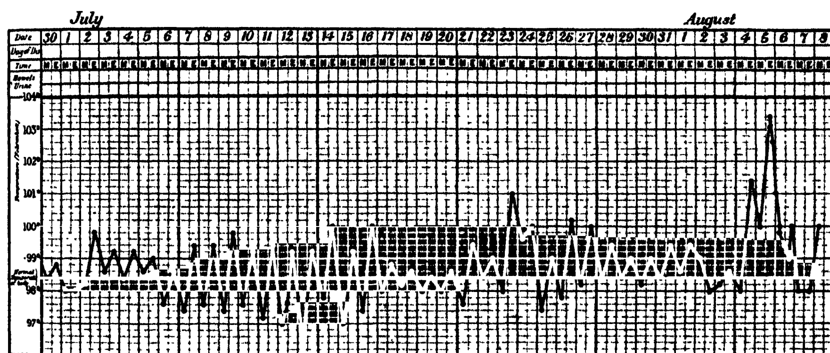


Fig. 124.—Chronic Pyæmia.—Frank T——, æt. thirty-one, had had an attack of gonorrhœal rheumatism two years before, from which he had recovered. The present illness had come on quite gradually a month or so before admission. Stiffness and pain in the joints being the chief symptoms, and the urethra being *absolutely normal*, it was regarded as a case of chronic rheumatism, though none of the usual remedies had any effect. The joints became progressively worse, and though he complained of abdominal pain from time to time attention was not directed to that cavity. He died some two months later suddenly from perforation of the appendix. A review of the case pointed to a chronic septic process having its origin in the appendix, and especially affecting joints which had been previously diseased.

of an abscess, there are marked constitutional effects such as lassitude, debility, pallor (though with a hectic flush on the cheeks), and loss of weight. The blood should always be examined, and the presence of leucocytosis with an increase in the polymorphonuclear cells will afford strong confirmation that pus is present.

Etiology.—Abscess or pent-up pus in any position may produce these symptoms, and careful search should be made for abscess of the liver, gall bladder, pelvic cellulitis or abscess, appendicitis (Figs. 123, 124), caries of the spine, mastoiditis, sinusitis, a dental apical abscess, intracranial abscess, empyema, pyonephrosis, subphrenic and perinephric abscess, etc. Pain is the chief localising symptom, but it may be absent, especially in children. On giving free exit to the pus the pyrexia should rapidly subside.

(*b*) Simple Inflammation, with or without Ulceration of an Internal

or EXTERNAL surface, is always attended by some degree of intermitting pyrexia, running a more chronic course than the foregoing. This fever also differs from the last in the usual absence of definite rigors. Sometimes the shivering may not amount to more than "chills down the spine"—perhaps thought to be malaria—and sweating which is hardly noticed. The morning temperature is normal, or almost normal, and it is raised one or two degrees some time during the day. Anæmia and failing health are always present, and some degree of leucocytosis is usual. This kind of fever, due to prolonged suppuration and attended by chronic wasting, was formerly known as *Hectic Fever* (Greek, ἑκτικός "habitual"). When due to a discharging sinus—for instance, connected with caries, or necrosis of a bone, or a bed-sore—the cause is obvious. But the condition may also be set up by inflammation or ulceration of any of the mucous membranes or internal passages—*e.g.*, ulcerative colitis, appendicitis (Fig. 124). It is called *Urinary Fever* when it arises from chronic infection of some part of the urinary passages—*e.g.*, with a stone impacted in the ureter, or a urethral stricture, or chronic pyelitis. This cause may be suspected if there be a history of renal colic. Similarly, *Acute Cholangitis* (infection of the biliary passages) may be suspected if there be a history of biliary colic. When the infection, due to gall-stones, is situated in the *gall-bladder*, colic and jaundice may be absent, and the patient complains only of the "chills."

§ 517. The **rarer causes of Intermitting Pyrexia** are fully described elsewhere.

Amœbiasis of the liver (§ 336) or amœbic hepatitis may for months show few signs other than bouts of irregular fever interspersed with periods of apyrexia, and no symptoms other than those of dyspepsia, flatulence, constipation or irregularity of the bowels. Occasionally there is a history of right shoulder pain. There may have been no dysentery, and examination of the stools and of fæcal material collected on sigmoidoscopy may fail to reveal cysts or vegetative forms of *Entamœba histolytica.* The liver signs may be indefinite, but generally the organ is demonstrably enlarged and tenderness is elicited below the costal margin on deep inspiration. When there are associated physical signs such as evidence of consolidation or fluid at the base of the right lung, or when X-ray reveals upward bulging or "splinting" of the diaphragm, an abscess of the liver is almost certainly present. There is a mild polymorphonuclear neutrophil leucocytosis, but even when the condition of hepatitis has gone on to abscess formation a neutrophilia not exceeding 80 per cent. and a total count not exceeding 15,000 per cu.mm. are common findings. Amœbiasis should be suspected in obscure cases of fever when the patient has lived in the tropics. Within 48–72 hours, the temperature responds to emetine. Such patients show marked improvement in health when the parasites are eradicated. It is among the amœbic carriers that many cases of hepatitis and liver abscess are found, as infection often persists unsuspected for long periods.

INFLUENZA, TYPHOID, and PARATYPHOID fever, especially when modified by previous inoculation, and other diseases described in Groups I and II may be attended by pyrexia of an intermitting type.

KALA-AZAR has usually intermittent fever after the first period, during which there is fever of a remittent type (§ 505).

TYPHOID FEVER during the first two weeks of its course is attended by typically continued pyrexia, but in the concluding stage of the disease the temperature gradually drops each morning to normal, and the case may be seen for the first time in this stage. Under certain other circumstances also the temperature may be intermitting —viz. : (i.) In rare instances it may commence with symptoms of ague (see *Varieties*, p. 601) ; (ii.) in very mild cases ; (iii.) after lasting a few days, the fever sometimes aborts and takes on an intermitting type.

Various LOCAL INFLAMMATORY DISEASES, other than the septic conditions previously mentioned, may at times be attended by intermittent pyrexia. In cirrhosis of the liver, for instance, a prolonged fever with daily oscillations is occasionally observed.

MALIGNANT ENDOCARDITIS (Multiple Systemic Embolism) (§ 50) is always attended by pyrexia of an irregularly intermitting type, sometimes with sweatings and rigors, very much resembling the chart of septicæmia, though the temperature is usually a little more diurnally regular, and rigors are not usually so frequent (compare charts, Figs. 122 and 124). The diagnosis of these two diseases is sometimes very difficult. Malignant Endocarditis is favoured by (i.) a loud cardiac murmur detected quite

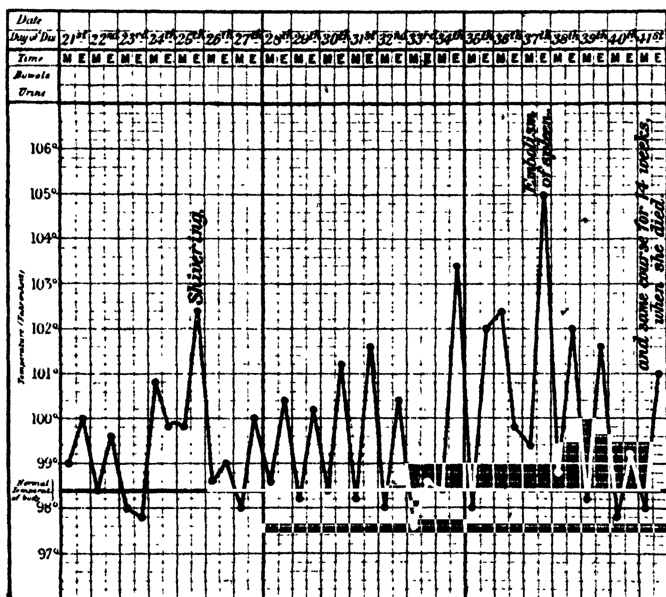


FIG. 125.—MALIGNANT OR ULCERATIVE ENDOCARDITIS in a woman, æt. forty-two. The three weeks shown illustrate the course of the temperature over a period of seventeen weeks, when she died.

early in the case ; (ii.) a history of acute rheumatism ; (iii.) the secondary emboli in this disease are more frequently found in the systemic arteries, such as those of the spleen, liver, and kidneys, and they do not result in abscesses. In PYÆMIA the emboli occur primarily in the arteries of the lungs, and from the very beginning they suppurate and form abscesses, which constitute centres of secondary infection elsewhere.

HODGKIN'S DISEASE is recognised by the enlargement of the lymphatic glands and pyrexia of a remitting or intermitting type. Sometimes a periodic temperature occurs with glandular enlargement confined to the mediastinal or retroperitoneal glands (Pel-Ebstein Syndrome).

In PERNICIOUS ANÆMIA the temperature is sometimes subnormal, but it is more frequently attended by exacerbations of fever of an intermitting type. The disease is also identified by the intense sallowness of the skin and the condition of the blood.

In ACUTE LYMPHATIC LEUKÆMIA the temperature is high and irregular, somewhat resembling that of septicæmia. It can be diagnosed by the examination of the blood, when there is found to be an increase in lymphocytes (§ 543).

The OPIUM OR MORPHIA HABIT (§ 900) is attended from time to time by attacks of intermittent pyrexia, during the reaction stage, in which there are cold, hot, and sweating stages. Cases are recorded where no cause could be found, but the attack ceased on giving opium.

MALIGNANT DISEASE, especially when involving the liver, is often accompanied by intermittent fever.

§ 518. **African Trypanosomiasis** (Syn.: Sleeping Sickness), a disease confined to tropical Africa, is characterised by enlargement of the glands, fleeting intermittent erythematous rashes, irregular pyrexia, excessive sleepiness and mental degeneration.

Symptoms.—The incubation period following the bite of infected tsetse flies varies from one to three weeks. Two stages are recognised: (1) In the *first* trypanosomes are demonstrable in the blood and lymphatic gland juice. Irregular, remittent or intermittent fever with periods of apyrexia may last for some months. The pulse is rapid, the respirations accelerated. Patches of circinate erythema appear, mainly on the trunk, and localised puffiness may implicate the feet, legs and face. Polyadenitis with painless enlargement of the posterior cervical glands is common; sometimes the epitrochlear, supraclavicular and axillary or femoral glands may be the group affected. The spleen is generally palpable at this stage and deep hyperæsthesia, especially over bones like the tibia, is characteristic; there is often a definite latent period after pressure before pain appears. This stage is well marked in Europeans, but may not be so evident in natives. Many months may elapse before the second stage, due to trypanosomes invading the central nervous system, occurs. (2) Here the cerebro-spinal fluid contains an excess of lymphocytes and globulin: trypanosomes are sometimes difficult to find. The patient first develops lack of concentration, headache, insomnia, loss of weight and slight tremor of the tongue associated with polyadenitis. Later, the countenance becomes apathetic and morose, emotional instability and laziness increase, and the patient drops off to sleep even when eating. The gait is shuffling, the speech mumbled and slow, and fibrillary tremors develop in the lips and hands. Ataxia is marked and the reflexes increased. Finally, owing to muscular weakness, the patient takes to bed; bed-sores and flexure contractures develop and coma or convulsions terminate the picture.

Ætiology.—The trypanosome is introduced into the body by the bite of a tsetse fly. There are two varieties of trypanosomiasis: (i.) a chronic form, lasting months to years, due to *Trypanosoma gambiense* and occurring more in the western half of tropical Africa. This is transmitted by *Glossina palpalis* and allied species; these tsetse flies require shade and proximity to water. (ii.) An acute form, lasting weeks or months, due to *T. rhodesiense*, occurs especially in Rhodesia, Tanganyika and Nyasaland. It is carried by *G. morsitans* and related species which breed in more open orchard bush.

Diagnosis.—This can only be made with certainty by finding the parasite by gland puncture, in the blood or in the cerebro-spinal fluid. White rats should be inoculated with blood in doubtful cases.

Prognosis.—With treatment many patients infected with *T. gambiense* and, in the early stages, with *T. rhodesiense* recover, but in the later stages of rhodesiense infections the outlook is grave.

Treatment.—Early infections of either type respond to suramin (G. 1 weekly)

intravenously for 5 to 10 doses: albuminuria and nephritis may follow its administration. The drug, a cyclical urea compound, does not penetrate to the central nervous system and consequently is ineffective in advanced infections. It is therefore usually combined with tryparsamide G. 2–4 weekly intravenously for 10 to 12 doses. This drug, a pentavalent arsenical preparation, enters the nervous system and must always be used in late cases. Unfortunately *T. rhodesiense* tends to be resistant to arsenical drugs and consequently neither drug influences the late stages of this infection. Tryparsamide may produce optic atrophy. Trypanosomes readily become resistant to arsenic; and various diamidine compounds, such as stilbamidine, pentamidine or propamidine (G. 0·1 to 0·3 intravenously daily for 7 to 14 doses) may be tried. These drugs may produce toxic effects and they often fail to clear parasites from the central nervous system. Propamidine is said to produce abortion in pregnant women. *Preventive measures* include travelling at night to avoid tsetse bites, the wearing of veils, the destruction of breeding-places and the trapping of flies. A single injection of suramin, or of pentamidine, is said to protect against infection for several months.

Chaga's disease in S. America is also due to a trypanosome. **Trichinosis** (§ 593) is usually accompanied by intermittent fever. **Rat-bite fever** may show a temperature of an intermittent type (§ 505).

THE GENERAL TREATMENT OF MICROBIC DISORDERS

Remedial treatment has, for the most part, been given under each disease, but there are some important matters relating to all infections in common which must now be referred to—viz., **Immunisation, Serum Therapeutics, Chemotherapy, Notification** and **Isolation, Disinfection, Diet,** and the treatment of **Pyrexia** and **Hyperpyrexia.** In the first three of these we find ourselves on the threshold of discoveries which are revolutionising the methods of prevention and treatment of infective disorders.

§ 519. **Immunity** means the ability of the body to defend itself against injury by pathogenic micro-organisms or by their toxic products. Immunity is, as a rule, only relative, and its effectiveness depends upon the balance between the virulence of the invading organism and the resistance of the body.

The virulence of bacteria, in general, depends upon their invasive power and their ability to withstand and overcome the defensive forces of the host, and also upon the production of toxic substances. The latter, in addition to having a damaging effect on the tissues of the host, may also paralyse the protective mechanism by inhibiting the production of phagocytes or by destroying circulating leucocytes. The toxic products of bacteria comprise:

1. *Exotoxins* are soluble substances elaborated by the living organisms and set free in the body fluids or in culture media. They can be obtained from the filtrate of cultures in fluid media after the bacteria have been removed by a Seitz or porcelain filter. Exotoxins are relatively unstable and the majority are destroyed by exposure to sunlight or by boiling for a few minutes. They rapidly deteriorate on storage and may be converted into less toxic substances known as *toxoids* by the action of certain chemicals. Inoculation of a suitable animal with an exotoxin stimulates the production of its particular *antitoxin*; toxoid is equally efficient for

this purpose and is preferable since it is much less poisonous. The organisms of diphtheria, tetanus, botulism and gas gangrene develop powerful exotoxins which in the case of the first three possess a great affinity for nervous tissue. Some strains of streptococci and staphylococci are also capable of producing exotoxins from which it has been possible to separate various components—*e.g.*, a fibrinolysin, a coagulase, an erythrogenic factor, and a spreading factor (hyaluronidase).

2. *Endotoxins* are intracellular toxins which are retained within the bodies of the bacteria until they die and disintegrate. In contrast to exotoxins they are not present in filtrates and are relatively stable, showing resistance to heat and deteriorating but slowly on storage. They cannot be converted into toxoids. Their injection into animals stimulates the production of antibacterial sera which have little or no antitoxic neutralising action. Meningococci, cholera and typhoid bacilli are examples of organisms which form an endotoxin.

3. *Lysins* are enzyme-like substances which digest tissue cells. A lysin may be designated according to the organism which forms it—*e.g.*, staphylolysin, or the cells it destroys—*e.g.*, hæmolysin.

4. *Aggressins* are toxic bodies produced by many pathogenic bacteria, especially when growing in the tissues. They are said to interfere with phagocytosis. Various bacterial products may, however, possess this property and the existence of a specialised substance of this type is uncertain.

5. *Ptomaines* are not substances secreted by bacteria but are toxic bodies resulting from the bacterial decomposition of proteins. They are closely allied to the vegetable alkaloids and are not destroyed by heat. The chief examples are cadaverin and putrescin.

Immunity may be either natural or acquired. NATURAL IMMUNITY is shown by the high resistance of some races to certain infectious diseases, and by the insusceptibility of certain animals to some human infections. Thus hens are resistant to tetanus toxin. But even in this case the immunity is only relative; given a sufficient dose of tetanus toxin, even a hen will succumb. So also sheep, which live an open-air life, are as a rule immune to the tubercle bacillus; but when they are kept penned in, in insanitary conditions, they may become infected. On the other hand, a natural immunity may often (though not always) be enhanced by suitable treatment.

ACQUIRED IMMUNITY is the resistance developed to an organism or its toxin as a result of natural disease or artificial treatment. It is common knowledge that some diseases rarely attack the same person twice, *e.g.*, typhoid, scarlet fever and small-pox. Here again the immunity is relative and not absolute; second and even third attacks have been described. Artificially acquired immunity is the crux of this subject, and is the line of research along which immense progress has been made, and even more is promised. Inoculation of small-pox is probably the earliest example. The serum from a small pustule in a mild case was rubbed into the skin,

and a mild attack of small-pox ensued. Later, Jenner, observing the immunity to small-pox of those already infected with cow-pox, succeeded in immunising people against small-pox by artificial vaccination with cow-pox (§ 480). Whether cow-pox is an allied disease, or a modified form of smallpox, is still a subject for argument, but quite immaterial from the point of view of immunity. After this discovery little progress was made till Pasteur began his work on hydrophobia and anthrax—the starting-point of our knowledge of this now vast subject.

Artificial immunity is divided into two classes, Active and Passive. In *Active immunity* the resistance is developed by the body as a result of the injection of the toxin, or organism or virus. Vaccination is therefore an example of active immunity. In other conditions it is developed by injecting graduated doses of the poison, beginning with weak and working up to strong doses. The modification of the strength is variously obtained: dead bacteria may be used, or old or overheated toxins, or the bacteria may be grown on unsuitable media, such as one containing disinfectants, or at an unsuitable temperature, or in the absence of oxygen or in excess of it. Minute doses only may be used or the appropriate anti-serum added. Later, larger doses or more toxic strains are employed, and finally it may be necessary to give large doses of living virulent bacteria, or fully potent toxin.

In *Passive Immunity* the serum from an animal which has been actively immunised against the poison in question is injected into the subject, thus effecting an almost immediate increase in the patient's resistance to it.

While the observed facts of immunity are clear and definite, the theoretical explanations of the problems are far less satisfactory. It is most important to remember that the theories are only explanations, and are valueless if they cease to provide a satisfactory basis for further research and speculation. As regards the neutralisation of toxin by antitoxin, three theories at present hold the field; not one is completely satisfactory. Ehrlich's theory is based on the presumption that toxin and antitoxin unite as do strong acid and strong base. However, the discovery of the fact that with age a toxin loses its toxicity, but will still unite with the same quantity of antitoxin, necessitated the introduction of a "toxone," a substance allied to a toxin; from this point there have been continual modifications of the original theory to meet newly discovered facts. The explanations are nearly as complicated as the facts they set out to explain—an obvious disadvantage. Nevertheless, this theory has been the starting-point of the greatest amount of work on the subject. Madsen and Arrhenius have suggested that the combination of toxin and antitoxin is comparable to "mass action" in chemistry, on the analogy of the combination of a weak acid and base; the theory has proved helpful, but does not cover all the possibilities. Lastly, Bordet, forsaking chemistry in its narrower sense, has suggested that the union is of the nature of adsorption, the physical electrical interaction of substances in the colloidal state, and this theory, which makes the least hard and fast rules, seems to be the most fully observed.

To account for the almost unlimited production of antitoxin which may follow the injection of comparatively small doses of toxin Ehrlich offers the following suggestions: Toxin and body cell consist (from the chemical point of view) of a number of complicated organic substances or "side chains," loosely attached to a central nucleus. A toxin can only exert its poisonous properties when one of its own side chains unites with the appropriate side chain of the cell. This union is permanent,

and so puts an end to the vital activities not only of the single side chain but also of the whole cell; when this process takes place extensively, death results. In the case of a non-fatal dose of toxin, the cells ultimately recover; as they recover they regenerate the side chain neutralised by the toxin. The number of side chains regenerated not only replaces those destroyed, but provides an excess; as the cells do not need them they are shed into the general circulation and form the antitoxin. As a result of repeated injections the body not only produces a great quantity of antitoxin, but develops the power of developing even more, and that without the stimulus of a further injection of toxin. An immunised animal may be bled repeatedly, till the total volume of blood lost is in excess of its total blood volume and yet the quantity of antitoxin in the new formed blood (*i.e.*, its antitoxin titre) will equal if not exceed that of the blood before the depletion.

The modern view of immunity is a compromise of the theories of Ehrlich, Madsen and Arrhenius, and Bordet, and it is now considered that both chemical and physical reactions involving molecular structure and colloidal phenomena are concerned in the process of antibody production and in the union of an antibody and the antigen which stimulates its formation. Chemical research has demonstrated that antibodies are serum globulins ("immune globulins") possessing specific chemical groupings that permit them to unite with their corresponding antigens.

The practical application of our knowledge of immunity in the treatment of disease must now be briefly considered.

§ 520. **Immune Therapy.**—This may be undertaken for prophylaxis or treatment. 1. Examples of *prophylaxis* in *passive immunity* are the use of diphtheria antitoxin, of tetanus antitoxin, and of convalescent measles serum for temporary protection of individuals exposed to these infections. *Prophylactic active immunity* may be produced by the use of vaccines as for typhoid and paratyphoid fevers, cholera, plague, staphylococcal and catarrhal infections; by a mixture of toxoid and antitoxin or by toxoid alone against diphtheria; by toxin against scarlet fever; and by the use of a related virus for smallpox or a modified one for hydrophobia.

2. *Therapeutic immunisation* carried out in the presence of the disease depends mainly on the administration of the appropriate antitoxic or antibactericidal serum. Such sera have proved of value in diphtheria, tetanus, botulism, meningococcal and pneumococcal infections.

It must be emphasised here that the success which has attended *chemotherapy* with penicillin, the sulphonamides and allied products has caused this treatment to displace the use of antisera in many infections (streptococcal, pneumococcal and meningococcal).

Vaccine Therapy is based on the principle of producing in the patient a condition of active artificial immunity, and various methods are employed, all leading to this end. The most usual is the injection of suspensions of the dead bodies of the infecting micro-organisms. Almroth Wright and Douglas, while studying the properties of phagocytosis by the leucocytes, originally described by Metchnikoff, found that the proportion of organisms ingested by the polymorphonuclear leucocytes was increased in tests where the blood of persons who had received injections of the organism in question was employed. Further investigation showed that this property of phagocytic increase was situated in the serum, for it did not occur in tests with washed leucocytes. On the other hand, it was found in tests with washed leucocytes from a normal person under treatment with the serum of an inoculated individual. The substance which was the cause of this phenomenon they called *opsonin*. The actual immunity, however, depends on many substances of which opsonin may be taken as a type: agglutinins, precipitins, bacteriolysins and bactericidal bodies.

In its essential constitution every vaccine is a suspension in some fluid of the various organisms employed, but the infinite variety of methods used in making this suspension render it impossible to describe every type. From the infecting focus a culture is made, and from this culture the various types of organism are picked off and examined. Pure cultures of each pathogenic type are prepared, then washed off in weak phenol-saline and a homogeneous suspension made. This is then standardised either macroscopically, by comparing its opacity against that of a standard suspension ; or microscopically, by comparing, in a stained film made from equal quantities of the suspension and of blood, the number of organisms and of red blood corpuscles. *Sensitised vaccines :* before standardisation the suspension of organisms is treated with the serum of an animal, or a person immunised against the organism in question. *Detoxicated vaccines* are made by dissolving or otherwise destroying the cell wall of the organism. The doses of most vaccines are calculated in millions of organisms per cubic centimetre, and may be put up in separate ampoules, or kept in bulk in rubber-capped bottles. A word of warning is needed. A vaccine consists of a suspension of protein in a very weak antiseptic, and is therefore a fair culture medium. Great care is necessary not to infect it before or during use. When supplied in ampoules it is unsafe to attempt to seal off a partly used ampoule for use next time.

Vaccines are usually administered subcutaneously over the deltoid, and the usual antiseptic precautions observed. When there is a marked local reaction in the arm, other sites such as the buttock, thigh or abdominal wall are preferable. It is usual to give vaccines in progressively increasing doses, with an interval of a week between the smaller doses, and 10–14 days between the larger ones. The beneficial results depend on the relationship between dose and interval, for the immunisation has to be developed by the patient. Vaccine therapy falls into undeserved disrepute when progressively increasing doses are given without reference to the reaction of the patient. It is a safe rule to increase at each successive injection unless there is a reaction. A *local* reaction consists of swelling, redness or soreness at the site of injection. A *general* reaction varies from slight fatigue and sleepiness to fever and malaise lasting for days, even weeks if the dose has been too large. A *focal* reaction is a lighting up of the infective focus. When the patient feels better after a dose, increase at first by half and later double the preceding dose. In delicate or allergic persons the initial dose should be very small. Never give a dose near a menstrual period, nor until a general reaction has passed off. The "negative phase" or phase of lowered resistance described by Wright and reputed to last from a few hours to a day or two after administration of a vaccine, is not now thought to be as significant as originally considered.

Prophylactic oral vaccination is extensively used on the Continent, especially against typhoid, dysentery and cholera, on the hypothesis that it is advantageous to render the local tissue (in this case, the intestinal mucosa) immune. Although agglutinins appear in the blood as a result of administration of a vaccine by this route they are of lower strength compared with subcutaneous or intramuscular inoculation.

§ 521. Serum Therapy.—In certain diseases, or when it is desired to develop immunity in the shortest possible time, it is possible to confer passive immunity on the patient by injecting an antitoxic or antibacterial serum. Those diseases in which the symptoms are chiefly due to exotoxins—*e.g.*, diphtheria, tetanus, gas gangrene, botulism, require treatment with a serum rich in antitoxins. When the symptoms are chiefly due to endotoxins, give a serum rich in antibacterial substances, although with the exception of Sclavo's serum for anthrax this type of serum is not usually so effective. Antitoxic and antibacterial sera are derived from animals immunised respectively to the particular toxin or organism in question: occasionally an antibacterial serum (or antiviral serum in the case of a virus infection) is obtained from a convalescent patient. In some cases the doses are calculated in arbitrary standard units ; in others, in terms of volume. The bulk of serum employed may be considerable.

Serum may be given by four routes : (1) Intravenous. This is the method of

choice when immediate immunisation is required, for the antitoxin is thus instantly available. It is also the best way when doses have to be large, or frequently repeated, as in dysentery or pneumonia, or when its use has been unduly delayed. On the other hand, the immunity is less lasting, and there are certain dangers which will be mentioned later. (2) Intramuscular. The gluteus maximus and the vastus externus are the most convenient muscles. This method is less rapid in its effects, the maximum concentration of antitoxin not being present till twenty-four hours after the injection. It is much safer, and requires less elaborate technique. (3) The subcutaneous method is the slowest and the easiest. The maximum concentration is not reached till seventy-two hours after injection. The injection may be made in the buttocks, abdominal wall or thighs. This route may be of great value as a reserve in support of the intravenous, the antitoxin being maintained in the circulation by slow absorption. (4) Intrathecal. In the treatment of tetanus this route may be of value. A lumbar puncture is performed and cerebro-spinal fluid is drawn off in excess of the quantity of serum which is afterwards injected. A Record or all-glass syringe (autoclaved or dry-sterilised) are essential.

Hypersensitivity to serum is due to the proteins of the serum and not to the antitoxins. It may manifest itself in two chief ways:

(1) *Immediate reaction.* This develops within a few minutes to half an hour of the injection and is particularly prone to occur in asthmatic and other allergic individuals, especially with intravenous injections. *Symptoms* vary from mild urticaria to those of acute anaphylactic shock with respiratory embarrassment and cyanosis ending in sudden death. Rarer symptoms are vomiting, abdominal pain and diarrhœa. To eliminate or minimise the degree of this immediate reaction it is important to determine before any therapeutic foreign serum is administered whether or not the individual is sensitive to such serum—especially when there is a history of previous serum administration or if the person has had asthma or any form of protein hypersensitiveness. Hypersensitivity is tested for by the intracutaneous inoculation of 0·1 c.c. of the serum. If the person is sensitive an urticarial wheal develops at the site of inoculation within half an hour. Then preliminary desensitisation must be carried out before giving the major part of the dose. This is done by beginning with a small subcutaneous dose of 0·1 c.c. diluted with saline solution and doubling the amount every half an hour until 1·5 c.c. have been given. If no reactions have occurred it is then usually safe to give the balance of the dose. *Treatment* of anaphylactic shock is by the injection of 1 c.c. of adrenalin B.P. intramuscularly. The action of the adrenalin is transitory and anaphylactic symptoms may recur after the immediate effects have worn off: in severe reactions it is necessary to repeat the dose. Artificial respiration may be performed and oxygen administered. If adrenalin be not available, atropin or pituitary extract may be given.

(2) *Delayed reaction or serum sickness* usually comes on from the 7th–12th day after an injection of foreign serum and may also occur in persons who have had an immediate reaction. There is usually an erythematous rash or urticaria starting first at the site of inoculation and later becoming generalised. Fever, joint pains, general glandular enlargement and malaise are common. These symptoms occur in about 10 per cent. of the cases when less than 10 c.c. are given and in about 90 per cent. when more than 50 c.c. are used. They are less frequent when concentrated sera are injected. A subsequent injection of the same protein causes the same symptoms, usually within twenty-four hours. *Treatment* is by the subcutaneous injection of adrenalin, and repeating this when necessary. Auto-hæmotherapy often helps (and see § 656).

ALLERGY is defined as a natural inherited condition of hypersensitiveness (cp. § 609). Serum sickness is one manifestation of allergy; it probably explains a number of other conditions. Of these the best known, and the most common, is Hay Fever, the result of hypersensitiveness to the pollen of Timothy and other grasses. Under this heading also comes the hypersensitiveness shown by certain people towards various foods, such as strawberries, eggs, shell-fish and the flour of various grains; to the dandruff of

some animals, as the horse and the cat; to dust, especially house dust, and to certain plants and flowers. Various symptoms are produced; food-stuffs as a rule cause urticarial rashes; animals and flowers, asthma. Complete sets of common proteins are available for testing by intradermal injection or scarifying the skin and applying the substance to be tested to the scratch. In from 10–20 minutes a reaction appears which varies in its intensity with the sensitiveness of the patient. A sensitive patient may react to many substances at the same or at different times. Treatment consists in preventing exposure of the patient to the protein to which sensitivity is proved, or, if this is impossible, in desensitization by a course of inoculations, commencing with minute doses of the substance responsible. Benadryl or antistin administered by mouth are of proved value in the prevention and treatment of allergic states by their antihistamine action.

Special Methods for each Disease.

Diphtheria.—An antitoxic serum has been on the market since 1895. When given early enough and in large enough doses, antitoxin has been found to be of the greatest value for patients suffering from the disease (see further details in § 494).

Treatment.—The remedy should be used as early as possible in the disease. In the mildest cases a single injection of 4000–8000 units should be given; in moderate cases two injections of from 16,000–48,000 units may be required; in severe cases 64,000–100,000 units, repeated on two or more days in succession. The smaller doses for mild cases can be given intramuscularly: the larger doses for cases of moderate and severe type must always be given intravenously: with modern refined sera, anaphylactic reactions are unusual. The earlier the antitoxin is given, the more favourable the prognosis. In suspicious cases, where there is delay before obtaining a bacteriological report, inject the antitoxin without waiting for the report. Children tolerate antitoxin well, and should receive the same doses as adults. *Effects.*—In the course of twenty-four hours there should be an improvement in the patient's symptoms: the membrane ceases to extend, or perhaps begins to loosen, the swelling abates, and the rhinorrhœa is diminished. Danger: serum sickness.

Prophylaxis.—By means of the Schick test we can find out who is susceptible to diphtheria and who immune. Technique: By means of a very fine needle, 1/50 of a M.L.D.[1] of diphtheria toxin in 0·2 c.cm. of fluid is injected into the skin of the flexor aspect of one forearm, while into the skin of the other forearm, as a control, is injected a similar quantity of heated (inactivated) toxin. The results of the reaction come under four heads: 1. *Negative:* complete absence of any reaction in either arm indicates that the patient is immune to diphtheria. 2. *Positive:* complete absence of reaction in the control arm. The test arm shows after 24–36 hours a red circumscribed flush which reaches its maximum on the fourth day, when it may be 1 to 2 cm. in diameter. After this it fades till by the seventh day a brown desquamating stain is left, which may remain for some weeks. Interpretation: the patient is susceptible to diphtheria. 3. *Negative and Pseudo:* by the end of twenty-four hours a diffuse red flush which is equal in both arms has developed. By the fourth day this will have faded to a brownish stain, equal in both arms. Interpretation: the patient is immune. The reaction is the non-specific result of a foreign protein. 4. *Positive and Pseudo:* at twenty-four hours the reaction is as in 3, a diffuse red flush equal on both arms, but by the fourth day the flush on the control arm has faded, while that on the test arm has developed to the circumscribed red area seen in 2, which in its turn fades by the seventh day. Interpretation: the patient is susceptible. If possible the patient should be seen daily till the seventh day; if only a single visit is possible, it should be on the fourth day.

The Schick test allows us to separate the immune from the non-immune. It now remains to immunise the non-immune. Two methods are available: 1. In cases, particularly children, who have been exposed to diphtheria and who have not been

[1] M.L.D. is the minimum lethal dose of toxin for a guinea-pig weighing 240–260 G.

previously actively immunised it is wise to confer a *passive immunity* by injecting 500–1,000 units of diphtheria antitoxin subcutaneously. This will afford protection for three or four weeks, but it may not be successful if the child is already incubating the disease. Antitoxin prevents the toxic results of infection but cannot eliminate risk of the latter. 2. *Active immunisation* is best carried out by Toxoid-Antitoxin "Floccules" (T.A.F.) or by Alum-Precipitated-Toxoid (A.P.T.). T.A.F. is a suspension of the precipitate of floccules formed when diphtheria toxoid and antitoxin are mixed in appropriate "neutralising" amounts. This preparation possesses very high immunising power with low liability to cause reactions. It is especially suitable for adults. Three doses each of 1 c.c. are given subcutaneously or intramuscularly at intervals of two to four weeks. A.P.T. is a suspension of the washed precipitate produced by adding a small amount of alum to diphtheria toxoid. The precipitate is relatively insoluble and the toxoid is gradually liberated at the site of inoculation. A.P.T. will induce a high immunity even with a single subcutaneous injection of 0·5 c.c. It is preferable, however, to give two injections of 0·3 c.c. and 0·5 c.c. at an interval of four weeks: this is the method of choice in children under eight years. Above this age and in adults it tends to produce a local reaction, and the use of T.A.F. is advised. Active immunisation in children is best carried out between the ages of six months to one year and a Schick test performed eight to twelve weeks after the last injection to confirm a satisfactory result. The immunity should be reinforced by a further single 0·5 c.c. dose of A.P.T. on first going to school, and by 1 c.c. of T.A.F. at the age of fourteen.

TETANUS.—*Passive immunisation* with tetanus antitoxin has proved very effective as a prophylactic measure: 3,000 units should be given subcutaneously or intramuscularly immediately after any deep injury which may be contaminated by soil or dirt, especially if there is laceration of tissue. As the immunity from a single dose lasts only about ten days a second dose should be administered after this period if the wound has not healed. It is wise to repeat the injection before any subsequent operation is performed on the infected area as this may cause acute tetanus. In actively immunised subjects a single prophylactic dose of antitoxin is sufficient.

Active immunisation.—Tetanus toxoid (the toxin rendered atoxic by formalin) is used to produce a high immunity in those persons who may be exposed to tetanus. In certain tropical regions where umbilical tetanus of infants is common the disease has been prevented by active immunisation of mothers during pregnancy. Dosage consists of two injections of 1 c.c. subcutaneously at six to eight weeks' interval. Reactions after injection are insignificant and the active immunity long lasting.

For *treatment* after symptoms have developed a dose of 100,000 to 200,000 units of antitoxin should be given intravenously as early as possible. Intrathecal injections of antitoxin are no longer used. Tetanus bacilli are susceptible to penicillin and in any case of established infection in which surgical excision of the wound is not possible, systemic treatment with this antibiotic in doses of 40,000 units subcutaneously three-hourly for three to four days should be employed to supplement the administration of full doses of antitoxin (and see § 784).

HÆMOLYTIC STREPTOCOCCAL Infections.—Scarlet fever is primarily a toxæmia caused by the toxin of a hæmolytic streptococcus present in the throat. The *Dick test* for susceptibility to scarlet fever is analogous to the Schick reaction for diphtheria, and is carried out with diluted scarlet fever toxin (*e.g.*, 1 in 1,000): 0·2 c.c. is injected intradermally into the skin of one forearm, and into the other is similarly injected as a control a like amount of toxin previously boiled in a water-bath for one hour to render it innocuous. The test is read at the end of twelve and twenty-four hours, being maximum at the latter time. A single reading is best made at twenty-four hours; it should not be delayed beyond this. In a positive reaction a bright red erythematous patch from 2 to 7 cm. in diameter appears in six to twelve hours at the site of injection of the toxin, remains for about twenty-four hours and then fades. There should be no reaction in the control arm. A negative reaction is indicated by a complete absence of erythema in either arm. Pseudo-reactions occur as in the case

of the Schick test and are interpreted as described under the latter. *Active immunisation* with scarlet fever toxin is produced by giving five weekly injections subcutaneously of 500, 2,000, 5,000, 25,000 and 50,000 skin test doses : this usually renders a previously positive Dick reaction either negative or only faintly positive. *Treatment.*—For the usual mild type of scarlet fever seen in Britain in which the symptoms are due to toxæmia, and septic complications dependent on invasion of the bloodstream or tissues by hæmolytic streptococci are absent, the principal treatment should be by concentrated scarlet fever antitoxin. This is obtained by immunising a horse with subcutaneous injections of the sterile filtrate from broth cultures of the causative streptococcus. The dose recommended for an adult is an initial one of 20–50 c.c. (approx. 3,000–12,000 units) intravenously, followed by smaller daily intramuscular injections. Penicillin or sulphonamide therapy is reserved for the treatment of septic complications and for septic scarlet fever. These substances are effective in controlling the septic symptoms in most types of hæmolytic streptococcal infections. The dose is assessed on body weight, but in the case of the sulphonamides children require at least a 50 per cent. larger dose than do adults. For the doses recommended, see Tables XXVIII and XXX.

STREPTOCOCCUS VIRIDANS Infections.—The importance of this organism in acute rheumatic fever, subacute bacterial endocarditis and in chronic rheumatism and rheumatoid arthritis is well established. In *treatment* of the latter two affections active immunisation with a vaccine of the organism does occasionally give good results. An autogenous vaccine derived from a known septic focus is the most promising. No antitoxin is available. This variety of streptococcus is susceptible to long-continued doses of penicillin but almost completely insensitive to sulphonamides. For the treatment of subacute bacterial endocarditis, when it has been proved to be the causative organism, massive systemic doses of penicillin over a long period—*e.g.*, 1 million units daily for six weeks – are necessary (§ 50). The type of lesion produced by the streptococcus viridans tends to shield it from effective concentrations of penicillin.

STAPHYLOCOCCUS AUREUS Infections.—In the *prophylaxis* of chronic or recurrent localised staphylococcus aureus infections a course of a stock, or preferably autogenous vaccine, administered in a quiescent interval may prove beneficial by inducing an active immunity. The promise of staphylococcal toxoid for such purpose has not been fulfilled. The great majority of strains of staphylococci are very susceptible to penicillin and relatively resistant to sulphonamides. Severe staphylococcal infections provide one of the strongest indications for treatment with penicillin. Acute osteomyelitis, cavernous sinus thrombosis, severe carbuncle, meningitis, septicæmia, endocarditis and pneumonia due to staphylococcus aureus from whatever focus indicate immediate penicillin systemic administration. Sulphonamides should be tried in these conditions only if penicillin is not available. Sycosis barbæ and carbuncle are both benefited by local penicillin application.

ANTI-CATARRHAL Vaccine.—Composed of suitable doses of all those organisms which may infect the nose, throat and lungs. These are the Streptococcus hæmolyticus and Streptococcus viridans, the Pneumococcus, the Pneumobacillus, the Influenza bacillus, Micrococcus catarrhalis, and various strains of Diphtheroid bacilli. It is usual to give this vaccine in three increasing doses, at weekly or ten-day intervals. Inoculations should be made at the beginning of September and repeated again in January. The protection bestowed lasts six to twelve months. Smaller doses than those usually recommended should be given to delicate or allergic persons and those subject to "colds"; otherwise a "cold" is precipitated.

TYPHOID AND PARATYPHOID Infection.—*Prophylactic* inoculation with typhoid and paratyphoid organisms is very effective and confers immunity for at least two years. The customary T.A.B. vaccine consists of a suspension of typhoid, paratyphoid A and B bacilli killed by heat and preserved with phenol : two injections of 500 and 1,000 millions respectively are given with a ten-day interval. Demonstration by Felix of the importance of the Vi antigen in immunisation with typhoid and paratyphoid bacilli, and its ready destruction by heat, has led to the use of an alcohol-

killed vaccine in which this antigen is preserved. This alcohol-killed vaccine (Lister Institute), which is tending to displace the heat-killed variety, consists of a suspension of 1,000 million typhoid and 500 million each of paratyphoid A, B and C bacilli per cubic centimetre in 25 per cent. alcohol as a preservative. The dosage for adult males is two subcutaneous injections of 0·25 c.c. and 0·5 c.c. (for adult females 0·2 c.c. and 0·4 c.c.) at three to four weeks' interval. Somewhat smaller doses are advised for subjects who are below average in physical vigour or development. Both general and local reactions are less marked than with the heat-killed phenolised vaccine.

Treatment of typhoid or paratyphoid fever (or the carrier state) by a combination of penicillin and sulphathiazole and by streptomycin has been claimed to give good results, but these await confirmation: chloromycetin is more promising.

TUBERCULOSIS.—Tuberculin has been used both in diagnosis and treatment. It consists of the broken up protoplasm of the bacillus plus its products in artificial culture medium. It was originally prepared from a six-weeks-old culture in glycerol broth, evaporated to one-tenth of its volume, sterilised by heat and filtered (Koch's "Old Tuberculin"). Koch's "New Tuberculin" is derived by grinding the bacilli (obtained from a growth on solid medium) in 50 per cent. glycerol. The *diagnostic application* depends on the fact that the tissues of a person infected with tuberculosis may exhibit hypersensitiveness (allergy) to tuberculin. In man, the *Mantoux test* is commonly employed: it consists in the intradermal injection of 0·2 c.c. of a 1 in 10,000 dilution followed a few days later by a 1 in 1,000 dilution of Old Tuberculin by means of a syringe with a fine needle. Uninoculated concentrated culture fluid may be used as a control. In a positive reaction an area of erythema and swelling appears at the site of inoculation within a few hours and attains a maximum in twenty-four to forty-eight hours. In a child, up to the age of five years, a markedly positive reaction at 1 in 10,000 is strongly suggestive of active tuberculosis. Negative results in adults, particularly if dilutions of 1 in 1,000 and 1 in 100 also give no reaction, exclude active tuberculosis. Tuberculin P.P.D.—purified protein derivative (Parke, Davis & Co.)—is claimed to be free of extraneous protein derived from the culture medium and to give greater accuracy and sensitivity than the ordinary Old Tuberculin. A specially prepared strip of gauze impregnated with tuberculin ("a tuberculin patch") may be applied to the skin, and avoids a needleprick in children. *Prophylaxis.*—B.C.G. (Bacille Calmette—Guérin) vaccine has been extensively administered in Europe to infants and to Mantoux-negative adults. Since the quality of the vaccine has been improved, it is claimed that it has very materially reduced the incidence of subsequent tuberculosis. *In treatment* tuberculin must be used cautiously, beginning with a very small dose and gradually increasing, care being taken to avoid a constitutional reaction. In pulmonary cases this form of treatment is undesirable. The most successful cases of tuberculin therapy are those of genito-urinary tuberculosis, Bacillary Emulsion (B.E.) being usually employed. The initial dose should not be more than 1/100,000 of a milligram and subsequent doses must be increased very gradually.

Streptomycin (§ 515) is known to act on tubercle bacilli, but its efficacy in the various clinical types of tuberculosis is not yet fully assessed. In early cases of tuberculous meningitis, some success has been achieved with combined intramuscular and intrathecal injections. The systemic dose for adults is 2 G. and for infants 0·02 G. per lb. body weight daily: the intrathecal dose by lumbar puncture for adults is 0·1 G. and for infants 0·05–0·075 G. daily.

HYDROPHOBIA.—The Pasteur treatment of hydrophobia with living virus has obtained a world-wide repute. Rabbits are passaged with street virus until fixed virus is obtained; then the spinal cords are removed and dried, the drying process attenuating the virus. At first the injections are made from emulsions of weak cords, *i.e.*, those dried for fifteen days; later, cords which have been dried only ten days are used: by this means the dosage is graduated. The carbolised vaccine of Semple, in which the virus is killed, is replacing the original Pasteur method.

Indications.—The earlier the treatment the better the results. This is especially the case in face bites, which are often fatal; the virus travels by the nerves, and the

nearer the bite to the brain the shorter the incubation period. The dog should not be killed but kept muzzled and chained up, and if the animal survives ten days it is certainly not infected with rabies ; if it dies, the head should be packed in ice and sent to a Pasteur institute for examination for inclusion (Negri) bodies in the cytoplasm of the brain cells. In cases of doubt start vaccine treatment at once. Immediate treatment consists in encouraging bleeding and carbolising each individual tooth bite. Never sew up the wounds until after three days ; this applies especially to face bites.

PLAGUE.—Haffkine prophylactic vaccine, which consists of a broth emulsion of plague bacilli, is an effective prophylactic for twelve months or so. The dosage is large (5 c.c.) and rather severe local and general reactions may follow. When practicable, it is preferable to give a smaller dosage, on two or three occasions, at weekly intervals. Serum treatment for plague has been tried with doubtful benefit.

CHOLERA.—A cholera bacillus vaccine has proved a valuable prophylactic in epidemics of this disease. Protection lasts for four months or so, and is of less value than T.A.B. in typhoid and paratyphoid. It is most satisfactorily employed during an epidemic, when all persons in the infected area may be immunised, which immunity will last till the emergency has passed. Two inoculations, one of 4,000 millions and the other of 8,000 millions with an interval of a week is the usual procedure. Specific treatment with serum has been disappointing but preliminary reports of chemotherapy with a new sulphonamide compound (" 6257 ")—a condensation product of sulphathiazole and formaldehyde—which can be given orally, are encouraging.

SNAKE POISON.—Calmette introduced an antitoxic serum called antivenene, prepared by inoculating animals with cobra venom, and advocated its general use in snakebite cases. It is, however, now known that antivenene in most instances is species-specific ; to be effective the antivenene must have been prepared from the same species that has bitten the patient. Polyvalent antivenenes are now available in many different countries for the prevailing poisonous snakes, including those of India (cobra and Russell's viper), Africa and South America. A monovalent antivenene is available for the Australian tiger snake (*Notechis scutatus*). The only poisonous snake in Britain is the adder, the minimum lethal dose of whose venom for an adult is probably 50 mgms. As the actual amount injected by this snake is between 5 and 10 mgms. the results are seldom grave except in small children or invalids. The antivenene most satisfactory for such cases is that prepared by the Pasteur Institute, Algiers, against the venom of the horned viper of Africa (*Cerastes Cornutus*). Supplies are available in this country through the Ministry of Health. Antivenene is given intramuscularly or intravenously in a severe case in doses up to 40 c.c. and saves life if administered up to two-thirds of the death time—*i.e.*, if the patient has received a sufficient dose of venom to kill in nine hours, serum therapy is effective if given within six hours. No case, however, is too ill to receive antivenene, and even paralysed patients with involvement of the respiratory muscles may make remarkable recoveries within half an hour of the injection. Sometimes paresis returns ; if so, serum should be re-administered without delay. Immediate ligature delays the death time, but in patients bitten by really poisonous snakes local treatment is rarely effective as a lethal dose of poison is so rapidly absorbed. Once this has happened specific antivenene is the only possible remedy. Benadryl 50 mgms. t.i.d. by mouth may combat local swelling and pain.

PNEUMONIA.—*Prophylactic* use of a vaccine composed of the prevalent types of pneumococci has proved useful in areas such as the Rand in South Africa where pneumonia reaches epidemic dimensions amongst the native workers. The protection afforded is type specific. *Treatment :* Chemotherapy with parenteral penicillin or with effective sulphonamides (Tables XXVIII, XXX), is the essential treatment for pneumococcal pneumonia. Penicillin is almost non-toxic compared with the sulphonamides, but needs to be given by frequent subcutaneous injection. It is probably preferable for elderly patients and when there is reason to believe the pneumococci are sulphonamide-resistant. As routine treatment, however, for moderately severe or mild

infections the sulphonamides are very satisfactory. The use of anti-pneumococcal serum has regressed following the success of chemotherapy but, combined with the latter, it is still of value if given early in bacteriæmic or severely toxic cases, in elderly subjects, and rarely in those patients who do not tolerate either penicillin or sulphonamide therapy. Commercial antipneumococcic sera are mainly antibacterial in action; they have little antitoxic value. Type-specific sera prepared from rabbits can be obtained (Lederle) for Types 1, 2 and 3 pneumococci and also for the vast majority of the distinct 29 serological types—the so-called "higher types"—previously classified as the heterogeneous Group 4. Treatment with serum necessitates accurate preliminary typing of the infecting pneumococcus and the use of its homologous serum. Sixty thousand units (10–25 c.c. according to type) intravenously is recommended as a minimal initial therapeutic dose. Subsequent dosage depends on clinical progress and evidence of blood infection.

Infections of the RESPIRATORY TRACT, such as chronic pharyngitis, certain types of bronchitis, bronchiectasis, asthma, and secondary infections of tuberculous cavities, react to autogenous vaccines. The infecting organisms may be streptococci, pneumococci, pneumobacilli, influenza bacilli, etc. In the throat and nose, if the predominating organisms be penicillin sensitive, a penicillin spray may prove efficacious. Infection by Vincent's organisms is amenable to penicillin.

ANTHRAX.—The serum of artificially immunised animals—*e.g.*, Sclavo's serum—is used in the treatment of anthrax and has almost entirely replaced surgical measures. It should be given promptly and in large doses—50 c.c. intravenously, and 50 c.c. intramuscularly. N.A.B. (0·6 G. intravenously) is also valuable as an adjuvant to serum and is even preferred to the latter by some authorities. The anthrax bacillus is susceptible to penicillin and encouraging results have been reported with this drug in the cutaneous form of the disease (Table XXX).

GAS GANGRENE.—In a dirt-contaminated wound with gross injury to muscle, prompt and efficient excision of the wound constitutes the most important prophylactic measure. *Passive immunity* is obtained with intramuscular polyvalent gas gangrene antitoxin, the recommended dose being *B. welchii* 9,000 units, *Vibrion septique* 5,000 units and *B. œdematiens* 9,000 units. For *treatment* the dose, given intravenously, should be at least three times the prophylactic doses and it should be repeated as long as symptoms of toxæmia persist. Since the above three chief organisms of gas gangrene are all equally sensitive to penicillin, the latter is indicated both in prophylaxis and treatment. Local application alone is not sufficient for an established infection and systemic treatment for three days is recommended. Penicillin should not replace but only supplement the other methods of treatment (excision of dead muscle and the administration of full doses of antitoxin).

MENINGOCOCCAL MENINGITIS.—Chemotherapy has entirely superseded serum treatment. Both sulphonamides and penicillin are highly effective. Of the former the compounds suitable for use are sulphathiazole, sulphadiazine, or sulphanilamide by mouth in the recommended dosage (Table XXVIII and § 503). In all severe cases, including those coming under treatment late in the acute stage, the first dose or doses of sulphonamide should be by intravenous injection. Intrathecal injections of sulphonamides are contraindicated. Penicillin, if employed, however, should be given intrathecally whenever possible, preferably combined with a short course of subcutaneous injections especially in bacteriæmic cases.

BACILLARY DYSENTERY.—*Prophylactic* vaccines have not proved satisfactory on account of their liability to cause severe reactions. Besredka's oral vaccine has had some success. *Treatment.*—Bacilli of the dysentery group are resistant to penicillin, but Shiga and particularly Flexner infections respond well to sulphaguanidine—a drug of the sulphonamide group which is not readily absorbed from the bowel. For Sonne infections succinyl-sulphathiazole appears to be more effective than sulphaguanidine. Streptomycin is also effective (Tables XXVIII, XXIX). In toxic cases, chemotherapy may be supplemented with 50–100 c.c. of antitoxic serum intramuscularly or intravenously. Shiga serum is the most potent, but the Flexner type is

also satisfactory: a polyvalent variety combining the two is available. No effective serum against the Sonne strain has been produced, but this form is generally mild.

§ 522. **Notification and Isolation.**—Two duties are laid upon the medical practitioner in cases of the commoner infectious maladies: (1) NOTIFICATION of the case to the medical officer of health of the district in which the case arises. The notifiable complaints in most districts are Anthrax: Cholera: Diphtheria (including Membranous croup): Dysentery, amœbic or bacillary: Acute encephalitis, infective or post-infectious: Enteric fevers (typhoid and paratyphoid): Erysipelas: Farcy: Food poisoning (Foods and Drugs Act, 1938): Malaria: Measles: Meningococcal infection: Ophthalmia neonatorum: Plague: Pneumonia, acute primary and acute influenzal: Acute poliomyelitis, paralytic or non-paralytic: Puerperal pyrexia: Relapsing fever: Scarlet fever: Smallpox: Tuberculosis (all forms): Typhus fever: Whooping-cough. Any infectious disease may at any time be added to the list at the option of the Sanitary Authority. A medical man is bound, under a penalty of forty shillings, to notify any of the maladies named "immediately on becoming aware" of its existence. (2) IMMEDIATE REMOVAL of the patient to a fever hospital is compulsory for the more dangerous infectious diseases; otherwise the parents or guardians must make *proper* and *adequate* arrangements for the isolation of the case at home. In some places the removal is superintended by the medical officer of health.

Unless Home Isolation can be prompt and thorough, it is better to remove the patient to a properly organised Fever Hospital. For ISOLATION at HOME, carpets, curtains, and superfluous furniture should have been previously removed. Books and articles in use must be such as can be afterwards burned. The nurse in charge of an infectious case should wear a washable dress when on duty, and should hold no communication with others, nor should she go out of doors without having first changed her wearing apparel, and, if possible, taken a bath. An airy, quiet room *at the top of the house*, having cubic space of about 12 × 12 × 10 feet, is desirable. The air in this space requires to be changed three or four times in every hour. The bedstead should be so placed as to be accessible on both sides. The temperature, read on a thermometer suspended near the bed, and away from draughts, should be 60° F.

VENTILATION must be ample in fever cases, because of the danger of mixed infections. Many of these cases are due to droplet infection from one patient to another, and that is why mixed infections are more apt to arise when there is not free ventilation and sufficient cubic space. This partly explains the higher death-rate from infectious diseases when overcrowding occurred in former days. The direction of the wind should be constantly noted, and to avoid draught, the windows or ventilators opened on the side of the room away from the wind. A "sash-board" is an excellent contrivance for avoiding draught. It should be about 6 to 8 inches broad, and fit across the bottom of the window, so that the lower sash can be raised without a visible opening, and then ventilation takes place behind the sash-board, and also *in the middle* of the window, the air in both cases being directed upwards. The chief principle in ventilation is that the current of air always takes place from a colder to a hotter medium—usually, therefore, from outside to the inside of a room. The chimney, when the fire is alight, is the only reliable *exit*. Make the window your *inlet* in preference to the door.

§ 523. **Disinfection and Prevention.**—Before discussing the means employed for disinfection, it is necessary to consider how infection is conveyed. There are three principal ways: through the *air*, by *water* or other ingesta, and by *direct contact* or inoculation.

(*a*) As regards the *air-borne* group, their infectivity varies, also the distance to which the contagion in an active state may be carried. For instance, erysipelas and typhus probably do not spread beyond a few feet, but small-pox and scarlet fever may spread a considerable distance. A frequent mode of spread of bacteria and viruses from the mouth and throat of an infected person is by *droplet infection.* Minute drops of saliva, or nasal discharge, with adhering organisms, are dispersed for some distance into the surrounding air during talking, coughing or sneezing. These organisms usually enter *via* the respiratory tract (nose, throat, tonsils or lungs);

occasionally milk or other foods are contaminated and may be the source of the infection. Streptococci, tubercle and other bacilli can remain virulent in dust for several weeks.

(*b*) Fevers conveyed by *water, milk* or *other ingesta* are typhoid, paratyphoid, cholera, dysentery, undulant and abortus fever, and rarely scarlet fever and diphtheria. Two facts form the basis of the propagation and prevention of these diseases :—(1) All matters coming from the patient's bowel and stomach are infective, in typhoid the urine also ; (2) to produce the disease the organism must be introduced by the mouth into the alimentary canal.

(*c*) In the third group the infection is introduced into the blood or tissues of the body by means of a *wound* or a *scratch*, or by the *bite of an insect*. Our profession pays a penalty every year to this group of disorders—a pathologist receives a scratch during post-mortem work, or a surgeon pricks his finger during an operation on a septic appendix. Some of these disorders were formerly considered to depend upon climatic influence, *e.g.*, malaria, which is now known to be introduced into the body by the bite of a mosquito. Tetanus enters through a wound or scratch contaminated with soil ; plague is conveyed by rat fleas ; typhus by lice. Others in this group are the fevers due to tick bites, glanders, anthrax and hydrophobia.

The procedure for disinfection differs somewhat according to which of the above three groups the fever belongs. There is now an increasing tendency to concentrate chiefly upon current disinfection during the illness. Formerly much stress was laid on fumigation and spraying the patient's room after the illness. With careful current disinfection it is unnecessary to have terminal disinfection after measles, scarlet fever and diphtheria.

Current disinfection, i.e., that carried out during the illness in the sickroom. All unnecessary furniture and furnishings such as curtains and carpets should be removed. Hæmolytic streptococci can be conveyed by books, but as a general rule infection (except with small-pox and typhoid fever) is rarely conveyed by bedding and other inanimate objects. Before being washed, the *bed-linen*, blankets and clothes must be soaked in a 5 per cent. solution of phenol for 12 hours. Food and drinking *utensils* should be boiled for 5 minutes ; they must also be protected from flies. *Thermometers* are kept in 5 per cent. phenol. The nurse should disinfect her hands in 1 in 1,000 perchloride of mercury or in dettol. *Sputum* and nasal discharge should be collected in gauze or paper handkerchiefs and burned. Allow no *dust* to accumulate.

With fevers conveyed by water and other ingesta, in addition to the above precautions, it is essential that the excreta are covered and mixed with 5 per cent. phenol, or an equal bulk of 20 per cent. chlorinated lime in water, and allowed to stand for two hours before being emptied down the drain pipe or buried in earth. *Prophylaxis.*— All drinking water should be boiled if there is the slightest suspicion of its being contaminated by leakage, soakage (however small) from cesspools, drains, or the reckless casting of slops, and by flies. Food utensils must be disinfected carefully by boiling, and flies must be prevented from access to food and to excreta. All handlers of food or every individual where large groups of men are crowded together, as in armies, should be examined and treated if found to be "carriers," *i.e.*, apparently healthy persons in whose excreta the cysts of the amœba of dysentery or typhoid, paratyphoid or cholera germs abound.

List of *common disinfectants* for use in the sick room : Extreme heat (200° F. or more, and preferably moist) ; fumes of burning sulphur (SO_2) ; chlorinated lime, 1 to 5 per cent. ; phenol, 5 per cent. ; dettol ; formalin, 2 to 10 per cent. ; lysol, 1 per cent. (or liq. cres. sap. fort., 1½ oz. to 1 gallon water) ; corrosive sublimate, gr. 10 to 1 gallon.

Terminal Disinfection.—Burn as many articles of clothing as possible ; boil others. Mattresses and blankets, books and all clothing which cannot be boiled, should be sent to be disinfected by steam. Boots and shoes can be washed over with lysol. Furniture should be moved from walls, and drawers, cupboards, etc., disinfected with a liquid spray. Wallpapers in some cases are stripped and the walls treated with

hot lime. Doors and windows are closed, crevices are stopped, and the whole room is kept closed for six hours after being thoroughly sprayed with formalin by means of a hand-worked pump. Formalin 8 oz., glycerin 8 oz., water 1 gallon, is the disinfectant most often employed. Then the windows are opened and all is washed down with hot water and soap, and the room is well aired.

Disinfection and the PREVENTION OF DISEASES caused by scratches, bites, etc., differ in each individual case. Thus septicæmia and tetanus almost ceased in surgical cases with the introduction of cleanliness and asepsis. Various tropical fevers are conveyed to man by the bites of mosquitoes, flies, fleas, and bugs. The prophylaxis of these conditions includes measures directed to the extermination of the insect responsible and avoidance of places in which they are known to be present. Insecticides and repellents such as D.D.T. and dimethylphthalate play a large part in preventing disease. Where plague is endemic rats must be destroyed; where bugs infected with disease are found it may be necessary to burn the huts, etc., in which eggs are likely to have been deposited. With many of these insect pests knowledge of their life-history is the necessary preliminary to effective steps for their destruction.

§ 524. **The Treatment of pyrexia and hyperpyrexia** comprises six indications:

(1) *Heat production can be diminished and heat loss increased* to some extent by means of *drugs*, known as antipyretics, such as antipyrine and phenacetin. The first of these is most efficacious, but it requires care, on account of its depressing effect on the heart, and the reaction which follows some hours later. Cryogenin gr. 10 to 15 is the least depressing antipyretic. Quinine in full doses (say 5 grains every three or four hours) may be given until the temperature comes down or physiological symptoms are produced (singing in the ears, deafness, headache, etc.). Salicylates, especially in rheumatic affections, and aconite are also useful. Among the diaphoretics are liquor ammoniæ acetatis, potassium nitrate, spiritus ætheris nitrosi, and camphor: also lemon drinks, dilute acids, and salines.

The Graduated Bath.—Place the patient in a bath one-third full of water at 90° or 95° F. Every five minutes reduce the temperature 5° until 60° F. is reached. If the fever be not then reduced to 100° F. or lower, continue for further quarter of an hour. The pulse must be closely watched, and stimulants given if necessary.

The Wet Pack.—Take off the night-shirt and superfluous bedclothes, and place the patient on a blanket. Moderately wring a sheet out of ice-cold water and lay it along his side. Gently roll him over on to it, and completely envelop him in it, head and all, except the face, so that it is next to his skin, without creases or air, between the legs and beneath the arms. Cover these latter with wet towels. Then put two cradles over the patient, and blankets over all. Leave him thus packed for twenty to forty minutes, until his temperature, taken in the mouth, is reduced to the required extent.

Tepid Sponging.—Lay the patient in a blanket and sponge him gradually all over with tepid water (about 75°). Do half the body at a time, the other half being covered up. Continue the process for twenty to forty minutes, until the fever is reduced.

(2) *The application of ice* in large ice-bags for the head, chest, and abdomen has been used when other means are not available, but the weight of the bags and their localised application are objections to their use.

(3) *Diminish the work done by the internal organs* by diet (§ 297. XVIII), and by promoting the action of the skin and bowels, in order to relieve the kidneys.

(4) In all fevers it is necessary *to watch the heart* and blood pressure carefully, and, if necessary, administer suitable stimulants, such as nikethamide B.P. (coramine) or leptazol. The pulse should be examined several times a day in all fever cases.

(5) *Symptomatic treatment* has been dealt with in the preceding pages.

(6) *Watch for and treat complications* as they arise. The chief of these are (i.) cardiovascular (*vide supra*), and (ii.) delirium and insomnia. If the delirium be of the *raving* kind, chloral and bromides should be given in full doses; if of the *muttering* or typhoid variety, stimulants. Insomnia may be relieved by the same treatment. (iii.) Pulmonary complications, (iv.) suppression or retention of urine, and (v.) collapse, are all dealt with elsewhere.

CHAPTER XVI

GENERAL DEBILITY, PALLOR, EMACIATION

A FEELING of general weakness and lassitude is a symptom common to many diseases, but we are now concerned with those in which this is the only obvious, or at least the most prominent, symptom for which the patient seeks relief. Diseases in which debility is the chief symptom may be classified clinically into two great groups according to whether they come on acutely and are attended by pyrexia, or not. Debility coming on acutely and attended by pyrexia was fully dealt with in the preceding chapter. There still remains a large group of diseases in which the weakness is of gradual onset, runs a chronic and indefinite course, and is unattended for the most part by any notable elevation of temperature; and these diseases may be attended by pallor or by emaciation. Here we shall often meet with the beginnings of disease, beginnings which may, however, lead to a serious and fatal issue. It is, therefore, of the highest importance that an exact diagnosis should be made, and treatment adopted as early as possible.

Many of the debilitating conditions mentioned in this chapter may be unattended by any other symptom, or only by the pallor of anæmia or the wasting of malnutrition, and many give rise to no characteristic anatomical changes: even their pathology may be obscure.

PART A. SYMPTOMATOLOGY

§ **525. General Debility.**—Malaise, lassitude, inability to complete a day's work, are some of the terms used to describe the symptom under consideration, which is essentially chronic in its course. The weakness is generalised, and it may affect the mind as well as the body, for there is not only a disinclination to take muscular exercise, but an inability to concentrate the attention or accomplish mental work. The weakness may vary in kind and degree from very slight malaise to a total incapacity to move. Many diseases in this category are apt to be overlooked in their earlier and more curable phases. The patient may attribute his ailment to "slight digestive derangement," or think he has "been working too hard," or "wants a change," and perhaps he calls on his doctor "as he was passing" just to confirm his own diagnosis and "give him a tonic." These cases may tax the young practitioner's skill and tact in several ways. Fresh from studying instances of marked diseases in hospitals, he may regard these cases as trivial and "uninteresting"; and even if he detects the beginning of some insidious malady the patient may meet his suggestion of serious ailment not only with surprise, but even with resentment and distrust. Some tact, therefore, is required, and the practitioner may find it wise to confide in some discreet friend or relative.

Fallacies.—The distinction of general debility from *paralysis* is not usually difficult, though patients with polyneuritis, early paraplegia, general paralysis of the insane, bulbar paralysis, and various other forms of paresis, often complain simply of weakness. Cases of *malingering* offer far greater difficulty in diagnosis from general debility, for in both cases we are almost entirely dependent upon the patient's own statements. The question of motive should be considered and an exhaustive examination made by the most up-to-date methods, but even then we may in justice be compelled to give the patient the benefit of the doubt. In many cases it is only by keeping the patient under daily observation, and with the aid of intelligent, experienced, and well-trained nurses, that a correct conclusion can be gained. *Hysteria* and *neurasthenia* may require to be distinguished from debility. The *Causes* of debility are discussed in §§ 535 and 557.

Pallor of the Skin—*i.e.*, deficiency of its normal colour—is a frequent accompaniment of cases in which debility is complained of by the patient. The causes are discussed in §§ 535 *et seq.*

Fallacies.—Slight *jaundice* may resemble some forms of pallor. In *town-dwellers* pallor of the face is common. In certain "delicate" families a pale face is more or less normal. Europeans who have lived long in the *tropics* are habitually pale and "anæmic" looking, but the blood may not reveal any changes of anæmia. On the other hand, patients with dyspepsia may have a flushed face, though undoubtedly suffering from anæmia. In many *nervous* conditions transient constriction of the vessels may cause a pallor which may be mistaken for anæmia. Many patients who "go white" with nervous emotion are mistakenly supposed to be anæmic; in anæmia the pallor has a waxy or yellow tinge, which is absent in pallor of vasomotor origin. With a glance at the colour of the lips, conjunctivae and mucous membranes one can usually distinguish the conditions.

Emaciation, or loss of flesh, may also be associated with general debility, and its presence adds considerably to the gravity of a case, for it indicates either serious organic disease such as cancer or tubercle, or definite defect in the alimentation or metabolism of the body, such as is produced by intestinal trouble or chronic nephritis. It is manifested to the patient by his clothes becoming looser, or his face becoming thinner, and to the physician by pinching up a fold of skin between the finger and thumb. But the only reliable test is a definite loss of weight, and it is advisable at the outset to record the weight of all patients who come to us complaining of debility. When possible *note the exact weight;* the patient should be weighed in a dressing gown, of which the weight is known. A reliable weighing machine should be in every consulting-room. The causes of emaciation are discussed in §§ 554 *et seq.*

Fallacies.—A normal loss of adipose tissue may occur about the climacteric, but the reverse is quite as usual. Amyotrophy, unless generalised, is not to be confused with emaciation; it is usually localised. The diet a person has been taking will, within certain limits, influence his weight

considerably, and one who has been taking only nitrogenous food may be many pounds under his normal weight.

PART B. PHYSICAL EXAMINATION

§ **526.** The physical examination of cases of general debility, pallor, or emaciation, comprise (1) EXAMINATION OF THE VISCERA; (2) OBSERVATIONS ON THE WEIGHT, and in some cases on the TEMPERATURE; and (3) AN EXAMINATION OF THE BLOOD.

1. An examination of the VISCERA AND EXCRETA should be systematically conducted (see Scheme, pp. 6 and 7), because we may be dealing with some incipient disease, the signs of which are obscure. Inquiries should be especially directed to the state of the digestive organs.

2. The WEIGHT of the patient should be noted, and, if possible, compared with previous records. It is a wise precaution to take the patient's TEMPERATURE, and to obtain a series of records (§ 471).

3. An examination of the BLOOD is necessary. This consists of (1) estimation of hæmoglobin; (2) blood-counts of the red and white corpuscles; (3) examination of blood-films. In most cases these three will be sufficient for a routine examination; but in other cases it is necessary to make (4) an examination for parasites and other abnormal constituents; (5) certain physical and chemical properties of the blood, and (6) biopsy of sternal bone marrow.

EXAMINATION OF THE BLOOD

§ **527. Apparatus and Methods.**—APPARATUS REQUIRED.—A Tallquist hæmoglobin scale or Sahli's hæmoglobinometer; a Thoma or Neubauer hæmocytometer; a sharp triangular needle; Hayem's solution; Toison's fluid; Geimsa or Leishman's stain; distilled water.

For the *estimation of hæmoglobin* Sahli's hæmoglobinometer is used. For *counting the blood-cells* the Thoma or Neubauer hæmocytometer is employed. Diluting solutions are required for this purpose. For counting the red cells a solution of normal saline may be used, or Hayem's solution; sod. chloride 1 grm., sod. sulphate 5 grms., hydrarg. perchlor. 0·5 grm., aq. dest., ad 200 c.c. For counting the white cells a 0·3 per cent. solution of acetic acid coloured by methylene blue is used, or Toison's fluid (methyl violet, 0·025 grm.; neutral glycerin, 30·0 c.c.; distilled water, 80·0 c.c. Add to this a solution of sodium chloride 1·0 grm., sodium sulphate 8·0 grm., distilled water 80·0 c.c., and filter). The instruments must be carefully cleaned before being put away, first with water, then, either by acetone alone, or by alcohol followed by ether, and finally dried.

METHOD OF OBTAINING BLOOD.—To obtain satisfactory results, any series of observations on a patient should be carried out as far as possible under similar conditions, to obviate physiological variations in the blood after meals, cold baths, exercise, etc. After gently cleaning the ball of the finger or the lobe of the ear with cotton-wool soaked in ether, puncture the chosen site with a sterile triangular needle: a sufficiently deep puncture should be made to obtain blood without squeezing. Alternatively 5 c.c. of blood from a vein may be transferred immediately into a tube containing 4 mgms. of solid potassium oxalate and 6 mgms. of solid ammonium oxalate: these quantities give an isotonic suspension.

§ **528. Hæmoglobin Estimation** may be performed (i.) by the *Tallquist scale*, which consists of a lithographed scale of tints. A drop of blood is sucked up by a piece of the blotting-paper supplied, and compared with the scale of tints as soon as the stain has lost its humid gloss. The figures beside the tints represent the percentage

of hæmoglobin present, the normal being 100. The estimation must be made in full daylight and gives only an approximate result. (ii.) By *Sahli's hæmometer* (Fig. 126). First add *N*/10 hydrochloric acid to the graduated tube up to the mark 10: then suck 20 cu. mm. of blood into the special pipette. Transfer the blood to the acid solution by gently blowing out the blood while the point of the pipette is under the surface of the acid: with alternate sucking in and blowing out, it is possible not only to wash all the blood into the acid solution, but to ensure

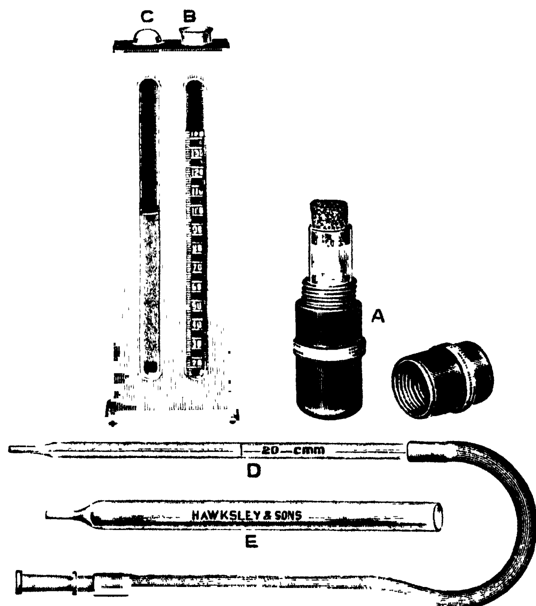


FIG. 126.—SAHLI'S HÆMOMETER.

thorough mixing with the acid. The blood soon turns dark brown as acid hæmatin is formed: after waiting 10 minutes for this to be complete, add water a little at a time, inverting the tube several times between each addition, until the colour exactly matches the standard. The figure on the graduation at the upper level of the fluid marks the percentage of hæmoglobin in the blood—the result is the same by artificial and natural illumination. (iii.) *Haldane's method* of converting hæmoglobin into carboxy-hæmoglobin and (iv.) the *photo-electric cell* method in which hæmoglobin is first converted to alkaline hæmatin, are only appropriate to fully equipped laboratories.

The percentage of hæmoglobin is always expressed as a percentage of a normal standard. Thus 87 on the scale indicates that the amount is 87 as compared with the normal of 100. Unfortunately most of the methods used have a number of different standards, some instruments being calibrated to one, and some to another. By the Sahli method, 100 per cent. usually represents 14·5 G. of hæmoglobin per 100 c.c. of blood: therefore examine the instructions with the instrument to see the standard used. To avoid confusion, many hæmatologists prefer to express the hæmoglobin in grammes per cent. (and see Table XXXI).

Significance of Increase or Decrease of Hæmoglobin.—An increase of hæmoglobin above 100-105 per cent. is unusual: a *temporary increase* occurs in states of dehydration due to the lowered volume of circulating plasma. A more *permanent increase* occurs after residence at high altitudes for some weeks, and also in polycythæmia (§ 31)—in this case the figure may rise even to 150 per cent. A decrease of hæmo-

globin (below 83 per cent., or 12 G. per 100 c.c.) is the essential feature in all anæmias. Before discussing further the significance to be attached to a low hæmoglobin value, it is well to estimate the number of red cells in the blood, in order to determine the *colour index.*

§ **529. Red Blood Cell Count.**—The apparatus consists of a mixing pipette, a graduated counting chamber or hæmocytometer (we recommend either the Thoma or the Neubauer pattern) and Hayem's diluting fluid. With clean dry instruments all to hand, obtain a drop of blood as already described (§ 527). Suck blood into the special pipette to the mark 0·5: it is necessary to be very precise with this measurement and if blood is accidentally introduced into the mixing chamber, the instrument must be cleansed and the process started again. When the blood reaches the required mark, wipe the end of the pipette clean, then plunge it into the diluting fluid and fill it to the mark 101 (dilution 1 in 200). [If the blood is collected at the bedside, it is necessary to ensure its safe conveyance to the laboratory: for this purpose, remove the rubber tubing and slip a broad elastic band over the pipette to seal the two ends.] Thoroughly mix the blood and the diluting fluid by rotating the pipette while it is held horizontally, and then blow out and discard about a third of the contents of the bulb. Hold the pipette at an angle of 45 degrees, let the tip of the pipette touch the space between the cover slip and the counting chamber (Fig. 127) and allow fluid to run under the cover slip by capillary attraction. Bubbles must be avoided: the fluid should just fill the space under the cover slip between the two grooves. If the fluid overflows from the counting chamber the apparatus should be cleaned and the operation repeated. Small bubbles may be got rid of by gently easing the cover slip in a sideways direction and bringing them to the edge of the counting chamber. When satisfactorily performed the counting chamber should show Newton's rings. Having adjusted the counting chamber under the $\frac{1}{6}$th objective of a microscope, proceed to count the cells present. The platform is ruled by cross lines each enclosing a space of $\frac{1}{400}$ of a square millimetre; the depth of each square with the cover-glass on is $\frac{1}{10}$ of a millimetre. The squares are marked out into sets of 16 by double lines. Count the red cells in five of such sets—that is in 80 squares. Where the corpuscles lie *upon* the lines, count those on the upper and left side lines only. *Calculation for counting red blood-cells* in 1 cu. mm. of blood: The 400 squares equal $\frac{1}{10}$ cu. mm.; 80 squares are counted, and equal $\frac{1}{50}$ cu. mm. They are found to contain, for example, 480 corpuscles. Adding 0000 to the number counted gives the number per cu. mm. For example, $\frac{1}{50}$ cu. mm. contains 480 cells; therefore 1 cu. mm. contains 480 × 50 × 200—*i.e.*, 480 × 10,000 = 4,800,000 cells.

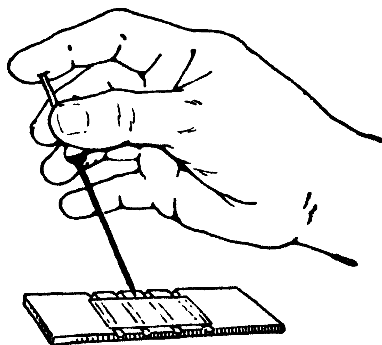


FIG. 127.—BLOOD COUNTING CHAMBER.
By permission from Whitby and Britton "Disorders of the Blood."

Significance of Increase or Decrease of Red Blood Cells.—*In health* the average number of red cells per cu. mm. is about 5,000,000 (Table XXXI). It is increased in plethoric persons, after fasting and sweating, after removal to high altitudes, and may be 7,000,000 or 8,000,000 in the newly born. It is decreased in the later stages of pregnancy. In *disease* it is increased (i.) when there is defective oxygenation in the lungs, as in chronic lung disease; (ii.) in congenital heart disease, when insufficient blood is oxygenated, the number may rise to 10,000,000 per cu. mm. or more; (iii.) when there is hæmo-concentration as after diarrhœa or vomiting, after severe burns and in the crises of Addison's disease. (iv.) In polycythæmia (§ 31). It does not occur with cyanosis *per se*, but only when cyanosis is accompanied by one of the conditions above mentioned. Diminution in number

is found after hæmorrhage and other secondary anæmias, in leukæmia and other blood disorders. In pernicious anæmia the diminution may be very great, and in hypochromic anæmia very slight.

The Relation of the Hæmoglobin percentage to the Number of the Red Cells is known as the *Colour Index*: this represents the amount of hæmoglobin in each red cell. It is calculated by working out a numerical fraction as follows: take as the numerator the amount of hæmoglobin found as a percentage of the normal, and as the denominator the number of red corpuscles found as compared with the normal number (5,000,000). For example, if the observed hæmoglobin is 40 per cent., and there are 4,000,000 red cells, the colour index is 40/100/4,000,000/5,000,000, *i.e.*, 0·50. When the Sahli method is used and the normal percentage is 85 per cent., the figures would be 40/85/4,000,000/5,000,000, *i.e.*, 0·58. The colour index is normally 0·85 to 1·15, and in anæmia may be lowered, raised, or within this normal range. An alteration in the index means that the average hæmoglobin content of the average red cell is increased or decreased. Theoretically, this may be accomplished in two ways: (i.) the size of the cell may remain constant and the hæmoglobin saturation be increased or decreased, but in practice the former does not occur, because the normal red cell is saturated with hæmoglobin; (ii.) the size of the red cell can be increased or diminished, the average hæmoglobin per *unit* volume of red cell (*i.e.*, the saturation) remaining constant. Then the average amount of hæmoglobin for each whole red cell compared with the normal cell will be increased or decreased. For this reason anæmias with large red cells (macrocytic anæmias), such as pernicious anæmia, have usually a high colour index, and anæmias with small red cells (microcytic anæmias) have a low colour index irrespective of whether the cells are *saturated* with hæmoglobin or not. Thus it is important for diagnosis and treatment to estimate the *mean corpuscular hæmoglobin concentration* of the red cells, which gives the amount of hæmoglobin per unit volume (saturation) of the red cells. This is calculated as follows:

$$\frac{\text{Hæmoglobin in grammes per 100 c.c. of blood}}{\text{Volume of packed red cells in c.c. per 100 c.c. of blood}} \times 100$$

The normal range is 32–38 per cent., and if the hæmoglobin concentration falls below 32 per cent. *hypochromia* is present and iron therapy is indicated. If the mean corpuscular hæmoglobin concentration lies between 32 and 38 per cent., the anæmia is termed *normochromic*. An increase above 38 per cent. seldom if ever occurs; the term "hyperchromic" should, strictly speaking, not be used at all. When it is used it should indicate a colour index greater than normal and should not imply necessarily saturation of the red cells with hæmoglobin.

The Size of the Red Cells as the *mean corpuscular diameter* may be estimated accurately by the Price-Jones method, in which 500 stained red cells are actually measured at a known magnification, or by the halometer, where the halo made by an unstained blood film is measured. This gives an approximate figure only. The normal range of the mean corpuscular diameter is 6·7–7·7 μ, with an average of 7·2 μ. The cell size as the *mean corpuscular volume* is usually now measured accurately and simply by the hæmatocrit. Wintrobe's hæmatocrit, a graduated glass tube 11 cm. long by 2·5 mm. bore, is the instrument recommended. Oxalated blood (§ 527) is pipetted into the hæmatocrit up to the 10 mark. The whole is then centrifuged till the red cells are packed into a constant volume and the percentage volume of the packed red cells is read. The mean corpuscular volume is calculated in cubic microns as follows:

$$\frac{\textit{Volume of packed cells in c.c. per 1,000 c.c. of blood.}}{\textit{Red cells in millions per cu.mm.}}$$

Thus when the volume of packed cells obtained from the hæmatocrit after centrifuging is 24 per cent. and the red cell count, 2,000,000, the mean corpuscular volume is 120 cubic microns. The normal range is 78–94 cubic microns. Blood with a figure above 94 cubic microns is macrocytic; blood below 78 is microcytic. From the

point of view of treatment, it may be said that generally liver extract should be given in all cases of macrocytic anæmia.

TABLE XXXI.—NORMAL HÆMOGLOBIN AND RED CELL MEASUREMENTS.

	Average.	*Range.*
Hæmoglobin		
Haldane scale (men) ..	113 per cent. (15·6 G.)	101–123 per cent. (14–17 G.)
,, ,, (women) ..	98 ,, ,, (13·7 G.)	85–112 ,, ,, (12–15·5 G.)
Sahli scale (men)	107 ,, ,, (15·6 G.)	(14–17 G.)
,, ,, (women) ..	94 ,, ,, (13·7 G.)	(12–15·5 G.)
Red Cells (men)	5,500,000	5,100,000–6,500,000
(women)	4,800,000	4,200,000–5,500,000
Colour Index	0·9	0·85–1·15
Mean Cell Diameter	7·20 μ	6·7–7·7 μ
Mean Corpuscular Hæmoglobin Concentration	34 per cent.	32–38 per cent.
Mean Cell Volume	86 cu. microns	78–94 cu. microns

§ **530. White Blood Cell (Leucocyte) Count.**—This is performed in a manner parallel to that of the red cells. Take the special pipette, marked 11 above the bulb, and from a finger prick suck blood to the mark 0·5. Then fill the pipette to the mark 11 with Thoma's or Toison's diluting fluid. Thoroughly mix the blood and fluid in the mixing chamber, and transfer a small portion to the counting chamber in the manner described for the red blood-cells (§ 529).

Counting the Leucocytes.—In the Thoma counting chamber the 400 small squares are counted and contain, say, 43 leucocytes. Another 400 small squares are counted and contain, say, 37 leucocytes. Now, since 400 squares equal $\frac{1}{10}$ cu. mm., 800 squares equal $\frac{1}{5}$ cu. mm., therefore $\frac{1}{5}$ cu. mm. contains 43 + 37 = 80 leucocytes, and 1 cu. mm. contains 80 × 5. But the blood is diluted 20 times, so that 1 cu. mm. of blood contains 80 × 5 × 20—*i.e.*, 80 × 100 = 8,000 leucocytes (*i.e.*, add 00 to the number counted). In the Neubauer hæmocytometer four large squares are ruled, each measuring one square millimetre. Two of these large squares should be counted and 00 added. In healthy adults, the leucocytes number 4,000 to 11,000 per cu. mm. (Table XXXII).

§ **531. Microscopic Examination of Blood and Blood Films.**—Alterations in the shape and size of the blood-cells may be seen by examination of fresh blood-films, but for accurate examination of the structure of the red and white cells and a differential count of the leucocytes it is essential that blood-films be fixed and stained. Blood may be obtained from the finger or ear by the method above described (§ 527). A microscopic examination of fresh blood may be made by applying a clean slide lightly to the drop of blood, placing a cover-glass on it, and examining under the microscope. For this method any good microscope will do with a $\frac{1}{6}$-inch English objective and a No. 4 eyepiece; but for the differential examination of leucocytes and for bacteria a $\frac{1}{12}$-inch oil immersion lens is necessary. It is a great advantage to have a nose-piece on the microscope capable of carrying two or three objectives, to examine the specimen first with a low, and then with a high power. It is well to be familiar with the changes the blood undergoes in a short time after such a method of preparation. To preserve such a specimen for some hours, ring the edge of the cover-glass with vaseline to prevent evaporation. In this simple way we can note any abnormality in the shape of the red cells, or the presence of abnormal constituents, such as particles of pigment, filaria sanguinis hominis, or the spirillum of relapsing fever. We may also note any excess of white cells. Rouleaux formation is also noted in normal fresh blood—*i.e.*, the red cells run together, leaving clear the concave spaces in which blood platelets are seen. The white corpuscles are spherical, clear, and nucleated.

A film may be made upon a cover-glass or a slide; for ordinary purposes the slide method is the easier. It is essential that the slides or cover-glass be absolutely clean

and free from greasiness. Lay the surface of the slide lightly on the drop of blood exuding from the finger or ear, and with the smooth edge of another slide held at 45° spread out the blood in a *thin* film by pushing the drop along the surface, so that it forms an even film. The film can be made thicker by increasing the angle between the slides or by pushing the slide more rapidly. Care must be taken not to handle the slides too much or to breathe upon them. Allow the film to dry, and it will if necessary keep for several days without further precautions.

Staining.—The protoplasm of the red cells takes up acid dyes only; normal nuclei take up basic stains. The granules in the protoplasm of the various leucocytes take up different stains; some have an affinity for acid stains such as eosin, and are known as eosinophil granules; some take up basic stains such as methylene blue, and are called basophil granules. The granules in the ordinary polymorph leucocyte were at one time supposed to take up both acid and basic stains, and hence were named neutrophil granules. It is now known that these granules take up faintly acid stains, though the cell is still named polymorph neutrophil for purposes of description and differentiation. In staining we therefore employ a composite stain which allows each part of the blood-cells to take up the dye for which it has affinity. When the film is dry, cover with Leishman's stain for half to one minute. Then add an equal part of distilled water; leave six to eight minutes; wash rapidly and dry rapidly by shaking the slide in the warm air a long distance above a Bunsen burner. Drying with blotting-paper tends to spoil films. The *Oxidase Staining Reaction* is used in order to distinguish young cells of the lymphocyte type from young cells of the myeloid series. It depends upon the presence of oxidases in the granules of the myeloid cells; the reaction is negative in myeloid cells which are very immature, *i.e.*, the more primitive myeloblasts, and therefore in these cells it does not serve to differentiate lymphoid from myeloid cells. Goodpasture's stain (an alcoholic solution of sodium nitro-prusside, benzidine, basic fuchsin and hydrogen peroxide) is used in the same way as Leishman's stain. The cells containing oxidases contain deep blue granules.

Variations of the Red Blood Corpuscles in Disease may consist of (1) variability in shape (poikilocytosis); (2) variability in size (anisocytosis); (3) polychromasia; (4) punctate basophilia; (5) reticulocytes; and (6) nucleated red cells. Normal red cells are circular, bi-concave, non-nucleated discs measuring in size 6·7 to 7·7 μ or $\frac{1}{3200}$ of an inch.

(1) Poikilocytosis is a variability in the shape of the red cells. They may resemble a flask, a pear or a kidney. This change is seen in almost all cases of severe anæmia, but is most striking in pernicious anæmia.

(2) Anisocytosis is variability in size of the red cells. This also is seen in its most striking form in pernicious anæmia. A preponderance of large cells gives a high colour index (macrocytic, megalocytic or "hyperchromic" anæmia); a preponderance of small cells, a low colour index (microcytic and usually hypochromic anæmia).

(3) Polychromasia (polychromatophilia) is a variation in the colour of the red cells. In health the red cell stains an even bright pink colour with Leishman's stain, except for the central pale area due to the thinness of the cytoplasm there, the cell being a bi-concave disc. In many cases of severe anæmia the red cells stain differently, some being blue, others blueish-red, and others bright pink. The bluer staining cells are younger cells and their presence in the blood is called polychromatophilia.

(4) Punctate basophilia ("stippling") is the term used to indicate the presence of blue dots in the pink cytoplasm of the stained cell. These blue dots consist of the same material which constitutes the blueness of polychromasia. Sometimes one or two much larger blue bodies are seen, believed to be nuclear remnants, and these are called Howell-Jolly bodies. Punctate basophilia is seen especially in anæmia due to lead poisoning, but also in severe anæmia due to other causes, as in pernicious anæmia.

(5) **Reticulocytes** are red corpuscles which when stained *supravitally* show a net-like structure within. As they are young recently-formed corpuscles, their numbers

PLATE III.

(Stained by Wright's method.)

Figs. a, b, c, d, e, f, are drawn to the same scale.

(*a*) Neutrophil polymorph leucocyte.

(*b*) Small lymphocyte, and a normal red cell.

(*c*) Large lymphocyte. The nucleus is about the same size, and has the same structure as that of the small lymphocyte, but the cytoplasm is abundant; this cell shows several "azure granules."

(*d*) Monocyte, or large mononuclear leucocyte. Note contrast between the structure of the nucleus of this cell and that of the lymphocytes. The cytoplasm is cloudy and proper staining shows numerous minute granules.

(*e*) Basophil leucocyte. Note the very pale nucleus, lobed like that of the neutrophil polymorph leucocyte.

(*f*) Eosinophil leucocyte. The nucleus stains a darker colour than that of the basophil leucocyte, but not so deep as that of the neutrophil. Note colourless cytoplasm in this and the basophil leucocyte.

Figs. g, h, i, j, are drawn to the same scale, which is smaller than that of the cells a to f.

(*g*) Part of a blood film of pernicious anæmia. Note large red cells without pale central area, indicating that they contain more hæmoglobin than the normal red cells. The nucleated cell is an early normoblast; the nucleus is characteristic; the cytoplasm is a pale blue, for the hæmoglobin in this particular cell is not fully developed. Some punctate basophilia is present.

(*h*) Part of a blood film of simple hypochromic anæmia. Note pallor of red cells; many have large, paler central areas, indicating that they contain less hæmoglobin than a normal red cell. The picture shows some blood platelets.

(*i*) Part of a blood film of lymphatic leukæmia. One cell, of the normoblast type, is a nucleated red cell, recognised by the characteristic nucleus and the pale pink cytoplasm. The other nucleated cells are lymphocytes; two with pale nuclei, containing nucleoli, are lymphoblasts. One lymphocyte is undergoing karyokinesis. The nuclei of some of the lymphocytes are abnormal.

(*j*) Part of a blood film of myeloid leukæmia, showing (1) a small basophil leucocyte, (2) a myeloblast, with a pale nucleus, several nucleoli, and no granules in the cytoplasm. (3) A pre-myelocyte, with a nucleus like that of the last cell, but the cytoplasm contains granules. The other three cells are two myelocytes, and a meta-myelocyte, the latter having a bent nucleus. (4) A normoblast in the centre, with the characteristic nucleus, and pink-stained cytoplesm.

PLATE III.





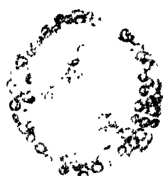


c





f



e

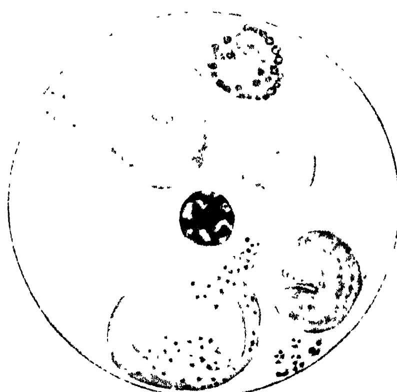


g

h



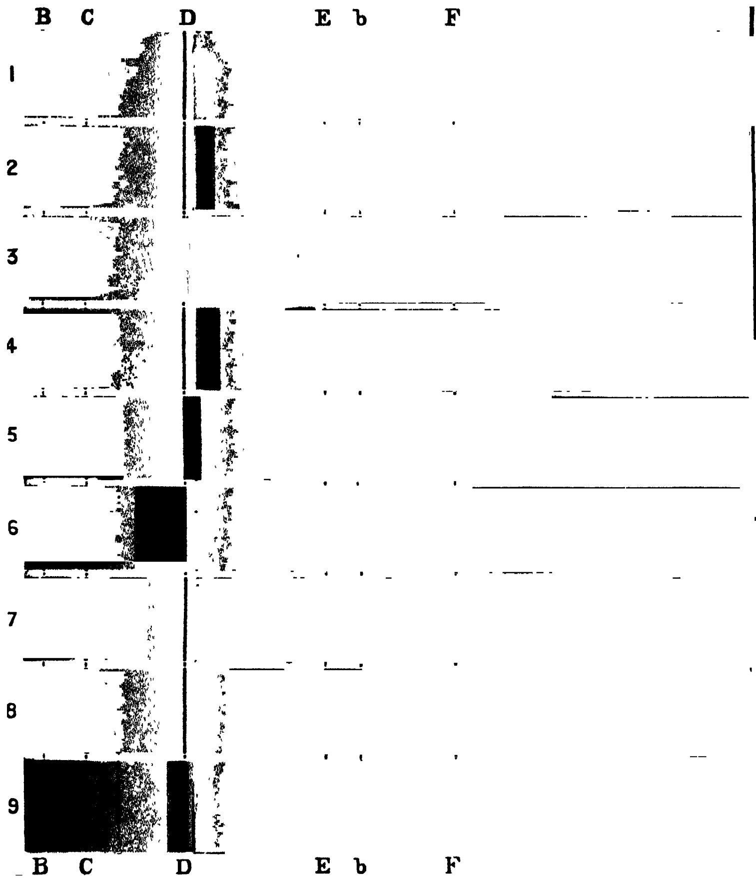
i



j

PLATE IV.

BLOOD-SPECTRA COMPARED WITH SOLAR SPECTRUM.



1. Solar spectrum.
2. Spectrum of dilute solution of oxyhæmoglobin.
3. ,, ,, reduced hæmoglobin.
4. ,, ,, carbonic oxide hæmoglobin.
5. ,, ,, acid hæmatin in ethereal solution.
6. ,, ,, alkaline hæmatin.
7. ,, ,, methæmoglobin.
8. ,, ,, reduced hæmatin (hæmochromogen).
9. ,, ,, acid hæmatoporphyrin.

indicate the rate at which red cells are being produced. In ordinary Leishman stained films, they appear either as polychromatic or as stippled cells. The normal percentage is 0·1–1·0 per cent. *Staining.*—Films of 0·3 per cent. solution of cresyl blue in absolute alcohol are made on one side of specially cleaned No. 1 cover slips by allowing drops of the cresyl blue to evaporate. A tiny drop of blood is placed on the charged surface of one cover slip, and another prepared slip placed on it in a corner-wise manner so that the blood lies between two layers of cresyl blue. After a minute, the two are slipped apart, the films allowed to dry, and counterstained with Leishman. The reticulocytes show a net-work or small specks of blue material in them.

(6) *Nucleated* red cells are found in patients with a great decrease in the number of the red corpuscles—*e.g.*, in pernicious anæmia and severe secondary anæmia; and in spleno-medullary leukæmia and myelosclerosis even without much diminution of the red cells. These nucleated red cells must be distinguished from lymphocytes, which resemble them approximately in size. The nucleated red cells differ in the character of the nuclei. The nuclei may show karyokinesis, or may be degenerate. There are three distinct forms of nucleated red corpuscles: (1) the late normoblast, about the same size as a normal red cell; (2) the early normoblast, a cell with the same type of nucleus as the first, but a much larger cell, with polychromatic cytoplasm (see Plate III); (3) the megaloblast, a large cell with a finely-woven nuclear structure, and cytoplasm which is often acidophilic, but may be polychromic. The megaloblast represents a primitive type of blood regeneration, and is rarely seen in the peripheral blood and then almost only in pernicious anæmia.

The most characteristic sign of pernicious anæmia is the presence of *macrocytes* (*megalocytes*), abnormally large red cells; and of certain hypochromic anæmias is the presence of *microcytes*, abnormally small red cells.

Variations of the Leucocytes (Plate III) occur in their absolute number (§ 530), their structure, and the relative number of one kind or another (differential count). There are several kinds of leucocytes, and it is possible to identify the cause of an increase in the leucocytes by the predominating variety present. For this purpose and in order to make a differential count to ascertain the relative proportion of the several varieties, it is necessary to employ the staining method given above.

TABLE XXXII.—NORMAL RANGE OF WHITE CELLS IN BLOOD.

The leucocytes found in healthy adults number 4,000 to 11,000 per cu. mm., and the approximate percentages and total numbers are:

	Percentage of total leucocytes.	Numbers of each type per cu mm.
Polymorph neutrophils	33 to 75	1,500 to 7,500
Lymphocytes	15 to 60	1,000 to 4,500
Monocytes	0 to 9	0 to 800
Eosinophils	0 to 6	0 to 400
Basophils	0 to 2	0 to 200

(1) In the *Polymorphonuclear Neutrophil Leucocytes*, which form 33 to 75 per cent of all leucocytes in the blood, and have an average diameter of 13·5 μ, the nucleus is long and lobed, giving the appearance of being multipartite, and the protoplasm of the cell contains fine neutrophil granules. The so-called neutrophil granules are really faintly acid. This cell originates from the bone-marrow, and is actively amœboid and phagocytic. (2) *Lymphocytes*, 15 to 60 per cent., are usually small, with one nucleus, and possess a very small amount of surrounding protoplasm. When much cytoplasm is present they are called large lymphocytes, though the nucleus is of the same size and character. They sometimes contain small granules, "azure granules." (See Plate III.) They come chiefly from the lymphatic glands and adenoid tissue. They are slowly amœboid but not phagocytic. (3) *Monocytes*, or

"large hyaline" leucocytes, have a large, finely-woven, kidney-shaped nucleus and blue cloudy cytoplasm containing numbers of fine red granules. They are the largest normal cells in the blood. Their origin is uncertain. They are slowly amœboid. (4) *Eosinophil polynuclear* leucocytes, with coarse, eosin-staining granules, coming from the bone-marrow, are amœboid, but not phagocytic. (5) *Basophil* leucocytes, or mast cells, with coarse basophil granules.

Leucocytosis and Leucopenia.—An increase in the total leucocytes (leucocytosis) above the normal number (4,000 to 11,000 per cu. mm.) is the commonest variation.

1. PHYSIOLOGICAL LEUCOCYTOSIS.—In the newly born the normal number is often over 15,000, and up to seven years of age 10,000 to 12,000 per cu. mm. After (1) exercise, (2) cold baths, (3) meals, there is an increase in the numbers of leucocytes, up to about 12,000 for a short time. (4) During late pregnancy there is usually also a moderate leucocytosis.

2. PATHOLOGICAL LEUCOCYTOSIS. (*a*) *Neutrophil polymorph* leucocytosis is the most frequent type. This occurs in (1) certain acute infections, such as septicæmia, pneumonia, appendicitis, erysipelas, scarlet fever, acute poliomyelitis, meningitis; (2) abscess formation, as in osteomyelitis, perinephric abscess, appendix abscess, empyema. The pus in acute abscesses is largely composed of degenerated polymorph leucocytes. In any of these conditions the absence of leucocytosis may indicate that the patient is failing to respond to the infection; this may be of grave prognostic significance. This is especially true if there is a diminution in the leucocytes, a leucopenia, instead of a leucocytosis. After the crisis in pneumonia, and after the efficient drainage of an abscess, the leucocytosis rapidly disappears. (3) During the absorption of blood, as in extensive bruises, and after blood transfusions. (4) After operations, especially those involving much trauma to the tissues, even in the absence of infections. (5) In acute gout and other intoxications, and drug poisoning. (6) In malignant disease especially when the liver and bone marrow are involved. (7) In certain blood diseases, for instance in the later stages of lymphadenoma, in polycythæmia, and myeloid leukæmia. In most of these conditions, especially in the infections, the neutrophil polymorph leucocytes have, on an average, fewer lobes in their nuclei than in health. The younger the cell, the fewer the number of lobes in the nucleus. This is known as a "shift to the left" in the nuclear index of Arneth or Schilling. An *artificial* leucocytosis may be stimulated by blood transfusions and by intramuscular injections of milk and nucleic acid preparations, and some believe this raises resistance to infections. (And see § 155*e*.)

(*b*) An increase in the *lymphocytes* (lymphocytosis) occurs in (1) lymphatic leukæmia, (2) whooping-cough, (3) tuberculosis (except tuberculous meningitis and sometimes miliary tuberculosis), (4) glandular fever and many other virus infections, (5) secondary syphilis, (6) during recovery from an acute infection (post-infective, or post-toxic lymphocytosis), (7) in subacute infections in infancy and childhood.

(*c*) An increase of *eosinophil* leucocytes (eosinophilia) occurs in (1) those suffering from parasitic diseases, *e.g.*, hydatids, trichinosis, filariasis, loa loa, bilharziasis and ankylostomiasis; (2) several skin diseases, notably dermatitis herpetiformis, prurigo, pemphigus and psoriasis; (3) in many cases of asthma and other allergic states, and in members of an allergic family who may not themselves have manifested an allergic condition. Two pulmonary conditions, which appear to have an allergic basis and which are associated with spasmodic bronchitis, leucocytosis and a very high eosinophilia are known as Loeffler's syndrome and Tropical eosinophilia (§ 127). (4) Occasionally in lymphadenoma; (5) after tuberculin injections; (6) during recovery from infections (post-infective or post-toxic eosinophilia); (7) during the absorption of a hæmorrhagic pleural effusion, and in cases of eosinophil pleural effusion; (8) often in myeloid leukæmia and sometimes in polycythæmia vera; (9) after taking certain drugs, *e.g.*, nirvanol and sulphonamides.

LOCAL ACCUMULATIONS of eosinophil cells occur (1) in the bronchial secretion of some cases of asthma, (2) in the conjunctival sac in one form of conjunctivitis, (3) in nasal secretions of allergic rhinitis, (4) in pleural effusions containing much blood,

and sometimes in simple post-pneumonic pleural effusions (eosinophil pleural effusions), (5) in the lesions of lymphadenoma in most cases.

(*d*) An increase of *monocytes* occurs in certain chronic infections, as in some stages of tuberculosis; and in malaria, kala-azar, glandular fever, trypanosomiasis, monocytic leukæmia and sarcoidosis.

(*e*) An increase of *basophil leucocytes* occurs in most cases of myeloid leukæmia and very rarely in other conditions.

(*f*) *Immature leucocytes* occur as (1) cells from the bone marrow, myelocytes, and their precursors, myeloblasts, in myeloid leukæmia. Myelocytes also occur in any condition in which there is an excessive call upon the bone marrow, as in severe infections with a high leucocytosis; in anæmia splenica infantum; and when the bone marrow is stimulated or irritated, *e.g.*, by malignant metastases in the bone, by osteosclerosis or by syphilis affecting the marrow (§ 542). (2) Lymphoblasts, the precursors of lymphocytes, occur in lymphatic leukæmia, especially the acute type.

Leucopenia, a decrease in the number of leucocytes, chiefly affects the neutrophil polymorph leucocytes, so that a relative lymphocytosis occurs. It occurs in (1) infection with the typhoid group of organisms, in undulant fever (due to *Brucella abortus* and *melitensis*), and in some chronic cases of malaria. An intense form occurs in Kala-azar. In many cases no actual decrease in the leucocytes occurs, but the absence of a leucocytosis in febrile conditions should arouse the suspicion of these infections. (2) In infections which are overwhelming, and in which the patient's resistance is small; (3) certain blood diseases, such as pernicious anæmia, splenic anæmia, aleukæmic leukæmia, especially in infants, aplastic anæmia, in agranulocytic angina (§ 155*e*), and in Gaucher's disease; (4) in poisoning with benzol compounds, sometimes with arsenic, antimony, mercury, lead, antipyrin, thiouracil and sulphonamide compounds, and after excessive exposure to radium or X-rays.

Melanæmia is a term applied when certain pigment granules occur in the blood after malaria, relapsing fever and some melanotic tumours. They appear either in minute black lumps, or are enclosed within the cells.

Lipæmia.—The blood plasma is laden with fat and of a pale pinkish colour in some cases of diabetes, and in subacute parenchymatous nephritis.

Blood Platelets.—The blood platelets are seen with a $\frac{1}{12}$-inch oil immersion lens as small irregular bodies, apt to run together in clumps. They are concerned with coagulation and are reduced in number in hæmorrhagic purpura. The normal number is 250,000 to 500,000 per cu.mm. To count platelets, rub the patient's finger with powdered potassium oxalate till a powdered effect is obtained; draw the blood with a needle-prick through the oxalate; make a blood film, and stain with Leishman's stain. A comparison with the number of red cells, together with a red cell count, makes it possible to determine the number of platelets present. Platelets are increased (1) after severe hæmorrhage, (2) after blood transfusion, (3) with chronic myeloid leukæmia, (4) after splenectomy and to a less extent after any severe operation, (5) in polycythæmia vera, and (6) in the rare condition of primary thrombocythæmia where the platelet count is exceedingly high and thrombosis and hæmorrhages are frequent. They are diminished in (1) pernicious anæmia, (2) idiopathic purpura hæmorrhagica, (3) scurvy, (4) aleukæmic leukæmia, (5) aplastic anæmia, (6) poisoning of the bone marrow by benzol or other drugs or (7) damage to the bone marrow by X-rays or radium.

§ 532. Parasites found in the Blood.—The detection of micro-organisms of clinical importance found in the blood is dealt with in Chapter XXI. *Bartonella bacilliformis* infects the red corpuscles of patients in Peru suffering from Oroya Fever. Certain pathogenic protozoa such as *malarial* parasites, *trypanosomes* and *Leishman-Donovan bodies* may be found in tropical patients, as well as filarial embryos; those of *Wüchereria bancrofti* are nocturnal, being found in the blood at night; those of *Loa loa*, from West Africa, which cause Calabar swellings, are found in the day time. In the relapsing fevers treponemota may be found during the pyrexial periods.

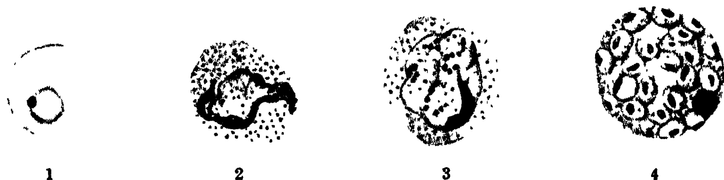
The Parasite of Malarial Fever is a protozoon, inhabiting the red corpuscles,

which it destroys, but if anti-malarial drugs are being táken, only sexual forms (gametocytes) are likely to be found.

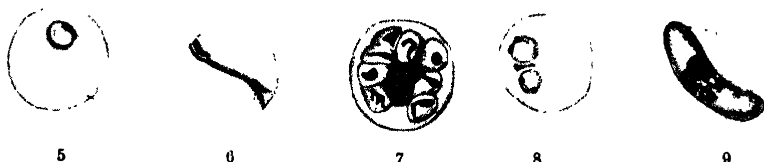
Staining.—Employ Leishman's method and examine with $\frac{1}{12}$th oil immersion lens. The cytoplasm of the parasites is stained bright blue, the chromatin pink, the granules are nearly black.

Varieties.—There are three well-marked species of the parasite, distinguished from each other by their intracorpuscular development, and these varieties correspond to the three types of malaria known as benign tertian, quartan, and malignant tertian fever. A fourth species of malaria parasite, *Plasmodium ovale*, has recently been recognised. In blood films it resembles the parasite of quartan malaria, but the fever is tertian, not quartan in type. The life-history of the protozoon runs through two stages: (i.) The asexual or intracorpuscular stage in man; and (ii.) the sexual form within the body of a mosquito belonging to the genus Anopheles (Fig. 120, § 510). The *benign tertian* parasite (Fig. 128) is first seen within the corpuscle as a small, clear, ovoid body about $2\,\mu$ in diameter, possessing active amœboid movement. It gradually increases in size, becomes vacuolated, and after the lapse of a few hours becomes ring-shaped (stage 1), with a peripheral chromatin dot. After about six hours the larger amœboid form develops (stage 2), containing one or two refractile granules of brown pigment, while the erythrocytes show enlargement, pallor and fine granules known as Schüffner's dots, staining red with Romanowsky stains. At the stage of full growth the parasite occupies nearly the whole of the enlarged corpuscle (stage 3), and now it may follow either of two lines of development: (i.) In the *asexual phase*, the pigment gathers in the centre of the parasite, the chromatin divides, and the protoplasm is arranged around these masses so that rosettes of from fifteen to twenty segments are formed (stage 4); these are set free as merozoites by the rupture of the red blood corpuscles containing them, and masses of insoluble pigment enter the blood-stream; this is subsequently phagocytosed by the reticulo-endothelial cells in the liver, spleen and bone marrow. This phase of "segmentation" is complete in about forty-eight hours, and corresponds clinically to a fresh paroxysm of the fever. These merozoites enter fresh red blood corpuscles and the cycle is repeated. (ii.) Some of the rings grow up but do not divide. These are the *sexual* gametocytes, which are

FIG. 128.—PARASITE OF MALARIAL FEVER SEEN IN PERIPHERAL BLOOD SMEARS.



1–4. STAGES OF BENIGN TERTIAN (P. VIVAX). (1) *Parasite with vacuole in centre and chromatin nucleus* (2) *Parasite developing into amœboid trophozoite: red cell enlarging and Schüffner's dots beginning to appear.* (3) *Trophozoite still increasing in size and beginning to divide (schizogony): Schüffner's dots more visible.* (4) *Divided schizont (forming merozoites) before rupture of red cell.*



5–7. STAGES OF QUARTAN MALARIA (P. MALARIÆ). *Similar stages to 1–4, but parasite remains more compact (sometimes assuming band forms—see 6): red cells do not enlarge and Schüffner's dots are absent.* 8–9. STAGES OF MALIGNANT TERTIAN (P. FALCIPARUM): (8) *Ring forms. The growing trophozoite and schizonts are not seen in the peripheral blood except in very severe infections.* (9) *The crescent is the gametocyte.*

of two types, male and female. When the blood is sucked into the mosquito's stomach all the asexual parasites perish, but the male gametocyte develops into a flagellated body, and one of the flagellæ unites with the female gametocyte. This fertilised gametocyte (the travelling vermicule) penetrates the wall of the stomach and comes to lie on its outer surface where it projects into the body cavity of the insect, and becoming spherical it develops within it (the oöcyst) a large number of curved, needle-shaped bodies (sporozoites). These are set free and reach the salivary gland of the mosquito from whence they are injected into the blood of the person bitten. They then find their way to cells of the reticulo-endothelial system and the cubical cells of the liver where they have an exo-erythrocytic cyclical stage, forming a reservoir of infection which later gives rise to invasion of the red cells (§ 510).

The *quartan* parasite, the easiest form for the beginner to study because of its visibility, first appears as a small, round, clear mass of cytoplasm which becomes vacuolated to form a ring difficult to distinguish from *P. vivax* (stage 5). It has feeble amœboid movement, develops slowly, and takes seventy-two hours to complete its cycle. After a few hours, coarse dark pigment granules appear, and a growth develops; the organism tends to be stretched as a band across the corpuscle (stage 6). By the third day pigment, coarser and blacker than that of the tertian form, gathers round its periphery. On the fourth day segmentation takes place, the pigment flows in towards the centre, and here forms the radiating lines which produce the beautiful "daisy rosette" so characteristic of the quartan parasite (stage 7). It breaks up eventually into eight to ten spores, and these with the insoluble pigment become free in the blood-stream. The development of the gametocyte resembles that of the benign tertian variety. There is no enlargement of the red corpuscle.

The parasite of the *malignant tertian* fever is first seen in the red blood-cells as a tiny, unpigmented, hyaline body, forty-eight hours being needed for its development. At first it exhibits energetic amœboid movements, but ultimately settles into a bright, colourless, ring-like form, with one or two chromatin dots (stage 8). There is frequently multiple infection of a single corpuscle. The rosette or sporulating stage is rarely seen in the peripheral blood. In about a week (during the period of remission) characteristic crescent bodies, containing masses of coarse pigment granules, begin to appear, and increase in number rapidly. They are incapable of sporulation, and represent the sexual form —the gametocyte—(stage 9), of which there are male and female forms. They ultimately degenerate if not taken up by the mosquito.



FIG. 129 —Thick dehæmoglobinised blood film showing microfilariæ of *Loa loa;* note sheath and the irregular and diffusely staining column of nuclei which extend almost to tip of tail. (× 400.)

Wüchereria bancrofti (Syn *Filaria bancrofti*) is transmitted by various species of anopheline, culicine or aëdine mosquitoes in which the embryos, sucked up with the blood, take ten to forty days to develop. The microfilariæ are readily seen in a fresh drop of blood in a cover-glass preparation examined under the low or medium power of the microscope; the detailed morphology may be studied in a thick film dehæmoglobinised and stained with hæmalum. The embryos may be found in 20 per cent. of apparently healthy residents in certain tropical countries. The embryos come into the peripheral blood at night (from 6 P.M. to 10 A.M.) and disappear during the day; the maximum number is usually found about midnight. It may be necessary to make repeated examinations at intervals of two hours to find them. Should a victim of the parasite alter his usual habits, and sleep during the day, the filaria periodicity is reversed. The adult filariæ inhabit the lymphatics, where they give birth to

immense numbers of embryos, a large number of which must be destroyed, or else the blood would contain them in incalculable numbers. In the Pacific Islands the mosquito transmitter is *Aëdes variegatus* which bites in the day time. No periodicity exists though the worm causing filariasis there appears identical with *W. bancrofti*. Adult parasites after their death may cause lymphangitis, various forms of elephantiasis, lymph-scrotum, hæmatochyluria, chylous diarrhœa and ascites, usually related to their blocking of the lymphatic circulation.

Loa loa.—Clinically, the adult worms produce puffy "Calabar swellings" in the subcutaneous tissues, and sometimes they migrate across the conjunctiva, giving rise to conjunctivitis. Generalised urticaria and asthma are occasionally produced. The sheathed embryos occur in the blood during the day time (diurnal periodicity) (Fig. 129), but may take several years to appear, and in such cases eosinophilia, a positive intradermal test, and a positive filarial complement fixation reaction enable the diagnosis to be made. Transmission is by a species of *Chrysops*, the mangrove-fly.

Trypanosoma.—The parasite of TRYPANOSOMIASIS (§ 518) is a flagellated protozoon. It is usually obtained by gland puncture, and can also be demonstrated in the blood and cerebro-spinal fluid. It is found free in the blood. One end of the parasite is drawn out into a whip-like process, the flagellum; the other end is bluntly conical; the body itself is short and thick, and its substance granular. It contains a trophonucleus and a kinetonucleus. Attached to one side is a transparent, flange-like process, the undulating membrane. The length of the parasite, including the flagellum, is about 18 μ to 25 μ. It is best stained by Leishman's or Giemsa's stain (Fig. 130).

The protozoa of KALA-AZAR are found in the spleen, liver, bone-marrow, the blood, and in the lymphatic glands. The commonest form found is a small ovoid body longer than it is broad, less than 2 μ in diameter, measuring about one-fifth of a red corpuscle in its longest axis. Stained with Leishman's stain the parasite shows two nuclei: one is small, rod-shaped, and stains deeply; the other is larger, rounded, and stains less deeply. Similar bodies are found in Oriental sore and in Espundia. Leishmania in culture elongate and develop a flagellum.

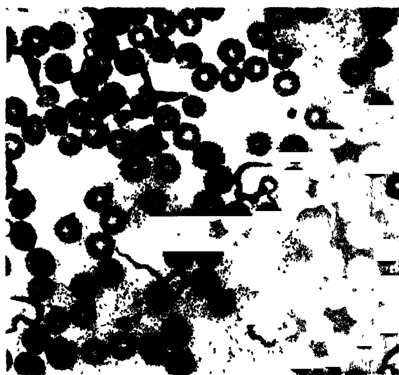


FIG. 130.—Trypanosoma rhodesiense in blood film. (× 480.)

RELAPSING FEVERS.—The *treponemata* (*spirochætes*) causing these fevers can be stained by the Leishman method in blood smears. Fine focussing is requisite, and the morphology varies considerably. In fresh blood their active motility renders them conspicuous against dark ground illumination. The treponemata are found in the blood during the pyrexial periods only.

§ 533. Physical and Chemical Properties of the Blood.—Biochemistry has made great strides within recent years. In Table XXXIII are some of the substances which can be estimated.

In every case the bio-chemist should either collect his own sample or indicate exactly how he wishes the sample to be collected, as different forms of technique are employed in different laboratories.

The FRAGILITY of red cells is tested by placing a drop of blood in salt solutions of strength varying by 0·05 per cent. and extending from 0·30 to 0·70 per cent. When the red cells have settled, a slight red tinge is seen above them in some tubes, whilst in others the red cells have completely hæmolysed. Hæmolysis is said to start in the tube in which the first red tinge is seen and to be complete in that in which the red cells are all hæmolysed. It is customary to test a normal person in a second set

TABLE XXXIII.

Substance.	Whole Blood.	Plasma or Serum.
Bicarbonate reserve as CO_2	— mgm. per 100 c.c.	53–77 vols. CO_2 per cent.
Calcium	5–7 ,, ,, ,, ,,	9–11 mgm. per 100 c.c.
Chlorides (as Sod. Chloride) ..	450–530 ,, ,, ,, ,,	560–620 ,, ,, ,, ,,
Cholesterol	100–200 ,, ,, ,, ,,	100–220 ,, ,, ,, ,,
Creatinine	1–2 ,, ,, ,, ,,	1–2 ,, ,, ,, ,,
Inorganic phosphate (as phosphorus)	—	(Adults, 2–4 mgm. per 100 c.c. Children, 4–6 mgm. per 100 c.c.)
Non-protein nitrogen	25–50 mgm. per 100 c.c.	18–30 mgm. per 100 c.c.
Phosphatase (acid)	—	0·5–5·0 units } King and Armstrong's Method.
,, (alkaline)	—	3·0–13·0 units } King and Armstrong's Method.
Potassium	150–250 mgm. per 100 c.c.	18–21 mgm. per 100 c.c.
Serum proteins	—	
albumen	—	3·4–6·7 G. per 100 c.c.
globulin	—	1·2–2·9 G. ,, ,, ,,
Sodium	170–225 mgm. per 100 c.c.	325–350 mgm. per 100 c.c.
Sugar (fasting)	80–120 ,, ,, ,, ,,	80–120 ,, ,, ,, ,,
Urea	20–40 ,, ,, ,, ,,	20–40 ,, ,, ,, ,,
Uric Acid	1–4 ,, ,, ,, ,,	1–4 ,, ,, ,, ,,

of tubes for comparison. Normally, hæmolysis commences in the 0·45 tube and is complete in the 0·30 tube. The fragility of the red cells is a diagnostic point in acholuric jaundice (§ 328). *Quantitative estimation* of hæmolysis gives more information than the simple qualitative test described, because it detects fragility of a small number of corpuscles in a sample in which most of the cells exhibit normal saline fragility.

SPECTROSCOPIC EXAMINATION of the blood (Plate IV).—The instrument chiefly used for clinical purposes is Browning's spectroscope. It is used by holding up a glass containing a very dilute solution of blood, and looking through it at the daylight, or at a white cloud, with a spectroscope placed between the blood solution and the eyes. Hæmatoporphyrin has been found in the urine in sulphonal poisoning (§ 382). Methæmoglobinæmia and sulphæmoglobinæmia are occasional complications of therapy with sulphonamide drugs. Methæmoglobin is also formed in nitro-benzol and potassium chlorate poisoning and in other conditions (§§ 32 and 382), and carboxyhæmoglobin in poisoning from coal gas, stoves, petrol fumes (§ 561).

BLOOD COAGULATION.—The clotting of blood occurs as follows: prothrombin reacts with the ferment thrombokinase (derived from blood platelets and tissue juices) in the presence of free calcium ions to form thrombin: thrombin interacts with plasma fibrinogen to form fibrin. THE CLOTTING TIME is estimated simply by Lee and White's method. 1 c.c. is taken from a vein by a *dry* needle and syringe and placed in a clean test tube 8 mm. in diameter. A rubber cork is inserted and at every ½ min. the tube is inverted till a firm clot is formed. The normal range is 4–7 minutes. The clotting time of shed blood is markedly prolonged in hæmophilia; definite prolongation also occurs in obstructive jaundice and acute toxic poisoning of the liver, and to a more variable degree in leukæmia, aplastic conditions of the bone marrow and in X-ray and radium overdosage. Coagulation time is shortened after hæmorrhage, splenectomy, a general anæsthetic and in many febrile conditions; and therapeutically by blood transfusion and by thromboplastin.

PROTHROMBIN CONTENT AND PROTHROMBIN INDEX.—Estimation of these is indicated in hæmorrhagic states, and especially in cases of obstructive jaundice prior to operation and in the hæmorrhagic diseases of the new-born—in both of which prothrombin is frequently deficient. The *Method of Estimation* is to mix optimal amounts of all substances required for clotting—thrombokinase or tissue extract, calcium and fibrinogen—with the exception of prothrombin. In the sample to be tested, oxalated blood is used and is recalcified at the time of the test with calcium chloride. The time from the moment of adding the latter to the time of occurrence of clotting is the *prothrombin time* (normal 15–25 secs., but somewhat variable with

different techniques): this is inversely proportional to the amount of prothrombin present. The *prothrombin index* is expressed as

$$\frac{\text{Prothrombin time of Normal Control}}{\text{Prothrombin time of Patient}} \times 100.$$

BLEEDING TIME.—This is independent of the clotting time, and is a measure of the power of the blood vessels to react to a wound or puncture. *Estimation* :—After puncturing the ear or finger in order to obtain blood, apply every thirty seconds a piece of blotting-paper to the drop, but do not touch the skin. Normally 2 to 5 minutes pass before the blood clots. The bleeding time is prolonged when platelets are few. Thus in essential thrombocytopenia the bleeding time increases from the normal 2 to 5 minutes to half an hour or longer. In hæmophilia the bleeding time is normal.

§ **534. A Biopsy of Sternal Bone Marrow** has now an important place in the diagnosis of obscure blood disorders. It may be carried out either by puncture of the sternum by a special thick needle, or by the removal of a disc of bone and marrow with a small trephine. In puncture, not more than 0·25 c.c. of marrow should be withdrawn and placed in an oxalate tube, and a total nucleated cell count carried out to give an idea of the cellularity of the marrow. Smears of marrow are also made on glass slides and sections of the fixed deposit (or of the actual disc of marrow if a trephine is used) are cut. A differential count of the various nucleated cells, both red and white, is also carried out. Results of diagnostic importance are obtained in leukæmia (especially aleukæmic leukæmia), Gaucher's disease and allied conditions, malignant disease in bone, multiple myeloma, aplastic anæmia, myelosclerosis, kala-azar, malaria and trypanosomiasis (and see § 919).

TABLE XXXIV.—DIFFERENTIAL COUNT OF NORMAL STERNAL MARROW FROM SMEARS (Percentages).

	Neutrophil.	*Eosinophil.*	*Basophil.*	
Polymorphonuclears	20-50	0-4	0-1	A
Metamyelocytes	2·5-12	0-2·5	—	A
Myelocytes	2-8	0-1		A
Premyelocytes		0·5-5		A
Myeloblasts		0-2·5		A
Lymphocytes		5-20		
Monocytes		0-5		
Plasma cells		0-1		
Late Normoblasts		7-19		B
Early and Intermediate Normoblasts		4-15		B
Pre-erythoblasts		0-4		B
Hæmocytoblasts		0-1		B

The proportion of cells of the myeloid series to nucleated red cells varies from 8 to 1 to 2 to 1 (A/B above). The total nucleated cell count, red and white cells, is from 20,000 to 100,000 per cu. mm

PART C. DISEASES WHICH GIVE RISE TO GENERAL DEBILITY, WITH OR WITHOUT ANÆMIA AND EMACIATION: THEIR DIAGNOSIS, PROGNOSIS, AND TREATMENT.

§ **535. Routine Procedure and Classification.**—Here, as elsewhere, we have three points to investigate:

First, the LEADING, and perhaps the only SYMPTOM complained of by the patient will be debility, or pallor of the skin, or loss of flesh.

Secondly, the HISTORY OF THE ILLNESS, its date, mode of onset and mode of evolution. Often these data are vague, but special enquiries should be directed to the condition of the digestion in times past, and any other points relating to nutrition.

Thirdly, the PHYSICAL EXAMINATION of the patient, commencing with that physiological system to which the results of our previous enquiries have directed attention, and then going through all the systems seriatim. An examination of the blood should be made in all anæmic or doubtful cases—viz., hæmoglobin, cell-counts, and films.

Classification.—If there is PALLOR OF THE SKIN and ANÆMIA is suspected, turn first to Group I, BELOW.

If LOSS OF FLESH is most prominent, turn to Group II, § 554.

If GENERAL DEBILITY (without obvious pallor or loss of flesh) is most prominent, turn to Group III, § 558.

GROUP I. PALLOR OF THE SKIN AND ANÆMIC DISORDERS

PALLOR OF THE SKIN may be due to (A) Anæmia, the colour of the skin being due to deficiency of blood in the capillaries. In other cases (B) the anæmia is slight and the pallor of the skin is due to a variety of causes, *e.g.*, syphilis, renal or hepatic disease. In every case an estimation of the hæmoglobin content of the blood (§ 528) must be made and in many cases a complete blood count is necessary.

The pallor presents to the experienced observer a difference in kind and degree in the several affections, and see § 10. Thus, the lemon-yellow of pernicious anæmia, the earthy tint of carcinoma, the sallowness of aortic disease and interstitial nephritis, the pasty white of parenchymatous nephritis, the greenish-yellow colour of chlorosis, and the transparent waxy look of lardaceous disease, are very suggestive to the careful observer. The microscopic examination of the blood also reveals differences which are mentioned below. The age of the patient often gives a valuable clue. For Fallacies, see § 525.

A. *The patient complains of* LACK OF ENERGY, SHORTNESS OF BREATH ON EXERTION *and the other symptoms of anæmia.* PALLOR *is marked and* THE BLOOD IS MARKEDLY DEFICIENT IN HÆMOGLOBIN. *The condition is one of* ANÆMIA.

Symptoms Common to all **Anæmias** are (1) failing strength for physical and mental work accompanied by pallor of the surface. The pallor is marked in the lips, the tongue, and conjunctivæ (as may be observed by pulling down the lower lid), and the sclerotics have a bluish colour. (2) Cardio-vascular symptoms, such as dyspnœa on slight exertion, palpitation, giddiness and fainting. The heart is dilated in many cases, with precordial pain. Hæmic murmurs are heard, especially over the pulmonary area. In marked cases the " bruit de diable " is present—a continuous hum heard when the stethoscope is gently placed over the jugular vein in the neck. Œdema of the ankles at night is common; venous thrombosis is rare. **Hæmic or Anæmic Murmurs** (§ 42) are usually soft and blowing, but may be loud and rasping; loudest in the pulmonary

area, but may be heard all over the precordium, rarely in the axilla; often louder when the patient is lying down or has rested, and apt to vary from day to day. (3) Disturbances of digestion—deficient or capricious appetite, discomfort or even vomiting after food; gastric atony and gastroptosis. Constipation is often present. (4) Symptoms referable to the nervous system—headache, neuralgia, tinnitus, vertigo, defective attention, nervousness, irritability or depression of spirits, spots before the eyes. Vasomotor signs are frequent; a tendency to "dead fingers," morbid flushing and variable temper. (5) Amenorrhœa is usual, dysmenorrhœa not infrequent; menorrhagia may lead to anæmia.

The CAUSES OF ANÆMIA may be:

(*a*) Hæmorrhage, either manifest or occult.

(*b*) Deficient formation or excessive destruction of red blood corpuscles.

(*a*) § **536. Hæmorrhage** is the commonest cause of pallor and anæmia and so is considered first. A patient rarely complains of symptoms until the hæmoglobin has fallen below 70 per cent. of normal, and many maintain fair health with much lower values. A sudden drop in hæmoglobin produces symptoms early; a slow drop is much less noticed by the patient.

Manifest hæmorrhage occurs with hæmatemesis, hæmoptysis, hæmaturia, epistaxis, menorrhagia, bleeding piles or melæna, or with surgical injuries. The history reveals the nature of the hæmorrhage, although in unobservant patients or in women with menorrhagia leading questions as to abnormal bleeding may be necessary. If the hæmorrhage is acute and severe, giddiness, sudden faintness or even collapse is present during the period of blood loss.

Occult hæmorrhage may be in such small repeated quantities as to be unnoticed, as when it occurs from a peptic ulcer or carcinoma of the stomach, when blood oozes in small amounts almost continuously. Small amounts of blood may be also lost in cases with internal piles or with ankylostomiasis; in these cases, the blood may be demonstrated by occult blood tests of the stools (§ 303). Sometimes hæmorrhage may remain hidden, when it occurs into the internal organs or serous cavities, *e.g.*, in a ruptured ectopic gestation or with suprarenal hæmorrhage in infants.

The hæmorrhage may be contributed to by an abnormal tendency to bleeding, such as occurs in the hæmorrhagic diseases: hæmophilia (§ 550), severe purpura, including thrombocytopenic purpura and Henoch's purpura (§ 584), in certain liver diseases (§ 333) and with hereditary capillary telangiectasis (§ 652. IV). In infants, scurvy and hæmorrhagic disease of the new born must be considered.

The *type of anæmia* resulting from hæmorrhage depends on whether the blood loss was sudden and large or frequent and small. In the first case the anæmia is usually normocytic and normochromic; in the second, as the iron stores in the body have probably been depleted by the long continued loss of blood, the blood picture is usually indistinguishable from that of a simple hypochromic anæmia (§ 540) and the red cells are microcytic and hypochromic.

Treatment consists in attacking the underlying cause. Blood transfusions may be necessary, and iron salts by mouth stimulate blood formation.

§ 537. **Blood Transfusion** is carried out for two main reasons: (1) Restoration of the oxygen-carrying capacity of the red blood corpuscles as in anæmia; (2) restoration of blood volume with any innocuous fluid which is well retained in the circulation (*e.g.*, plasma or serum)—as in shock. These two fundamental indications often overlap. Also (3) transfusion of fresh blood may be needed to furnish a patient with leucocytes—as in agranulocytosis, with platelets and clotting factors—as in essential thrombocytopenia and other hæmorrhagic diatheses, or with immune bodies (complement or specific antibodies)—as in infections.

INDICATIONS.—(1) In the anæmia of acute or chronic hæmorrhage replacement by blood should be considered imperative when the hæmoglobin falls to 40 per cent. —a critical level below which life is endangered. Fresh blood has a hæmostatic effect, and when hæmorrhage has been controlled convalescence is shortened. No anæmic patient should have a major surgical operation with a hæmoglobin level below 70 per cent. Post-operative repair of wounds is often delayed when the hæmoglobin remains below this figure. (2) In "shock" when the diminished blood volume is wholly or mainly due to loss of blood, the blood volume should be restored by transfusion of blood to maintain a blood pressure above 100 mm. Hg. No patient suffering from "shock" should be operated upon with a blood pressure much below this figure. (3) During a severe acute or a chronic infection, with a hæmoglobin value below 65 per cent., transfusion aids the patient's resistance by combating anæmia and if fresh blood is used by supplying human complement and other immune bodies. Examples are septicæmia, ulcerative colitis and rheumatoid arthritis. (4) Hæmorrhagic states and blood diseases. For hæmophilia transfusion of fresh blood is a specific, though temporary, remedy. In an adolescent with prolonged coagulation time, 300–400 c.c. is an essential pre-operative safeguard, and has a hæmostatic effect. In essential thrombocytopenia by raising the number of platelets above the critical level, dangerous hæmorrhage may be controlled. The hæmolytic anæmia of Lederer and an allied type seen in pregnancy respond, although pregnancy may have to be terminated. In severe Addisonian anæmia, transfusion will tide the patient over the first few days until liver therapy takes effect. For aplastic anæmia it is the main treatment, although of transient value only; in the chronic leukæmias, especially the myeloid variety, and in lymphadenoma, it prolongs life by combating anæmia and allowing effective irradiation. (5) Poisoning by substances which restrict the formation of oxyhæmoglobin, *e.g.*, in coal-gas poisoning. After venesection, a large transfusion of healthy blood may save life by increasing the available oxyhæmoglobin.

Relative Merits of Fresh and Stored Blood.—Blood collected with sterile precautions by the method described below and kept refrigerated at 2°–4° C. is of recognised value for raising the hæmoglobin level and for restoring blood volume up to three weeks from the date of collection. After a few days, blood rapidly loses its functional leucocyte content, platelets and other clotting factors, and its immune bodies. Fresh blood (less than 24 hours old) is always preferable for patients with a blood disease (where maximum durability of the transfused red cells is desired), a hæmorrhagic disease or with continuing hæmorrhage (where clotting factors are required) and for anæmia associated with sepsis (where immune bodies may be valuable).

Blood Grouping.—Landsteiner in 1901 divided human beings into three groups on the basis of the agglutination reactions of their red cells and serum. A fourth and rarer group was added by von Decastello and Sturli in 1902. Jansky in 1907 made the first numerical classification of the four blood groups, numbering them 1, 2, 3 and 4, and the independent publication of Moss in America in 1910 extended such a numerical scheme. In his terminology, Groups 1 and 4 were reversed as

compared with that of Jansky—a situation which subsequently caused much confusion. To obviate the risks attendant on this, the official International nomenclature, which also is the most helpful in visualising the phenomena of iso-agglutination, was introduced. In it, as originally postulated by Landsteiner, the four blood groups are assumed to be dependent upon the presence or absence of two agglutinogens A and B in the red blood-cells and two agglutinins α (or anti-A) and β (or anti-B) in the serum or plasma. Interaction between an agglutinogen and its corresponding agglutinin in serum or plasma (*e.g.*, Group A cells with the α agglutinin) results in clumping of the red cells or agglutination. Therefore an agglutinogen and its corresponding agglutinin cannot exist together in the same blood. When Group O blood is given to a patient other than Group O, the α and β agglutinins in the donor's blood are so diluted in the recipient's circulation that their final concentration is too weak for any significant reaction to take place: exception occurs rarely with some Group O donors in whom the α and β agglutinin titre is very high. The accompanying table explains the three nomenclatures:

TABLE XXXV.—BLOOD GROUPS AND BLOOD GROUPING.

Groups. International Nomenclature.	*Agglutinogens in Red Cells.*	*Agglutinins in Serum.*	*Jansky Numbering.*	*Moss Numbering.*	*Donor can give Blood to*	*Recipient can receive Blood from*	*Percentage Incidence of Groups (Europe).*
AB	A and B	Neither	4	1	AB only	All groups (Universal recipient)	4
A	A	β (anti-B)	2	2	A and AB	A and O	42
B	B	α (anti-A)	3	3	B and AB	B and O	9
O	Neither	α and β	1	4	All groups (Universal donor)	O only	45

Technique of Grouping.—Stock high-titre anti-A and anti-B sera derived from a Group B and a Group A donor, respectively, are required. Place a small drop of each on an opal glass tile or microscope slide with a separate pipette and add a similar-sized drop of a weak suspension (approximately 3 per cent.) in normal saline of the blood to be grouped. Rock the tile gently to mix the red cell suspension and the sera and then leave to stand. After 10 to 15 minutes agitate the tile more strongly. Agglutination is indicated by the homogeneous red mixture taking on a fine or coarse brick-dust appearance which can be easily seen with the naked eye—in case of doubt use a hand lens. The determination of group is as follows:

TABLE XXXVI.

Anti-B serum from Group A Individual (containing the α Agglutinin).	Anti-A serum from Group B Individual (containing the α Agglutinin).		Group of Blood Tested (Agglutinogens in Cells).
+	+	=	AB
O	+	=	A
+	O	=	B
O	O	=	O

(+ = agglutination)

Confirmation can be obtained by testing the serum of the unknown blood for agglutinins with suspensions of known Group A and Group B red cells using the above technique.

The Rh. agglutinogen was discovered in 1940 by Landsteiner and Weiner, who found that the serum of a rabbit which had been inoculated with the red cells of a rhesus monkey agglutinated the red cells of 85 per cent. of American white subjects. This indicated that they possessed a hitherto unrecognised agglutinogen—termed the Rh. agglutinogen, because of its presence in the blood of the rhesus monkey—such

people are Rh.-positive. The important point is that the remaining 15 per cent. of subjects whose red cells are unagglutinated, and therefore lack the agglutinogen (Rh.-negative), are liable to form an antibody—Rh. agglutinin against the Rh. agglutinogen if it is introduced into their circulation. This may occur in an Rh.-negative woman if she becomes pregnant with a fœtus whose blood-cells are Rh.-positive (derived from the father), or in Rh.-negative persons of either sex should they be transfused with Rh.-positive blood, particularly if this is repeated. The possible formation of anti-Rh. agglutinins by an Rh.-negative person, whether as a result of pregnancy or of transfusion, is of great significance since a subsequent transfusion of Rh.-positive blood may lead to a hæmolytic reaction of varying degree, which sometimes proves fatal. In 90 per cent. of cases in which an infant is afflicted with hæmolytic disease of the new-born (manifested by icterus gravis, hæmolytic anæmia or hydrops fœtalis with intra-uterine death), the mother is found to be Rh.-negative and the infant Rh.-positive. In such cases the mother produces an anti-Rh. agglutinin during pregnancy which passes into the fœtal circulation across the placenta and destroys the fœtal erythrocytes and damages other tissue-cells.

Test serum for the Rh. agglutinogen is usually derived from such mothers: Add a drop of a weak saline-suspension of the red cells to be tested to a similar-sized drop of the test serum in a small tube and incubate for 2 hours at 37° C. The red cell sediment is transferred to a slide with a pipette and examined under the low power of the microscope for agglutination—if present the cells are Rh.-positive; if absent, Rh.-negative. Rhesus grouping prior to blood transfusion should be carried out on (1) patients of either sex who are likely to require multiple transfusions or who have had a transfusion more than seven days previously; (2) all young females; (3) all mothers giving a history of repeated still-births or jaundiced or anæmic babies. The necessity for such grouping will be appreciated on referring to the indications for Rh.-negative blood (*infra*). It is now known that the Rh. agglutinogen is very complex and that at least six different elementary antigens, for all of which specific agglutinins have been identified, are involved. Various combinations of these elementary component antigens occur so that a large number of different Rh. sub-types are possible. An individual under a suitable stimulus (pregnancy or transfusion) may form an anti-Rh. agglutinin to a particular elementary antigen which he lacks in his own red cells. The property "Rh.-negative" is now recognised as meaning not merely the absence of the Rh.-positive factor but implies the presence of particular elementary antigens. For further details special text-books must be consulted.

Choice of Donor.—The donor should be a healthy subject of either sex with good arm veins. Age does not matter within wide limits. Syphilis, malaria and infective hepatitis are transmissible diseases that must be excluded in whole-blood transfusion though infective hepatitis is more related to plasma and serum transfusions. The usual safeguard against syphilis is the Wassermann or related serological reaction, but it must be appreciated that in the late primary or early secondary stages, which are the most infectious periods, this test is occasionally negative or doubtful. It is safe to use a donor with a past history of malaria provided he has (in the absence of treatment) had no febrile bout attributable to the infection for three years, nor resided during this period in a malarial district. Individuals who have had infective jaundice during the previous twelve months and allergic subjects are best avoided. Processing serum and plasma eliminates the malarial parasite and the spirochæta pallida by the Seitz filtration and the subsequent drying, but the icterogenic virus survives these procedures. The donor should be of the same ABO blood group as the patient; the use of Group O as "universal donor" blood for recipients other than Group O, although warrantable under emergency conditions, carries some risk of damage to the recipient's red cells, particularly in large volume transfusions, when the agglutinin titre in the transfused plasma is high. In addition to routine grouping, a direct matching test for compatibility between the patient's serum and a weak suspension in normal saline of the donor's red cells should always

be carried out. The tile method as described for grouping may be employed but will not detect Rh. incompatibility.

INDICATIONS FOR RH.-NEGATIVE BLOOD.—As a counsel of perfection blood from an Rh.-negative donor should always be used for an Rh.-negative patient. Since the supply of such blood does not render this practicable it is recommended that Rh.-negative blood (of appropriate ABO group) be used for—

1. Rh.-negative patients of either sex who are likely to require multiple transfusions or who have had a previous transfusion even some years previously. Repeated transfusions of an Rh.-negative recipient with Rh.-positive blood may induce sensitisation with the production of Rh. antibody (anti-Rh. agglutinin) and risk of incompatible reaction.
2. Rh.-negative young females. Sensitisation of an Rh.-negative woman by transfusion of Rh.-positive blood may cause even her first child to suffer from hæmolytic disease should she subsequently marry an Rh.-positive male.
3. Mothers of infants with hæmolytic disease. In 90 per cent. of such cases the child is Rh.-positive and the mother Rh.-negative with Rh. antibody in her circulation.
4. Infants with hæmolytic disease. These are usually Rh.-positive and immediately after birth may possess in their circulation much Rh. antibody derived from the mother. On this account, transfusion with Rh.-negative blood is likely to be much more durable than Rh.-positive. Although probably Rh.-negative, the mother's whole blood is unsuitable owing to the Rh. antibody in her plasma. A suspension of her red cells in saline after removal of the plasma would be satisfactory.

For Rh.-positive patients (85 per cent. of British people) the Rh. group need not affect the choice of a donor since sensitisation even if given Rh.-negative blood is extremely improbable. It is also unnecessary to pay attention to the M and N factors —two other red-cell agglutinogens—in the routine selection of a donor since naturally-occurring agglutinins for them are extremely rare and when they do occur are usually of low titre and active mainly at low temperatures.

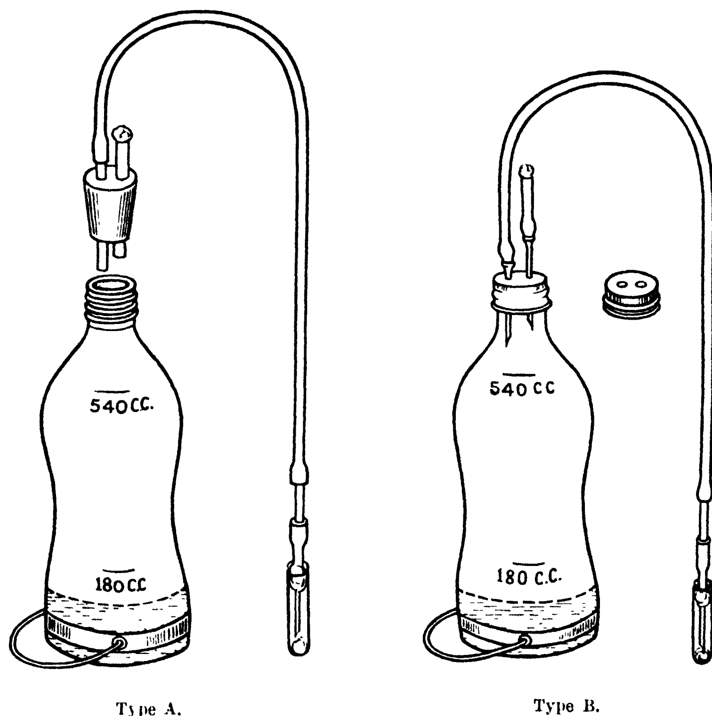
COLLECTION AND ADMINISTRATION OF BLOOD.—The Medical Research Council Blood Transfusion outfit is now standard in this country and has almost entirely replaced older methods. The bottle, slightly waisted for holding, is fitted with an aluminium screw cap bearing a 4-mm. rubber wad and is provided with a metal band and loop at the base for hanging it when inverted. The bottle is marked 540 c.c. and 180 c.c., the latter because the volume of anti-coagulant originally used was 180 c.c. The anti-coagulant now employed for the standard volume of blood collected (420 c.c.) is 120 c.c. of a solution in freshly distilled water of disodium hydrogen citrate (2 per cent.) and glucose (2·5 per cent.). This is satisfactory for fresh or stored blood and permits satisfactory storage for a period of three weeks from the date of collection. Glucose preserves the red cells in stored blood and the disodium salt is better than the trisodium salt previously used. For fresh blood transfusion the anti-coagulant may consist of 120 c.c. of tribasic sodium citrate (2 per cent.).

Collection of Blood from a Donor.—Two patterns of apparatus are in common use, depending on whether the cap of the collection bottle is replaced by a rubber bung and glass tubing (type A) or perforated by two needles (type B).

Type A (Fig. 131).—The screw cap is removed and placed between the folds of a sterile towel. The bottle is then fitted with a sterile " taking set " comprising a rubber bung pierced by two 7·5-cm. glass tubes, one of which is lightly plugged with cotton-wool to serve as an air outlet. To the other, which projects slightly further through the bung, is attached about 35 cm. of rubber tubing and a sharp stainless-steel needle (24/10 mm. diameter × 35 mm. long) protected by a small glass test-tube. A short length of glass tubing about 5 cm. above the needle serves as a window.

The donor lies flat on a bed or couch, with his head supported by a pillow, and the elbow resting on a firm cushion. A sphygmomanometer cuff or a tourniquet is applied to the upper arm to produce venous stasis and the skin over the selected

antecubital vein cleaned with ether, ether soap or 1 in 1,000 phenyl mercuric acetate in 70 per cent. spirit. Approximately 0·1 c.c. of 2 per cent. procaine hydrochlor. is injected intradermally over the selected vein. The needle of the taking set is uncovered, inserted into the vein and held in position by elastoplast placed over its shoulder whilst the bottle (which should be gently shaken) fills. A steady flow of blood is encouraged by giving the donor a suitable object in the hand on the same side on which to open and close his fingers. When the bottle has filled to the 540-c.c. mark, release the tourniquet and withdraw the needle from the vein; the blood remaining in the rubber tube is run into a sample tube for laboratory tests. Remove



Type A. Type B.

FIG. 131 Blood Collecting Apparatus.

the rubber bung with its tubing and re-apply the aluminium cap with sterile precautions. Lastly, invert the bottle to ensure adequate mixing of the contents.

Type B is similar to type A, but the rubber bung is replaced by an aluminium cap perforated by two holes 3 mm. in diameter. After autoclaving, the holes are sealed by a viscose cap or a strip of adhesive tape. Immediately before collecting the blood, the seal is removed, two needles are inserted into the bottle (Fig. 131) by perforating the rubber which shows through the holes in the aluminium cap: one serves as an air outlet. After completion of bleeding the needles are withdrawn and the holes resealed by fresh adhesive tape.

Administration of Blood to a Patient.—The standard administering apparatus (Fig. 132) consists of a rubber bung pierced by two pieces of glass tubing. One

25 cm. long reaches almost to the bottom of the bottle and serves as an air inlet; the outer orifice is fitted with a small cork. The other tube, 6·5 cm. long, has attached in sequence to its outer end 15 cm. of rubber tubing, a drip-feed bulb, and 90 cm. of rubber tubing terminating in a sharp stainless-steel narrow-bore needle (15/10 mm. diameter × 35 mm. long). The last 5 cm. of tubing attached to the needle is detachable by means of an adaptor fitting. A short distance above this is a screw clip by which the rate of flow can be controlled. A metal cannula can be attached directly to the male adaptor after removal of the needle and its tubing when it is necessary to cut down on the vein.

In order to prevent clots entering the delivery tubes, a gas-mantle filter or a close-meshed stainless-steel gauze filter, 6·5 cm. in length and 1·5 cm. in diameter, protects the inner end of the short outlet tube in the rubber bung.

Before giving a bottle of stored blood it should be inspected on removal from the refrigerator. If hæmolysis is evident in the zone of plasma immediately above the sedimented red cells, or if the blood exhibits a pigment change (*e.g.*, violet-permanganate colour) it should not be used.

Next invert the bottle two or three times to secure even mixture of the contents. Then warm the blood by placing it for 20–30 minutes in water at 37° C. (overheating must be avoided as it causes hæmolysis). The aluminium cap is next removed and the rubber bung of the giving unit inserted firmly into the mouth of the bottle. The screw clip on the rubber tubing is adjusted to close its lumen and the bottle hung up by the metal band and loop so that the apparatus is suspended vertically above the vein to be used. Disconnect the needle and short piece of rubber tubing from the rest of the giving unit at the adaptor fitting and place them within a sterile towel. Remove the cork from the air inlet tube and unscrew the screw clip to allow blood to flow and expel all the air. Next clamp the tubing by artery forceps or by the screw clip. A level in the drip bulb is established by tilting it up momentarily whilst the blood is flowing through it.

A vein in the antecubital fossa or in the forearm is usually chosen for the transfusion, the elbow being fixed with a back splint if the patient is restless. Having prepared the vein as described above for the donor, the needle with the short piece of rubber tubing attached is inserted into the vein, a satisfactory insertion being indicated by a free efflux of blood from the female adaptor. As soon as this occurs the pressure round the upper arm is released and the male adaptor on the suspended unit joined to the female adaptor. The rate of flow is adjusted by means of the screw clip. The needle and its tubing is secured to the skin of the patient's arm by strapping.

With the fine needle employed a local anæsthetic is not essential. To give subsequent bottles of blood, the same administering unit is used again. Reconstituted serum or plasma is administered by the same method. A record must always be made in the patient's notes of the serial number of stored blood or batch number of plasma or serum. Such a record permits investigation of any reaction and the recognition of an icterogenic batch of blood products.

Dosage and Rate of Flow.—Dosage must be decided for each case. In children it is higher in proportion to body weight than in adults and for them 15 c.c. per kilogram (2¼ lb.) body weight is an approximate guide. The anticipated rise in hæmoglobin after the transfusion of one bottle of whole blood (with the usual proportion of anti-coagulant solution) is approximately 8 per cent. When only one or two bottles of blood are to be given the blood (or plasma or serum) can be run in at a fast drip rate—one bottle per half-hour or less—in the absence of cardiac or circulatory embarrassment. With volumes above this, the rate of flow should be reduced to 40 drops per minute through the drip bulb (approximately one bottle per 4 hours).

Choice of Vein.—An antecubital vein is usually the most prominent. With prolonged transfusions, a forearm vein allows the patient greater freedom of movement at the elbow. In infants and young children, and in some adults, the internal saphenous vein in front of the internal melleolus is recommended, but a cannula is

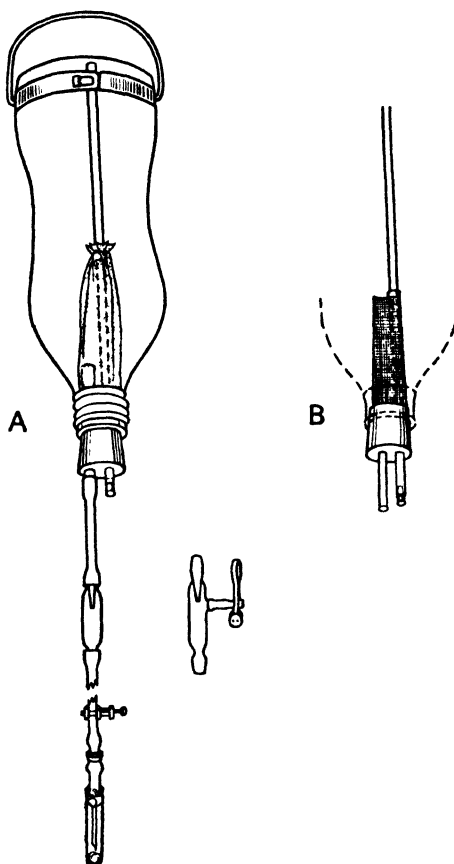


FIG 132 —Standard Blood Administration Apparatus.
(A) With cotton mantle filter (B) With metal gauze.

usually necessary. In very young infants a scalp vein and a No. 14 record needle may be employed.

Intramedullary administration of fluids into the marrow cavity is used where no surface veins are available, *e.g.*, extensive burns or scalds. In adults and in children over two years the manubrium sterni is the most convenient site: in infants the antero-medial surface of the tibia at the level of the tibial tubercle may be used although the risk of sepsis here appears greater. A Salah sternal puncture needle with winged guard is inserted as for a diagnostic sternal puncture (it may be used through burns or scalded areas): after removal of the stilette connect the needle with the male adaptor of the giving unit, prepared as for an ordinary intravenous transfusion. Gravity usually suffices for a satisfactory flow; transfusion into the tibial marrow tends to be slow at first but subsequently to increase in rate.

HUMAN PLASMA AND SERUM are only stored in the dried state since the liquids prove unstable with storage. Serum is derived from clotted blood and contains no fibrinogen (protein content approximately 7 per cent.). Plasma is obtained from citrated blood, and although possessing fibrinogen has a lower protein content (4–5 per cent.) owing to the added diluent. In their preparation the serum or

plasma of various blood groups is pooled; the resulting low agglutinin content permits safe usage for a patient of any group.

Reconstitution.—The standard issue is of dried solids from 400 c.c. of normal human serum or citrated plasma contained in a M.R.C. bottle. This is redissolved in the equivalent amount of sterile distilled water (supplied in another bottle) immediately before transfusion. Solution is aided by shaking the bottle and by warming to 37° C. The reconstituted fluid appears turbid. By diminishing the volume of distilled water used the protein concentration can be correspondingly raised. The risk of bacterial contamination necessitates that reconstitution should be carried out only just before administration. Dried serum and plasma are extremely stable and will keep indefinitely in a dark cool atmosphere; refrigeration is not required.

Indications.—Reconstituted serum and plasma contain a high content of physiological protein and possess valuable powers of restoring and maintaining blood volume, being infinitely preferable to normal saline. Allowing for the difference in protein content, clinical experience has shown that there is little to choose between the therapeutic results and reaction rate of serum and plasma. The main indication is in the treatment of shock where blood loss is not significant and especially when hæmo-concentration is present—*e.g.*, with extensive surgical procedures or with multiple injuries in which but little hæmorrhage has occurred: with crush injuries: or with burns or scalds. In all these conditions there is a lowered blood pressure accompanied in the latter two groups by hæmo-concentration. The aim in plasma or serum transfusion in these states is, as with blood given to combat shock dependent on hæmorrhage, to raise the blood pressure and to maintain it at about 100 mm. Hg, sufficient amount being administered to achieve this. When large volumes are necessary, dilution of the patient's red cells (hæmoglobin below 70 per cent.) is prevented by giving whole blood. A ratio of two bottles of plasma or serum to one of blood controlled by repeated blood pressure and hæmoglobin estimations is satisfactory. Concentrated plasma or serum (× 2 or × 3) is claimed by some to give better results in shock due to burns or scalds, since these patients are particularly liable to develop hypoproteinæmia: also the increased protein content of the transfused fluid tends to reduce local œdema in the injured area. Reactions are, however, more common with these concentrated protein solutions.

Treatment of the reduced plasma protein in patients with nephrosis or nephrotic nephritis by the transfusion of such solutions is only of temporary benefit. When blood is indicated, but not immediately available, or should it be necessary to wait for the results of Rh. grouping, plasma or serum can be given meanwhile.

Concentrated Red Cell Suspensions ("Packed Cells") are prepared by taking two M.R.C. bottles of whole citrated blood of the same group in which maximum sedimentation of the red cells has occurred (usually 5 to 6 days after collection). After syphoning off the supernatant plasma in each the red cell deposits are mixed in one bottle. Because of the slight risk of infection during preparation and because the removal of the plasma and of the glucose (derived from the anti-coagulant) makes the red cells less durable, the suspension must be used within 24 hours. Concentrated red-cell suspension is particularly useful for raising the hæmoglobin and red cell count rapidly with minimal increase in blood volume, in the treatment of anæmia (*a*) prior to operation, (*b*) with an enfeebled myocardium, (*c*) occurring with nephritic œdema, and (*d*) aplastic anæmia. The anticipated hæmoglobin increase per bottle in an adult is 15 per cent., and the reaction incidence is probably lower than with whole blood, serum or plasma. This suspension possesses poor volume-restoring power.

Transfusion Reactions and Complications.—Reactions may be classified as febrile, hæmolytic, or allergic.

Simple febrile reactions occur in 5–15 per cent. of transfusions. They are most commonly due to foreign protein, dead bacteria—or living but non-pathogenic organisms derived from unclean apparatus, improperly prepared or stale distilled

water used in the anti-coagulant solution, or from delay in autoclaving this after preparation. Infected stored blood is also a cause. The common febrile (non-hæmolytic) reaction usually occurs towards the end of or shortly after a transfusion, and if severe may be accompanied by a rigor: it is best controlled by the hypodermic injection of morphia gr. $\frac{1}{6}$–$\frac{1}{3}$ and liq. adrenalin hydrochlor. B.P., ♏ 10–15, the doses varying with the size of the patient and the degree of reaction. The patient should be kept warm with hot-water bottles and half an ounce of brandy is often beneficial. Rigors and severe febrile reactions are further reduced in incidence by slowing the rate of transfusion.

Hæmolytic reactions are of two kinds: those due to specific incompatibility between the bloods of recipient and donor and those due to non-specific causes such as the use of overheated or time-expired blood. The former should be excluded by careful grouping and by the additional safeguard of a direct matching test. Generally speaking an ABO incompatibility produces a more acute and violent reaction than one due to the Rh. factor. The *symptoms* include pain in the loin (always a danger signal), rigor, respiratory embarrassment, circulatory collapse, hæmoglobinuria, urobilinuria, jaundice, urticaria, and symptoms due to small hæmorrhages or emboli in the brain, mesentery, myocardium and gastro-intestinal mucosa. The terminal stages of those who survive the immediate " hæmolytic shock " are characterised by renal failure, with oliguria and a high blood urea. With a rhesus incompatibility the symptoms are usually (but not invariably) more silent and jaundice appearing twelve hours later may be the first indication.

Simple mechanical obstruction of the renal tubules by blood pigment from hæmolysed red cells is probably not the significant factor producing renal failure. In consequence, alkalinisation of the urine is now regarded as of doubtful therapeutic value. The lesion is one of acute renal damage, caused by arterial spasm and ischæmia, and treatment is planned on the basis that kidneys so damaged require time for regeneration and recovery. Therapeutic principles are:

(*a*) Phase of hæmolytic shock. During this initial period an adequate amount of certainly compatible blood—(alternatively serum or plasma)—should be given immediately to combat the fall in blood pressure.

(*b*) Phase of renal insufficiency which lasts 8–12 days. The fluid intake should be limited to that excreted. The damaged kidneys are unable to excrete large amounts of water and salts, or to be stimulated by drugs, during this oliguric period and attempts to flush them with diuretics are to be deprecated. An adequate dietary intake including Vitamins B and C should be ensured and severe acidosis (detected by alkali reserve estimations) combated.

(*c*) Phase of recovery or diuresis, which ultimately occurs unless renal insufficiency culminates in uræmia. The main emphasis now must be to replace water and salt lost in the urine and to avoid dehydration.

Mild allergic manifestations—e.g., urticaria, localised œdema—are often seen after transfusion of blood or blood products, and respond to adrenalin, benadryl or antistin. Grossly infected blood may produce a clinical state simulating severe protein shock.

Complications are (1) Circulatory, with cardiac failure and pulmonary œdema dependent on massive or too rapid transfusion. In an anæmic patient with a poor myocardium the risk of heart failure due to overloading the circulation demands careful consideration of volume and rate of administration. (2) Transmittance of disease from donor to recipient is referable mainly to syphilis, malaria and homologous serum hepatitis. Risk of the former in this country is small since the incidence of the disease is very low and spirochætes rapidly perish in citrated blood stored in a refrigerator. The hazard of malarial transmission applies to fresh and stored blood as the parasites can probably survive refrigeration for several weeks. Careful choice of the donor is the important safeguard. Homologous serum hepatitis—so-called because it is much more frequent after giving serum or plasma than whole blood—depends on infection of the recipient by a hepatitis-producing agent, probably a

virus, derived from apparently normal donors. It is now the most important disease transmitted by transfusion. The original incidence of 5–10 per cent. in patients receiving plasma or serum transfusions has been reduced by diminishing the size of the pool (maximum six to eight donors) from which these blood products are processed. The condition has an incubation period of 60–90 days after transfusion – an interval which may mask the association between the jaundice and the previous plasma or serum administration unless the possibility is borne in mind. Although usually mild, homologous hepatitis may be severe and rarely fatal from extensive hepatic necrosis (§ 333). Recognition and elimination of icterogenic batches of plasma and serum depend on carefully recording the batch number of the product used at the time of transfusion.

Infusion of Isotonic Saline Solution or dextrose saline may be indicated in shock or dehydration (water and salt depletion) and in certain toxic conditions—*e.g.*, uræmia, puerperal eclampsia, diabetic coma, or poisoning by carbolic acid, strychnine or phosphorus. Isotonic saline is 0·9 per cent. NaCl in distilled water, but isotonic Ringer-Locke solution is preferable owing to its balanced salt content. 4 per cent. dextrose and 0·18 per cent. NaCl (one part of normal saline to four parts of 5 per cent. dextrose in distilled water) is isotonic and is very satisfactory for the maintenance of water and salt balance after severe surgical operations. Routes of administration are: (1) Rectal—1 pint in 4 to 6 hours by a slow continuous drip (60 drops per minute using a No. 6 rubber catheter). Half or quarter strengths of saline (0·45 or 0·22 per cent. NaCl) are suitable for rectal injection. (2) Subcutaneous—in the loose cellular tissues below the clavicle, around the breasts or of the anterior abdominal wall or the thighs. Normal saline is given at the rate of 250 c.c. in 4 to 6 hours. (3) Intravenous—normal saline, 5 per cent. dextrose, or 0·18 per cent. NaCl and 4 per cent. dextrose, may be given rapidly, *e.g.*, 1 pint in 15–20 minutes, or by a slow continuous drip 4–6 pints may be given in 24 hours. Intravenous saline therapy for the restoration of blood volume in cases of shock when the plasma proteins have been depleted by hæmorrhage or local plasma loss may be dangerous and has to be employed with great care. The normal amount of water cannot be retained in the circulation on account of the reduction in plasma protein with a risk of pulmonary œdema; plasma, serum or blood are preferable.

(*b*) § **538.** *The patient is* ANÆMIC, *but* HÆMORRHAGE HAS BEEN EXCLUDED *as the cause of the anæmia. There must be* PRIMARY DISEASE OF THE BLOOD FORMING ORGANS, *with either* DEFICIENT FORMATION OR EXCESSIVE DESTRUCTION *of the hæmoglobin and red blood cells.* The causes are:—

Commoner.

I. Pernicious anæmia (§ 539).
II. Simple hypochromic anæmia (§ 540).
III. *Associated with acute and chronic septic or toxic processes* (§ 541).
Acute: especially Septicæmia (§ 515).
Malignant endocarditis (§ 50).
Acute rheumatic carditis (§ 582).
Chronic: especially Focal sepsis, Suppuration, and Tuberculosis (§ 541).
Lardaceous disease (§ 404).

Rarer.

I. Aplastic anæmia (§ 542).
II. Lederer's anæmia (§ 542).
III. Chlorosis (§ 542).

With enlarged spleen and/or lymph glands.

IV. Leukæmia (§ 543).
V. Lymphadenoma (§ 572).
VI. Acholuric jaundice (§ 328).
VII. Splenic anæmia (§ 544).
VIII. Marble bones and Myelosclerosis
IX. Gaucher's disease (§ 544).

With spongy gums, tender limbs and a hæmorrhagic tendency.

X. Scurvy (§ 545).

Commoner.	*Rarer.*
IV. *With marked emaciation.* Carcinoma (§ 542). Leuco-erythroblastic anæmia (§ 542).	*With history of residence abroad.*
	XI. Malaria, sprue, tropical nutritional anæmia and other tropical and parasitic diseases (§ 546).
	XII. Ankylostomiasis (§ 547).
	XIII. Sickle cell anæmia (§ 548).
	XIV. *With history of exposure to X-rays, radium or industrial poisons* (§ 549).
	XV. *With a history of recurrent severe bleeding after trivial injuries.* Hæmophilia (§ 550).
	XVI. *With a history of hæmoglobin in the urine.* Paroxysmal or Nocturnal hæmoglobinuria (§ 409).

For the Anæmias of Infancy and Childhood see § 551.

The blood shows marked **diminution of hæmoglobin** *and other changes, and there is* NO DISCOVERABLE HÆMORRHAGE OR PRIMARY ORGANIC LESION. *The disease is probably* PERNICIOUS ANÆMIA *or* SIMPLE HYPOCHROMIC ANÆMIA, *the colour-index and the size of the red cells of the blood being very different in the two diseases.* CHLOROSIS has become rare.

§ **539.** I. **Pernicious Anæmia** (Syn. Addison's Anæmia) is a severe form of anæmia met with principally in middle-aged or elderly persons of both sexes, and characterised by relapses and remissions. Until the introduction of liver treatment it was sooner or later always fatal. This anæmia is the most important member of a large group of anæmias, termed macrocytic (megalocytic) anæmias, which are characterised by the fact that the red corpuscles are larger than the average.

The *symptoms* may be divided into two groups: Those due to anæmia *per se* (see § 535) and those peculiar to the disease. The latter comprise (1) A lemon-yellow tint in the skin, with a distinct malar flush. There is usually loss of weight, but it is not a marked feature. An irregular fever may occur from time to time. (2) The tongue is sore and shows a glossitis in at least 50 per cent. of cases. It may be red and raw or even ulcerated, but usually is smooth, bald and atrophied. Dysphagia with the Plummer-Vinson syndrome may occur (§ 227). (3) Severe attacks of abdominal pain or of vomiting or diarrhœa, may take place, as a result of the achylia gastrica. No acid or pepsin is secreted even after histamine injection. Failure of gastric secretion affects first the hydrochloric acid, then the pepsin, lastly the intrinsic factor; therefore the presence of free hydrochloric acid in the gastric secretion of a patient suspected of pernicious anæmia should make one question the diagnosis. (4) The spleen may be somewhat enlarged. (5) Nervous symptoms, due to subacute combined degeneration of the cord, vary from slight ataxy to paralysis of all limbs. Sometimes these may precede the anæmia and cause

difficulty in diagnosis (§ 811). (6) Hæmorrhages may rarely occur, particularly into the retina or ear, sometimes into other organs. (7) Blood changes (Plate III): (*a*) The red cells are greatly reduced in numbers, frequently 1 to 2 million when the patient is first seen. It is typical of this anæmia that the patient can walk into the consulting room with such a low red cell count; (*b*) the hæmoglobin is usually 20 to 40 per cent.; (*c*) the colour index is high, due to the size of the red cells (§ 529), while the mean corpuscular hæmoglobin concentration is within normal range, although it may be low, if a superadded iron deficiency is present (§ 529); (*d*) marked anisocytosis, with numerous macrocytes and some microcytes; poikilocytosis is invariable; (*e*) the mean corpuscular diameter is commonly above 8 μ and the mean corpuscular volume is usually from 115 to 150 cubic microns; (*f*) nucleated red cells of all types may be present; (*g*) the white cells show a leucopenia with a relative lymphocytosis and the platelets are diminished; (8) the lemon-yellow colour in the skin is accompanied by an excess of bilirubin in the serum (giving a positive indirect van den Bergh reaction, § 331), and an excess of urobilinogen in the urine (§ 383); (9) the bone marrow in a typical case shows numerous megaloblasts.

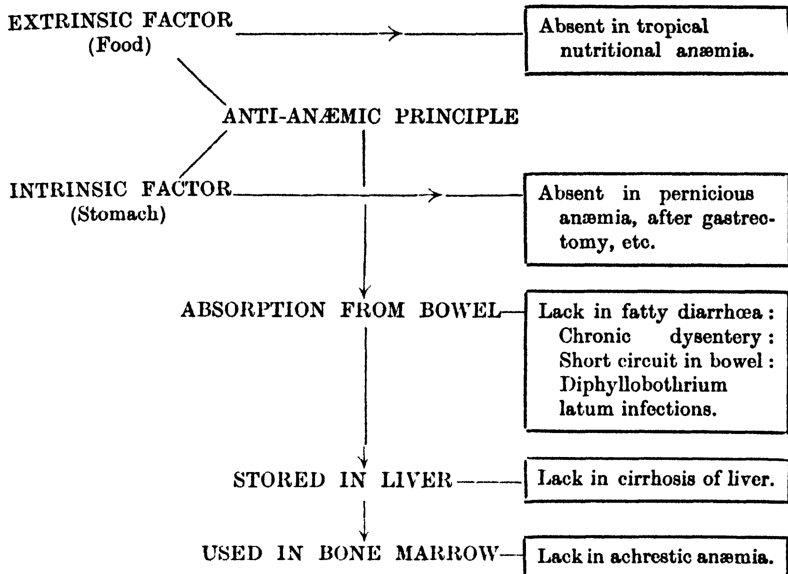
Diagnosis.—The age and appearance of the patient, the insidious onset, the low red count, high colour index and high mean corpuscular diameter and volume, and the achylia make the diagnosis relatively easy in most cases. The other causes of similar *macrocytic anæmias* described under etiology must be excluded. Macrocytosis with nucleated red cells in the blood can occur during very rapid blood formation, when a severe strain is placed on the bone marrow, so that large young red cells are poured out in the blood stream. This may occur in *acholuric jaundice* (particularly the acquired type), *Lederer's anæmia*, and in some cases of *leukæmia*. In the latter disease the infiltration with leukæmic cells probably also hinders the proper maturation of the red cells. The *simple hypochromic anæmias* are differentiated by the size and the severe hypochromia of the red cells. *Carcinoma of the stomach* may rarely lead to a similar anæmia, and must be excluded clinically or by X-ray.

Prognosis.—The disease is slow but progressive, and, before the introduction of liver therapy, was almost invariably fatal. With regular treatment the patients have now an almost normal expectation of life, and if complications occur they are a sign of inadequate control. The main *complications* are cardiac weakness, vomiting and diarrhœa, visceral hæmorrhages, polyneuritis and degeneration in the spinal cord, chiefly affecting the posterior columns (§ 811). Headache, nervousness and prostration are fairly constant, but the intellect is usually clear to the end; sometimes convulsions and coma occur.

Etiology.—Pernicious anæmia comes on insidiously, without apparent reason, usually after the age of 40. Several members of a family may be affected, due presumably to the inheritance of a vulnerable, weak gastric mucosa which fails to produce the intrinsic factor (see below).

A contributory cause is gastritis with resulting atrophy. Other pathological changes present include fatty degeneration of the heart muscle and liver, megaloblastic red bone marrow in the longbones, deposition of iron in the liver, spleen and kidney (giving a Prussian-blue reaction), atrophy of the gastric mucosa and sometimes degeneration of the spinal cord.

TABLE XXXVII.



This anæmia is the most important member of a large group, the macrocytic (megalocytic) anæmias, which are characterised by the fact that the red corpuscles are larger than the average. In almost all members of this group, lack of the *Anti-anæmic Principle* is the etiological factor. Castle showed that the gastric juice of healthy man contains a ferment, secreted by the stomach.[1] This ferment he named the (1) *Intrinsic Factor* (*hæmopoietin*). In health this combines with (2) an *Extrinsic Factor*, found in protein foods, to form (3) the *Anti-anæmic Principle.* The Anti-anæmic Principle is required for the maturation in the bone marrow of primitive megaloblasts to normoblasts. In its absence the bone marrow continues to produce and store megaloblasts, but few other red cells ; therefore the blood shows an anæmia with macrocytes. Macrocytes are large, because they are formed from the large megaloblasts, which lose their nuclei, gain hæmoglobin and enter the blood stream without passing through the various stages of the normal development to smaller sized cells. The Anti-anæmic Principle is absorbed from the small

[1] Experiments with different portions of human stomach have recently shown that in man the intrinsic factor is secreted by the cardiac area of the stomach : this is in contra-distinctfon to the pig, where it is formed in the pyloric glands.

intestine and stored in the liver (and to a slight extent in the kidneys) and passes, as it is required, to the bone marrow. It is obvious, therefore, that any breakdown in the system—Intrinsic Factor, Extrinsic Factor, Anti-anæmic Principle—will prevent the maturation of the megaloblasts in the bone marrow, and lead to macrocytic anæmia. Thus: (1) the Extrinsic Factor may be deficient in the diet (tropical nutritional macrocytic anæmia, § 546); this failure is seen chiefly in parts of India and Africa, especially with pregnancy. (2) The Intrinsic Factor may not be secreted by the stomach; this characterises pernicious anæmia and cases of partial gastrectomy. (3) The Anti-anæmic Principle may be formed, but not absorbed, owing to (i.) damaged intestinal mucous membrane, as by the toxins of diphyllobothrium latum, or (ii.) intestinal hurry, such as occurs after gastro-enterostomy or with gastro-colic fistula; or (iii.) both these causes combined, as in chronic diarrhœa, idiopathic steatorrhœa, cœliac disease and sprue. (4) The Anti-anæmic Principle may be normally formed and absorbed, but the liver may be so damaged (*e.g.*, by cirrhosis) as to be unable to store a sufficient amount of it.

(5) Finally, there are certain macrocytic anæmias which hæmatologically resemble pernicious anæmia but do not react to treatment with liver extracts containing anti-anæmic principle: the gastric acidity is usually normal and neurological symptoms are absent. Wilkinson names these *achrestic anæmias*, and suggests they are due to failure of the bone marrow to utilise the anti-anæmic principle in the liver.

Thus widely differing conditions can lead to a very similar anæmia. The name pernicious anæmia is retained for that variety which is due to failure of the stomach and duodenum to secrete intrinsic factor.

Treatment.—Rest in bed is necessary at the commencement. If the patient is in a very serious condition a small blood transfusion may be needed. The essential part of treatment is to supply the missing anti-anæmic principle. Lightly cooked or raw minced whole liver ½ lb. daily was first used. This was later replaced by daily doses of dessicated stomach powder (B.P.C.) 10–30 G., by liquid or dry liver extracts (B.P.), and more recently by proteolysed liver preparations. In most cases, intramuscular injections of purified liver extracts are now used, as being more reliable and cheaper. If the treatment is effective the reticulocyte percentage in the blood rises to a peak of 10–40 per cent. (depending on the initial red cell count) between the 5th to 10th days, then falls, followed by a rapid rise in the blood count. Therapy *must be continued* for the rest of the patient's life, and controlled by periodic blood examinations. The maintenance dose varies greatly with the brand of liver extract used; commonly 2 c.c. of a concentrated liver extract, *e.g.*, anahæmin, intramuscularly once a fortnight or once a month is sufficient. Each patient must be treated on his own merits, and the minimum dose found which will maintain him at the normal level, in order to prevent relapses or complications. In the presence of sepsis or arterio-sclerosis larger doses may be necessary. If the hæmoglobin rise is very slow or if the mean corpuscular hæmoglobin concentration is below 32 per cent.,

a course of iron by mouth should also be given. It is essential to keep the hæmoglobin level at 14 G. per cent. to prevent complications such as subacute combined degeneration of the cord. In uncomplicated cases of pernicious anæmia highly purified liver extracts in small doses (*e.g.*, anahæmin) are eminently satisfactory, but in cases such as sprue where a blood picture of pernicious anæmia is present, many cases need the extra factors found in the cruder liver extracts (*e.g.*, plexan or campolon). When the extrinsic factor is lacking, this can be supplied by marmite (½ oz. t.d.s., at first, reduced to 1 drachm t.d.s. as a maintenance dose) in warm water, milk, on bread and butter; or by giving liver extract. Folic acid by mouth in doses of 10–20 mgm. a day converts megaloblastic bone marrow to a normoblastic state, and in most cases will rapidly improve the anæmia. As it has no effect on nervous symptoms it should never be given for long periods alone without liver extract. Dilute hydrochloric acid, in drachm doses well diluted, taken with meals, improves the appetite and may stop diarrhœa. Removal of foci of infection should be carried out as a satisfactory response to liver therapy may not be obtained in the presence of chronic sepsis. In achrestic anæmia large doses of liver extract produce little benefit and the effects of blood transfusion are not lasting: proteolysed whole liver by mouth has given better results. A red crystalline substance containing cobalt has now been isolated from liver and found in cultures of Streptomyces griseus. This substance, called vitamin B_{12}, in a dose of 1–10 micrograms (μg.) a day is very effective in macrocytic anæmias and improves cases with Subacute Combined Degeneration of the Cord.

§ 540. II. **Simple Hypochromic Anæmia** chiefly affects females, from thirty-five to fifty years of age, who suffer from general ill-health, loss of appetite, indigestion, chronic abdominal pain, constipation, menstrual disorders, headache, pallor, weakness, and other symptoms due to anæmia (§ 535). The tongue may be sore and glazed with sometimes difficulty in swallowing (Plummer-Vinson syndrome). In most cases there is achlorhydria, or a low acid content of the gastric juice. On examination there is little evidence of disease, apart from the pallor, hæmic murmurs over the heart, and sometimes an enlarged spleen. Spoon-shaped nails (koilonychia) and brittle nails are common. The disease may last many years. It appears to be due to faulty diet or insufficient absorption of iron from the food, for rapid improvement occurs when large doses of iron are given by the mouth. It is aggravated by excessive menstrual loss and by pregnancy. As the name implies, the anæmia is of the low colour index type, the hæmoglobin being reduced much more than the red cells. The colour index may be 0·4 or 0·5. There is variation in size and shape of the red cells, they are pale, and often consist of a ring of cytoplasm around a large unstained centre. (See Plate III.) The cells are microcytic with a mean diameter commonly of 6·2 to 6·7 μ, a mean corpuscular volume below 78 cubic microns and a mean corpuscular hæmoglobin concentration below 32 per cent.—frequently 27 per cent. The leucocytes and platelets are normal or a little reduced in numbers.

Diagnosis.—It is important to exclude other conditions causing low colour index anæmia, such as tubercle and other chronic infections, new growth, chronic loss of blood, as from hæmorrhoids, an unrecognised hæmorrhage from the bowel or ankylostomiasis. If the patient fails to respond promptly to iron, the diagnosis must be revised, and some more serious disease giving rise to a secondary anæmia be sought for.

Treatment.—Iron is specific in this disease. Inorganic are superior to organic preparations, as regards both price and efficacy, but sufficient dosage is more important than the form of iron given. A daily minimum by mouth is 90 grains of iron and ammonium citrate, 9 grains of ferrous sulphate, 45 grains of freshly prepared Blaud's pill or 24 grains of ferrum redactum. These doses may cause loose bowel action, rarely constipation; this effect is less when the iron is taken in pill form. Ferrous salts are superior to ferric salts. Intravenous injections of saccharated oxide of iron (ferrivenin) have proved of great value. Occasionally chlorophyll and ascorbic acid are required to assist the action of iron: it is not uncommon for the total plasma proteins to be low, and then a high protein diet is necessary. The diet should contain abundance of foods with a high content of available iron such as liver, oatmeal, brown bread, dried fruits and pulses. When there is dyspepsia, dilute hydrochloric acid, one drachm three times a day, is of value. As relapses are common, the blood should be examined at intervals after recovery, and the course of iron repeated if indicated.

The patient is PALE *and* ANÆMIC, *there is* RECURRING PYREXIA *and careful search reveals an* ACUTE *or* CHRONIC SEPTIC PROCESS.

§ **541.** III. **Sepsis** causes anæmia by depressing erythropoiesis of the bone marrow by the action of toxins. In addition, such organisms as the hæmolytic streptococcus, the staphylococcus and, most important of all, Cl. welchii (gas gangrene), produce a hæmolytic toxin which may occasionally act directly on the circulating red cells, destroying them and thus causing a hæmolytic anæmia. The anæmia in these infective processes is usually normocytic or microcytic in type and frequently hypochromic. Septicæmia, gas gangrene, malignant endocarditis, acute rheumatic carditis and acute fevers in general, osteomyelitis, ulcerative colitis, puerperal sepsis, perinephric abscess and other chronic suppurations, and tuberculosis all cause anæmia to a greater or less degree. In latent tuberculosis the pallor is often much more marked than the blood count indicates. Usually there is also intermittent pyrexia (§ 512), weakness and wasting (§ 131). If pallor and general debility in a young patient do not respond to iron and general tonic measures, tuberculosis should be suspected.

Treatment is that of the underlying condition. To ensure the presence of adequate hæmatinic factors, the diet should be as full and varied as possible and in the presence of hypochromia, iron should be administered. Blood transfusion may also be required if the anæmia is severe. It helps not only by supplying red cells, but also by introducing into the blood stream fresh complement which is necessary for certain immunological

reactions (complement is commonly considerably diminished in the blood of patients with severe illnesses).

The patient is PALE *and* ANÆMIC *and* LOSS OF WEIGHT *is marked. Physical Examination or special investigation by X-rays, etc., reveals* MALIGNANT DISEASE.

§ **542.** IV. **Early** or **Latent Carcinoma** and **Sarcoma** are attended by anæmia, pallor, weakness, and emaciation; they form the essential parts of cancerous cachexia. The pallor does not yield to iron. Emaciation is, however, usually the most constant and prominent feature, and therefore malignant disease is considered under this symptom (§ 555). The anæmia may be one of the most prominent clinical symptoms, and may be accompanied by hæmic murmurs and on rare occasions intermitting pyrexia; it is usually normocytic or microcytic, but when carcinoma of the stomach is associated with a blood picture of pernicious anæmia, it is macrocytic. Repeated small hæmorrhages, as with carcinoma of the stomach, colon, rectum or uterus, aggravate the condition.

Leuco-erythroblastic anæmia is due to a disturbance of the bone marrow by a variety of causes, especially (i.) *secondary deposits of growth* in the bone marrow; (ii.) *syphilitic disease* of the marrow; (iii.) *osteosclerosis*, (marble bone disease of Albers-Schönberg, § 544. VIII), where the bone marrow is largely replaced by dense sclerotic bone; and in (iv.) *myelosclerosis*, where the bone marrow is replaced by fibrous tissue. The blood may show an anæmia, either macrocytic or microcytic, with the presence of nucleated red cells and myelocytes. X-ray examination of the ribs or long bones is of great help in elucidating the cause.

Rarer Causes of Anæmia

I. **Aplastic anæmia** is a disease in which the bone marrow loses its power of producing all forms of blood cells. Most cases occur in young adults and both sexes are equally affected. The disease occurs in an *idiopathic* or primary form of unknown origin or in a *secondary* form due to bacterial infection or to the action of certain poisons, especially benzol and arsenic and their derivatives, trinitrotoluene, mercury and lead salts, the sulphonamide and thiouracil derivatives, tridione, and overexposure to X-rays or radium. The *symptoms* are those of profound anæmia; of liability to hæmorrhages which may be correlated to a large extent with the severe thrombocytopenia which is present; of stomatitis and ulceration of mucous membranes which may be correlated with the severe neutropenia which is a characteristic of the disease. The red cells are usually normocytic and normochromic but sometimes may be slightly macrocytic (with a high colour index). Evidence of blood regeneration, *i.e.*, reticulocytes and nucleated red cells, is absent. The leucopenia is due almost entirely to diminution of the neutrophil polymorphonuclears so that there may be a relative lymphocytosis of 98 per cent. or even more. The *Diagnosis* can only be made by a careful examination of the blood, including a biopsy of the sternal bone marrow (§§ 534, 919). In these cases the marrow is frequently replaced by gelatinous fatty material with a very scanty cell content. Some cases occur where the marrow is not completely aplastic, and repeated blood transfusions may keep these patients alive for years. The *Prognosis* is extremely bad and the disease is rapidly fatal except in the cases where aplasia is only partial. *Treatment.*—Repeated

blood transfusions are indicated. Proteolysed liver orally gives some benefit in the milder cases: and splenectomy, by removing the most important organ of blood destruction, has been occasionally curative.

II. **Lederer's Anæmia** (Acute febrile anæmia) is a rare disease of young adults, characterised by rapidly developing hæmolytic anæmia, with fever, hæmorrhage, and sometimes jaundice. There may be myelocytes in the blood, rendering the disease difficult to distinguish from acute leukæmia, unless recovery occurs. In some cases hæmolysins have been found in the blood.

Prognosis.—Most cases rapidly end fatally; or fever may fall and recovery occur in a few weeks.

Treatment.—A single blood transfusion may be curative or may have to be repeated.

III. **Chlorosis** has practically disappeared. It is a condition of low colour index anæmia, with a normal red cell count, occurring in adolescent girls. Other diseases which might cause a low colour index anæmia must be excluded (§ 540) before the diagnosis is made. The peculiar greenish-yellow colour of the complexion used to render the diagnosis of these cases easy. The disease responds to iron in adequate doses, combined with general measures to improve the health, such as fresh air, exercise, correction of constipation, etc.

The patient is **pale**; *there is* ENLARGEMENT OF THE SPLEEN, *or the* LYMPHATIC GLANDS, *or both, and* EXAMINATION OF THE BLOOD *may reveal characteristic changes.* The disease is probably LEUKÆMIA, LYMPHADENOMA, ACHOLURIC JAUNDICE, SPLENIC ANÆMIA, a SEQUELA of MALARIA, MARBLE BONE DISEASE, MYELOSCLEROSIS OR GAUCHER'S DISEASE.

§ **543.** IV. **Leukæmia** is a condition characterised by persistent changes in the circulating leucocytes, qualitative changes being the most important, but the majority having a great increase in the total numbers; progressive anæmia, enlargement of the spleen and lymphatic glands, and a fatal termination. There are three chief varieties—*myeloid,* in which the changes chiefly affect the cells from the bone marrow; *lymphatic,* in which the lymphocytes are affected; and the rarer *monocytic,* in which the monocytes are affected. All cases show infiltration of the bone marrow, spleen, liver, and other organs with leukæmic cells. Should the total leucocyte count be normal or below normal, the leukæmia is termed an *aleukæmic leukæmia.* The different types may be grouped into acute and chronic leukæmias.

In ACUTE LEUKÆMIA the total white count is usually not higher than 60,000 per cu.mm.; many cases are aleukæmic. The cells found in the blood are very primitive, with myeloblasts or lymphoblasts in the two chief varieties. Acute leukæmia chiefly occurs in young people and is rapidly fatal, death taking place usually a few days to a few weeks after the initial symptoms.

Symptoms.—(1) Hæmorrhages from the mucous membranes, into the skin, into the joints, serous cavities or tissues generally. (2) Fever, sometimes very high, without discoverable cause. (3) Enlargement of the spleen. (4) In the lymphatic type, enlargement of the lymphatic glands. (5) Severe anæmia. (6) Spongy gums and gangrenous ulceration of the fauces. (7) Leukæmic retinitis is often present. (8) Tumours may form in the skin, or in the bones of the skull; they are sometimes green in the fresh state and are then known as *chloromata.* (9) Not infrequently pains in the joints simulating acute rheumatic fever are present.

Diagnosis is made by a careful blood examination. The presence of a high percentage of myeloblasts or lymphoblasts in the blood and bone marrow is characteristic. Sternal puncture is especially helpful in the aleukæmic cases.

Treatment.—No effective treatment is known: fresh blood transfusions and folic acid antagonists (aminopterin) may give temporary benefit.

CHRONIC MYELOID LEUKÆMIA (Synonyms: Leukæmic myelosis, spleno-medullary

leukæmia). *Symptoms.*—(1) Great splenic enlargement is often the first complaint, giving rise to abdominal swelling. (2) Symptoms of general ill-health, *e.g.*, lassitude, physical and mental depression. (3) Symptoms of anæmia (§ 535). (4) An unexplained venous thrombosis, sometimes in the corpora cavernosa. (5) Hæmorrhage from the nose, stomach, bowel, or into the skin or any other organ. (6) Occasionally irregular fever. (7) Sometimes pruritus.

Diagnosis.—The most important sign is the great enlargement of the spleen. The liver is also enlarged, sometimes greatly; the lymphatic glands may be slightly enlarged. The diagnosis is made by blood examination. There is anæmia, which becomes progressively more severe as the disease progresses; the colour index is usually about normal. Nucleated red cells are present. The total leucocytes may be normal, but are usually greatly increased, 200,000 to 500,000 per cu.mm. being quite usual; of these 30 per cent. or more are immature cells of the myeloid type, myelocytes, and their precursors, myeloblasts; eosinophil myelocytes also occur, and basophil leucocytes are also increased.

Etiology.—The disease occurs in males and females at or past middle age. Nothing certain is known as to the cause. It is possibly due to tumour formation affecting the marrow cells. A similar disease which occurs in domestic fowls has been demonstrated to be due to a filtrable virus, but all attempts to transmit the human disease to animals have failed. The morbid anatomical changes are those of over-growth of the immature cells of the bone marrow, which cells are found in large numbers in all the organs of the body, especially in the spleen and liver.

Prognosis.—This disease is invariably fatal, in the present state of our knowledge, though some live for five years; cases have been known to live twenty-five years from the time of diagnosis. On the whole the disease is more rapidly fatal when there is a high proportion of myelocytes, and especially of myeloblasts. Death occurs from cachexia or hæmorrhage.

Treatment.—X-ray applications over the spleen and bone marrow reduce the size of the spleen and the total numbers of leucocytes with resulting feeling of improved health, but it is doubtful if there is any definite effect upon the progress of the disease: the dose given must be controlled by frequent blood counts. Recently urethane, a growth-inhibiting substance, has been given by mouth, 1–2 G. a day, with results similar to X-ray therapy: nitrogen mustard has been used but is much more toxic.

TABLE XXXVIII.—DIFFERENTIAL DIAGNOSIS OF THE LEUKÆMIAS, SPLENIC ANÆMIA, ETC.

	Enlargement of Spleen.	*Enlargement of Glands.*	*Leucocytosis.*	*Other Characteristics.*
Myeloid Leukæmia (Spleno-medullary Leukæmia).	Very massive.	Usually none.	80,000 to 500,000, with myelocytosis	Progressive anæmia; liver often enlarged.
Lymphatic Leukæmia (Lymphæmia).	Usually slight; rarely massive.	Moderate, soft as a rule.	50,000 to 200,000, with lymphocytosis.	Progressive anæmia.
Lymphadenoma.	Slight; less often massive.	Marked, and hard as a rule.	None or slight.	Little anæmia until late stages.
Splenic Anæmia.	Massive.	None.	Leucopenia.	Gastric hæmorrhage; cirrhosis of liver later.
Gaucher Splenomegaly.	Massive.	None.	Leucopenia.	Familial; pigmentation of face, hands, sclera; red cells not fragile.
Acholuric Jaundice.	Moderate or massive.	None.	None or slight.	Familial; jaundice; red cells unduly fragile.

CHRONIC LYMPHATIC LEUKÆMIA (Synonym: Chronic lymphadenosis). *Symptoms.*—(1) Progressive enlargement of lymphatic glands. (2) General symptoms of

ill-health, such as lassitude, weakness, loss of appetite. (3) The symptoms of anæmia (§ 535). (4) The spleen is sometimes so much enlarged as to cause symptoms by its bulk, though this is never so striking as in myeloid leukæmia. (5) In both forms, but chiefly in the lymphatic leukæmia, the skin may show nodules on face and body, or diffuse erythrodermia with intense itching (§ 647) and purpura may occur.

Diagnosis.—The enlargement of the lymphatic glands and spleen, together with the characteristic blood changes, enables the diagnosis to be made. There is anæmia, not severe in the early stages. The total leucocytes may be normal (aleukæmia), but more usually there is a great increase (leukæmia), up to 200,000 or more, 90 per cent. or more being lymphocytes; immature lymphocytes, lymphoblasts, are present in the more acute cases. Nucleated red cells occur.

Etiology.—The disease is most common about middle life, being rare below 40, and males are affected four times more frequently than females. Nothing is known as to the cause, though it is probably a tumour disease of the lymphocytes. All the lymphatic structures throughout the body are affected, there being great hyperplasia. This affects the lymphoid structures of the spleen, liver, and intestines, as well as the lymphatic glands.

Prognosis.—The disease is invariably fatal, though many live for five to ten years after the diagnosis is first made. Death is caused by cachexia or hæmorrhage.

Treatment.—No effective treatment is known. X-ray applications to the enlarged lymphatic glands may serve to diminish the total numbers of cells, and the patient may have improved health in consequence, but it is doubtful if this has any effect upon the progress of the disease. Urethane, 1–2 G. daily, may have similar effects.

Leukæmoid blood pictures: Certain diseases are sometimes accompanied by blood changes which, if divorced from the clinical picture, may suggest a leukæmia. Very rarely the true diagnosis cannot be made till autopsy shows the absence of any true leukæmic infiltrations. Measles, glandular fever, and whooping cough may simulate lymphatic leukæmia, while acute infections, tuberculosis, bee stings and mercury poisoning are among the conditions in which a myeloid leukæmia may falsely be diagnosed from the blood picture.

V. LYMPHADENOMA (§ 572) manifests itself clinically by enlargement of groups of lymphatic glands. At a later stage anæmia and splenomegaly become evident.

VI. In ACHOLURIC JAUNDICE (§ 328) there are anæmia, enlarged spleen, and weakness. It often occurs in families, and there are recurrent attacks of jaundice.

§ 544. VII. **Splenic Anæmia** is a rare disease, the characters of which are: (1) Splenic enlargement which cannot be connected with any recognised cause; (2) absence of any enlargement of the lymphatic glands; (3) secondary anæmia; (4) leucopenia, with relative lymphocytosis; (5) an extremely prolonged course lasting years; and (6) a tendency to hæmatemesis from time to time. Its nature is still in doubt, but there is an increasing tendency to consider that this term includes a large number of cases of generalised reaction to infection of the lymphoid tissues. It is undoubtedly true that chronic infective splenomegalies are much more frequent than has been recognised in the past. The patient may only come under observation during the *second* stage of the disease when anæmia with its concurrent symptoms is complained of. In most cases the splenic enlargement appears to precede the anæmia, and the patient may not seek advice until his spleen has reached the umbilicus. During periods of enlargement of the organ attacks of pain occur. The spleen in this disease attains an enormous size, often as great as that which occurs in myeloid leukæmia. The enlargement is due to venous congestion of the organ with proliferative fibrosis and some atrophy of the Malpighian bodies. As the disease progresses there is loss of strength without emaciation, accompanied by gastric disturbance and a tendency to hæmatemesis. Sometimes there is a moderate enlargement of the liver. Pyrexia is present during the active stages of the disease. The blood shows a diminution of the number of the red cells, and a greater diminution of the hæmoglobin. Poikilocytosis may be present. In the *third* stage of the disease all the symptoms are aggravated, and in a few cases of the disease the so-called "Banti

disease" supervenes, with fatal termination. The name "Banti's disease" has been given to a group of symptoms comprising cirrhosis of the liver and ascites, consequent on splenic anæmia, as described above.

The *Diagnosis* from most forms of *secondary anæmia* is effected by the great enlargement of the spleen, and from *leukæmia* by the characteristic blood changes in that disease (§ 543). *Pernicious anæmia* is rarely associated with a greatly enlarged spleen; and the macrocytosis of pernicious anæmia is diagnostic. The chief practical difficulty lies in diagnosing the disease from *cirrhosis of the liver* with accompanying enlargement of the spleen. *Banti's disease* may be almost impossible to diagnose from cirrhosis of the liver unless a history including the blood changes of previous years can be obtained. Some cases regarded during life as splenic anæmia are found after death to be due to *visceral syphilis*, cirrhosis of the liver, or thrombosis of the portal vein. In *Kala-azar* there is a history of residence abroad, and liver or sternal puncture reveals the parasite.

Prognosis.—The disease is a chronic progressive disorder. It used to be said that death occurred in six months to two years, but it is now known that cases may live ten, twelve or even twenty years after the commencement of the disease. Death takes place by asthenia, occasionally by syncope or gastric hæmorrhage.

Etiology.—Men are more often affected by this disease than women; it occurs mostly in adult life, but may occur at all ages. The cause is unknown.

Treatment is symptomatic. Iron in large doses is given for the anæmia. X-rays may help. Splenectomy together with portocaval anastomosis is of value in the early stage; later the operative risk is greater. It should be performed only if the blood platelets are normal or diminished, because after splenectomy a great rise occurs. If excessive, this leads to fatal thrombosis, usually of the portal vein. Death from hæmorrhages, even years after splenectomy, is not uncommon.

VIII. **Marble Bones** (Albers-Schönberg disease) (§ 598) and **Myelosclerosis** are two rare and separate diseases, but both have a similar effect on the blood picture, causing a leuco-erythroblastic anæmia (§ 542). In marble bone disease excessive growth of dense bone replaces the bone marrow, while in myelosclerosis the marrow is replaced by fibrous tissue. Blood formation must thus, to a large extent, take place in extra-medullary foci which are found chiefly in the spleen, which is enlarged, and also in many other organs, *e.g.*, liver, kidney or even muscle. These diseases are very chronic and are incurable. Their cause is unknown although in the case of marble bones a hereditary factor is evident. Hæmatinics should be given but the large spleen must not be removed as it may be the chief site of blood formation.

IX. **Gaucher Splenomegaly.**—This condition is the most important member of a group of diseases, usually congenital and familial, with a predilection for the Jewish race, and due to disordered lipoid metabolism. In Gaucher's disease the organs, especially the liver and spleen, are filled with cells distended with the lipoid kerasin; in Niemann-Pick's disease, with the lipoid lecithin; in Hand-Schuller-Christian's disease, with cholesterol. In Gaucher's disease there is splenomegaly and hepatomegaly without cirrhosis, leucopenia, hypochromic anæmia, and a yellowish-brown pigmentation, chiefly on the face, hands and sclera. The disease is eventually fatal, but may be so chronic that death does not occur till middle age. **Niemann-Pick's** disease has similar symptoms. It shows (1) enormous enlargement of the liver and spleen and often of the lymphatic glands; (2) a brownish skin, (3) moderate anæmia, and (4) progressive cachexia, always fatal before the age of 2. No treatment avails. In **Hand-Schuller-Christian's** disease the bones are chiefly affected, particularly the skull, usually causing exophthalmos and diabetes insipidus by pressure.

The patient is very **pale** *and anæmic, and there are or have been* SPONGINESS *of the* GUMS, PURPURIC SPOTS, *and brawny indurations of the legs.* The disease is probably SCURVY.

§ 545. X. **Scurvy** is a "deficiency disease" due to a too long continued diet lacking in Vitamin C. Scurvy is attended by extreme debility, anæmia, sponginess of the gums, and hæmorrhages.

The disease may occur in adults or in infants, especially in the first 12–18 months of life. Certain *symptoms* are common to both. (i.) The gums become spongy, swollen, and bleed readily. Sloughing may follow, and the teeth become loosened; the breath is very offensive. When there are no teeth this symptom is usually absent. (ii.) Hæmorrhages occur (*a*) Into the skin, especially in adults where purpuric spots and swellings of brawny consistence are found about the flexures of the joints, especially the popliteal space. These swellings are due to hæmorrhages into or beneath the skin; if the former, they are purple; but if beneath the skin, the colour may be pale. (*b*) Hæmorrhages from the mucous membranes produce epistaxis, melæna and hæmaturia. In infancy hæmaturia may be the first sign. (*c*) Under the periosteum of the legs large painful swellings due to blood effusions. (*d*) In infants hæmorrhage may occur under the periosteum of the orbit, producing proptosis, and occasionally intracranial hæmorrhage produces cerebral symptoms. (iii.) Pains in the back and limbs are complained of early, and in infants cause them to scream when touched. (iv.) A marked anæmia is due to deficient maturation of the red cells: blood platelets are usually scanty. (v.) Profound weakness and prostration, with mental depression and syncopal attacks, made it impossible for sailors afflicted during the Middle Ages to secure fruit and vegetables even when they reached shores of desert islands. (vi.) Constipation and albuminuria are often met. (vii.) Death may ensue either from syncope, asthenia, or complications. Among the latter may be mentioned sanguineous effusion into the pleura or meninges, pneumonia, and sloughing of the skin. *In infancy* (Barlow's disease) the symptoms are those already enumerated, the special features being (i.) The child cries when washed or dressed, screams if the legs are touched, and is very still when at rest (pseudo-paralysis). (ii.) Muscular weakness becomes marked, but emaciation is often absent. (iii.) The temperature may be raised, especially after large or recent hæmorrhages, to 100–102°. (iv.) X-ray examination shows a loss of differentiation in the bone structure, leading to a "ground glass" appearance. In acute cases there is widening of the epiphysial line and even dislocation of the epiphysis, whereas in the more chronic cases there is a clear space (Trummerfeld zone) in the diaphysis near the epiphysial line.

Diagnosis.—The diagnosis of scurvy from other causes of *purpuric* eruption is afforded by the condition of the gums, and the hard brawny swellings, which are peculiar to scurvy, and also by the degree of prostration present. Slighter cases are, however, very difficult to diagnose, as similar symptoms may be seen with purpura. The capillary resistance test usually proves positive. A blood count at once distinguishes scurvy from *acute leukæmia,* which is also accompanied by stomatitis and hæmorrhage. *Acute rheumatism* affects the joints, whereas in scurvy they are free. In infants with *syphilitic* pseudo-paralysis, crepitation and pain on moving the limb occur, due to separation of the cartilage from the diaphysis. *Infantile paralysis* is not accompanied by spongy gums, swellings of the legs or hæmorrhages. When there is Vitamin C lack after giving a test dose of 300 mgm. of Vitamin C to an adult, most of it is retained in the body. The amount excreted in the urine may be estimated by titration with dichlorophenol-indophenol: this detects early stages of deficiency.

Prognosis.—Under vigorous treatment symptoms rapidly subside in the course of a week. If the patient is seen at a late stage, or if from failure to diagnose the disease the diet is not altered, death occurs from syncope or complications such as diarrhœa, bronchitis, and pneumonia, or any of the acute specific fevers. Unfavourable symptoms are severe dyspnœa, syncope, scanty urine, and elevations of temperature.

Etiology.—Scurvy is due to the absence of Vitamin C (Ascorbic Acid). It used to be the scourge of the British Navy, until the introduction of lemon juice as a prophylactic. It is occasionally seen when prolonged strict dieting has been practised for the treatment of peptic ulcers, and in those who never eat fresh vegetables and fruit. It occurs in infants and invalids fed only with proprietary foods and peptonised,

sterilised or condensed milk. The vitamin is stored in the suprarenal glands, and increased amounts are needed in the presence of any infection. The average daily requirement for an adult is 60–70 mgm. and for an infant 25–50 mgm.

Treatment.—Prophylaxis consists in giving Vitamin C: it is contained in large amount in the juice of fresh lemons, oranges, and fresh green vegetables; in moderate amount in roots, such as swedes and potatoes; and in small amount in milk and fresh meat. Prolonged boiling and alkalies destroy the vitamin; soda should not be used in cooking vegetables. *Cure* is obtained with the juice of two or three lemons or oranges daily, together with fresh meat and vegetables. Ascorbic acid, 40–100 mgm. by mouth, daily, is successful; in urgent cases, give the same dose intravenously. To children of a year old give ascorbic acid, 20–30 mgm. by mouth, daily, or give fresh milk, grape, orange, lemon, and swede juice, with potato pulp and raw-meat juice. The potatoes are steamed, rubbed through a sieve and beaten up with milk to the consistence of thick cream. *Local* treatment consists in wrapping the limb in cotton wool and preventing movement.

There is **pallor** *of the skin and the patient has* BEEN ABROAD. Inquiry should be made for MALARIA, CHRONIC DYSENTERY, WORMS and other PARASITES, or other TROPICAL DISEASES.

§ 546. XI. Various **Tropical Diseases** and **parasitic** conditions rarely, if ever, seen in England, are attended by intense anæmia. There are three groups: (1) megalocytic anæmia resembling pernicious anæmia, showing anisocytosis and large numbers of megalocytes *in the blood picture; generally the Price-Jones curve is broadened at* the base and displaced to the right. The average diameter of the corpuscle in these anæmias exceeds 7·7 microns and the colour index generally equals or exceeds 1·0. Hyperchromia or orthochromia is the rule; hypochromia, if found, suggests some complication. (2) Normocytic anæmia, when the average diameter of the corpuscle equals or is less than 7·7 microns, the colour index is 0·8 to 1·0 and the Price-Jones curve is normal, orthochromia or hypochromia is present. Malaria constitutes a good example of this type of anæmia. (3) Microcytic hypochromic anæmia as seen in ankylostomiasis. The Price-Jones curve is displaced to the left, microcytosis is marked, and the average diameter of the corpuscle is below 6·7 microns.

1. *Megalocytic Anæmias.*—Most of the megalocytic anæmias are of nutritional or alimentary origin. The most important is in *sprue* (§ 311), in which the degree of anæmia varies at different stages of the disease, sometimes being absent during the first few months; generally the anæmia is megalocytic in type, but not as marked as in pernicious anæmia except in severe cases where the blood picture may be quite indistinguishable. The histamine test meal shows acid in 70 per cent. of sprue cases, which with the characteristic stools, intestinal flatulence, great loss of weight and absence of spinal cord involvement distinguish it from pernicious anæmia. A similar hæmatological response to liver extract therapy is observed in sprue provided the diarrhœa is controlled by suitable dietary (§ 297. XVII) and rest. *Nutritional megalocytic anæmia* is another disease common in parts of India and Africa; it particularly affects pregnant women and appears to result from a diet deficient in sources of animal protein, such as meats, eggs and milk. Two types occur: (1) non-hæmolytic and (2) hæmolytic, associated with chronic malarial splenomegaly. The latter group shows evidence of blood destruction such as hyperbilirubinæmia, mild hæmolytic jaundice and bilious or chocolate-coloured stools. The indirect van den Bergh reaction is strongly positive (3 to 10 units). In both types the oxyntic cells secrete hydrochloric acid, thus clearly differentiating the condition from Addisonian pernicious anæmia (see § 539). The non-hæmolytic type of nutritional macrocytic anæmia responds satisfactorily to marmite (1 drachm thrice daily), and also to liver extract or folic acid by mouth or parenterally, in the dosage effective in pernicious anæmia. In the hæmolytic type massive dosage with marmite and large injections of liver extract are necessary to elicit a satisfactory hæmatological response and sustained blood regeneration. This is

due to the fact that a secondary hæmolytic factor is at work as well as the nutritional deficiency which causes the megaloblastic degeneration of the bone-marrow and megalocytic anæmia. *Diphyllobothrium anæmia* may be indistinguishable from pernicious anæmia, being megalocytic in type and responding to liver extract therapy. Only 1 in 500 cases develop anæmia, however; then achlorhydria is frequently present. Relapse occurs unless the worm is removed. The anæmia of *Oroya fever* is hæmolytic, being caused by the *Bartonella bacilliformis* located in red cells. Megaloblastic hyperplasia of the bone-marrow follows, and a severe degree of megalocytic anæmia, closely resembling pernicious anæmia, ensues. Hyperbilirubinæmia is present, but there is a leucocytosis of some 20,000 per cu.mm., the neutrophils equalling 60 to 70 per cent. No therapeutic response to liver extract is to be expected.

2. *Normocytic Tropical Anæmias.*—This group may originate in *nutritional diseases* like beri-beri and pellagra, or from bacterial infections such as leprosy and undulant fever. *Protozoal* and *helminthic* causes include chronic *amœbiasis*, especially if associated with hepatitis or liver abscess, *malaria*, where it may be associated with a hæmolytic strain of malignant tertian, *blackwater fever*, *trypanosomiasis* and *kala-azar*. In the latter disease, anæmia is associated with an intense leucopenia, which accounts for its liability to secondary infections like broncho-pneumonia, cancrum oris, etc. Agranulocytosis may develop during treatment. *Helminthic* infestations frequently cause anæmia in the tropics and are often associated with an eosinophilia. The blood flukes, especially *S. hæmatobium* and *S. japonicum*, cause blood destruction owing to the flukes in the portal system living on the red corpuscles.

§ 547. XII. **Ancylostome Worms** may be present without producing symptoms' but sometimes anæmia (of the microcytic, hypochromic type), asthenia and debility result. In severe cases the skin becomes yellow and dry, the mucous membranes pale, and dyspnœa, palpitation, pulsating cervical veins, hæmic murmurs, retinal hæmorrhages, œdema of the feet and serous effusion may be found. Epigastric tenderness, enlarged liver, dyspepsia, mental and physical lethargy are characteristic. Occult blood may occur in the stools, but melæna is rare. The two common species affecting man are *Ancylostoma duodenale* and *Necator americanus*; their ova are voided in the fæces. In warm, moist earth rhabiditiform larvæ subsequently develop, and infect man by boring through the skin, invading the blood-vessels, passing to the right heart and finally the lungs, whence they proceed *via* the trachea, œsophagus and stomach to their adult habitat—the duodenum and jejunum. Although infrequently fatal, ancylostomiasis predisposes to intercurrent diseases like pneumonia and dysentery. Ancylostome dermatitis may result during invasion of the skin by the larvæ, and later anæmia with a low colour index (0·5) and eosinophilia may be found.

The *Diagnosis* is made by finding the eggs in the fæces either in ordinary smears or by a flotation concentration method. Anæmia associated with eosinophilia in miners or people from the tropics should arouse suspicion.

Treatment.—Prevention includes the wearing of good shoes and boots, the proper disposal of night soil and the treatment of carriers. In miners proper sanitary arrangements are essential. Medical treatment consists in the administration of tetrachlorethylene 4 c.c., after preliminary light diet and saline purgation. A further purge should be given an hour after the drug. Toxic effects on the liver may be lessened by the administration of calcium lactate (gr. lx.) daily for a few days beforehand. If the ova reappear, carbon tetrachloride 3 c.c., oil of chenopodium 1 c.c. or thymol gr. 60 may be tried. Iron should be given as for hypochromic anæmia (§ 540), and a well-balanced diet given, adequate in proteins and vitamins.

§ 548. XIII. **Sickle-Cell Anæmia** (African anæmia) is a hæmolytic anæmia almost exclusively confined to negroes or negroid stock. The disease is hereditary and transmitted as a Mendelian dominant (cf. Cooley's anæmia, § 551). Of all negroes, 5–7 per cent. show a latent sickle cell trait in their blood, although only some of them are anæmic.

Symptoms are (1) those of anæmia, but the patients are ill-nourished and abdominal crises simulating those of tabes and rheumatic-like pain in the bones and joints may occur. (2) There may be mild hæmolytic icterus, enlarged lymph glands and splenomegaly. (3) Chronic ulcers of the legs often occur and X-ray may show osteoporosis. (4) Exacerbations of fever, jaundice and enlargement of the spleen are not uncommon.

Diagnosis.—(1) A hanging drop preparation of blood ringed with paraffin, after standing a few hours, shows the formation of sickle-shaped or oat-shaped red cells. (2) The anæmia is generally normocytic in type, but megalocytes, microcytes, anisocytosis, poikilocytosis, polychromasia and normoblasts may be present. Leucocytosis is constant (10,000 to 30,000 cells per cu.mm.).

Prognosis.—Symptoms generally develop at the age of 13 and many die before the age of 40. Once symptoms have developed ultimate recovery is rare.

Treatment.—No specific treatment is known; transfusion benefits, but splenectomy and iron and liver extract have all proved disappointing.

§ **549.** XIV. *There is a history of exposure to* RADIUM, X-RAYS, *or to* INDUSTRIAL *or other* CHEMICAL POISONS.

Radium is much the more dangerous and radium workers insufficiently protected and receiving small doses of radium over long periods may develop aplastic anæmia. Even with relatively good protection to radium there is often a leucopenia with reduction of neutrophils and relative lymphocytosis with some degree of anæmia. Those exposed to *X-rays* (radiologists and radiographers) may show pallor and anæmia due to working in dark and sometimes ill-ventilated rooms. With modern methods of protection there is no evidence that X-ray *per se* causes anæmia, but leucopenia can result and workers should have frequent blood examinations: when there is a *progressive* fall in the absolute lymphocyte count below 1,500 per cu. mm. or the polymorphonuclear count below 3,000 per cu. mm. a holiday is imperative. The use of *deep X-ray therapy* for malignant disease requires expert blood examination of the patients; it should seldom be given if leucopenia is present; the condition of the blood must be watched throughout any course of treatment.

Lead poisoning is dealt with in § 553. *Mercury* does not often lead to anæmia. *Potassium chlorate* and *nitrobenzene* may cause severe anæmia with low colour index and leucocytosis. *Sulphonamide* derivatives, T.N.T. and chronic *benzene* poisoning can cause a condition like aplastic anæmia; it can occur in workers with rubber and with benzol.

Snake venom and tissue poisoning after *severe burns* may cause severe anæmia, high leucocytosis and hæmoglobinuria.

The patient is a YOUNG MALE *and complains of* SEVERE BLEEDING *after trivial injuries and probably has a history of* JOINT AFFLICTIONS. *The disease is probably* HÆMOPHILIA.

§ **550.** XV. **Hæmophilia** is a hereditary disease affecting males and characterised by a constitutional tendency to uncontrollable hæmorrhage without sufficient cause.

The *Symptoms* are divided into three sets: (1) Hæmorrhages from mucous membranes, or, after some slight injury, from the skin. Nothing abnormal may be noted in a subject of hæmophilia until he has a tooth extracted or a trifling abrasion, when uncontrollable bleeding due to capillary oozing sets in, and lasts for hours or days. When the bleeding occurs from a mucous surface, large blood tumours may form as the blood coagulates. (2) Interstitial hæmorrhages occur spontaneously or after injury in the form of petechiæ or hæmatomata. (3) Affections of the joints, especially the knees and elbows, are met with, and three stages are described: (i.) Recurrent hæmarthroses or effusions of blood into the joints, of acute onset, sometimes attended by pyrexia; (ii.) reactionary synovitis; and (iii.) cicatrisation which may lead to permanent joint changes.

Diagnosis.—A single severe hæmorrhage does not warrant a diagnosis of hæmophilia, but recurrent hæmorrhages with slight cause are characteristic. The clotting time is greatly prolonged but the bleeding time is normal. There is a family history of a tendency to bleeding. The joint affections are diagnosed by the presence or history of other signs of hæmophilia.

Prognosis.—The disease usually becomes evident during the first few years of life, and as a rule tends to be less troublesome as life advances, disappearing about the age of thirty or forty. The tendency to hæmorrhage is much greater at some times than at others. Great anæmia occurs from excessive bleeding, and life has been lost after trivial operations such as the extraction of a tooth or circumcision.

Etiology.—Hæmophilia occurs in families for generations. It is met with in males, rarely if at all in females, but the diathesis is transmitted as a Mendelian sex-linked recessive character through the female who herself may remain unaffected. The cause of the condition is unknown: theories advanced include those of excessive stability of platelets and excess of an anti-thrombokinase in the blood.

Treatment.—Males in a bleeder's family should be guarded from any injury or operation. The daughters are not endangered by parturition, but their sons will probably be bleeders, and their daughters will pass on the same tendency to their offspring. The only persons in a hæmophilic family who can marry with impunity are the unaffected males and their descendants. When bleeding occurs, rest is essential, and immediate local treatment should be carried out. Loose clots should be sponged away with hot water and a swab soaked in thrombin or Russell's viper venom in 1 in 10,000 solution, applied to the bleeding surface. 10 c.c. of horse serum, or better, normal whole blood should be injected intramuscularly and if the hæmorrhage does not stop a small blood transfusion seldom fails. If operative proceedings are essential, a blood transfusion should be given before or during the operation.

XVI. PAROXYSMAL HÆMOGLOBINURIA *and* NOCTURNAL HÆMOGLOBINURIA are described in § 409.

§ 551. **Anæmia in Infancy and Childhood.**—All the forms of anæmia above described, with the probable exceptions of Pernicious Anæmia and Chlorosis, may occur in children under fourteen, and are produced by the same causes which affect adults, but they appear in a very different order of frequency and present certain distinguishing features. (*a*) The spleen tends to become enlarged in all forms of anæmia in children, but is markedly

In Infancy.	*In later Infancy and Childhood.*
I. Nutritional Anæmia.	I. Nutritional Anæmia.
II. Anæmia due to Infection.	II. Anæmia due to Infection.
III. Infantile Scurvy (§ 545).	III. Parasitic diseases, especially malaria (§ 510) and intestinal worms (§ 546)
IV. Von Jaksch's Syndrome.	IV. Acholuric Jaundice (§ 328).
V. Hæmolytic Disease of the New Born.	V. Lederer's Anæmia (§ 542).
VI. Cooley's Anæmia.	VI. Sickle-celled Anæmia (§ 548).
VII. Hæmorrhagic Diseases of the New Born.	

so in splenic anæmia of infancy, leukæmia, and lymphadenoma. (*b*) The blood changes also differ considerably from the blood changes met with in the same diseases in adults. In infancy and childhood slight causes lead to blood alterations of a marked type which, if occurring in an adult, would signify severe disease. The reason is that in the child there is no reserve of yellow bone-marrow which can be changed into

active red marrow in times of stress. So any extra demand results in an overactivity which furnishes only increasing numbers of immature cells to the blood stream. In anæmias, the number of red cells is reduced at a comparatively early stage and nucleated red cells readily appear. Leucocytosis, chiefly a lymphocytosis, is easily produced by the activity of the large amount of lymphoid tissue in the child. However, when a polymorph leucocytosis occurs, myelocytes are not uncommon.

I. **Nutritional Anæmia of Infancy and Childhood.** Anæmia in *infancy* is avoided by ensuring (1) an adequate antenatal store of iron in the liver, and (2) a full supply of maternal milk. As little iron is contained in maternal milk, and less in artificial foods, anæmia tends to develop when an infant does not obtain a regular full feed of breast milk from a mother who is herself receiving a sufficient diet. Nutritional anæmia occurs (1) with premature and multiple births, due to subnormal stores of iron in the liver as two-thirds of the iron in the fœtus is laid down in the last three months of intra-uterine life ; (2) with milk feeding over too prolonged a period, as in later infancy more iron is needed than is obtained from milk alone. This anæmia is hypochromic and reacts well to iron in the form of iron and ammonium citrate grs. ii increased to grs. v t.d.s.

In later infancy and childhood a nutritional anæmia may be caused by poor diet or poor absorption of an adequate diet, such as may occur in chronic diarrhœa (§ 310) and cœliac disease (§ 307).

II. **Anæmia due to Infection** is more important in later infancy and in childhood. The rapid growth of a child necessitates rapid absorption and utilisation of the hæmatinic factors, particularly iron in the food, if anæmia is to be avoided. Any infection, especially if long standing, tends to depress the erythropoietic tissue of the bone marrow and also tends to lessen the absorption of the iron in the food and thus a hypochromic anæmia is very common in pyogenic and respiratory infections, in syphilis and tuberculosis and to a lesser extent in the acute specific fevers. *Treatment* is that of the underlying cause, with full diet and iron.

III. **Infantile Scurvy** is described in § 545.

IV. **Von Jaksch's Syndrome** (Synonyms: Anæmia Infantum Pseudo-leukæmia, Splenic Anæmia of Children) occurs in children from six months to two years of age, and is characterised by anæmia, leucocytosis and enlargement of the spleen. The splenic anæmia of adults is not the same disease.

Symptoms.—(i.) Pallor due to anæmia of insidious onset, sometimes preceded, sometimes followed, by (ii.) enlargement of the spleen, which may attain a great size. Attacks of pain may occur, due to perisplenitis. (iii.) The liver is moderately enlarged, and in some cases the glands also. (iv.) There is irregular pyrexia and gastro-intestinal disturbance. (v.) The patient may remain plump throughout, but in severe cases, usually becomes greatly emaciated. (vi.) In serious cases hæmorrhages occur from the mucous membranes and into the skin. (vii.) The blood changes are characteristic —anæmia, usually with a normal colour index, large numbers of nucleated red cells of the normoblast type, and a high leucocytosis, with large numbers of immature cells of the myeloid type.

The *Diagnosis* is difficult only in the early stages. In both *syphilis* and *rickets* we

often meet with anæmia, enlargement of the spleen and of the liver; but the spleen never attains the same size, and the blood changes are never so marked, as in splenic anæmia of infants. In children severe *secondary anæmia* may present leucocytosis, but the polymorphism of the leucocytes is not found. *Myeloid leukæmia* rarely occurs in children, and the leucocytosis has different features. The diagnosis of Von Jaksch's syndrome depends on different features at different stages; and it would appear that this disease has been described by various observers, under different names, according to the stage under observation. In the early stage the changes in the red corpuscles are prominent, resembling those in severe anæmia. In the later stages the leucocytosis becomes more noticeable; hence the name "pseudo-leukæmia."

The *Prognosis* is good. The course is short, and recovery usually complete, but cases relapse under bad hygienic conditions. Hæmorrhages and petechial eruptions are serious symptoms. The lower the number of red, and the higher the number of white corpuscles, the graver is the prognosis. Death occurs from exhaustion or intercurrent diseases.

The *Etiology* is varied. Most believe that the condition is a reaction of the infant's bone marrow to many different causes of anæmia. It may be associated with rickets or syphilis.

The *Treatment* consists in remedying the causal conditions. Intestinal disorder must be rectified. Fresh air is essential, and a full adequate diet should be given. Of drugs, liver extract and iron are the best; cod-liver oil and malt are useful adjuncts.

V. **Hæmolytic Anæmia of the New Born** (Syn. Erythroblastic Anæmia) is characterised by large numbers of normoblasts in the blood and a severe anæmia of hæmolytic type. Foci of extramedullary erythropoiesis are frequent in the liver, spleen, and elsewhere. These anæmias may occur in the fœtus with gross œdema, *Hydrops Fœtalis*: and in the new born, with jaundice in *Icterus Gravis Neonatorum* (§ 327) or without jaundice as *Anæmia Hæmolytica Neonatorum*. Recently it has been shown that most cases of erythroblastic anæmias are due to an hereditary factor, the Rhesus (Rh.) factor, which is present in the red cells of the fœtus (§ 537). This Rh. factor leads to the formation of agglutinins in the mother's blood which pass the placental barrier and wreak havoc on the fœtal red cells. The condition most commonly arises where the mother is Rh.-negative, while the fœtus (and thus the father), are Rh.-positive. *Treatment* consists of giving one or more blood transfusions with Rh.-negative blood preferably not derived from the mother (§ 537). In some cases there may be a prothrombin deficiency in the blood also and vitamin K should then be given.

VI. **Cooley's Anæmia** (Syn. Mediterranean Anæmia) is a special type, found almost exclusively in children of Mediterranean origin. The blood shows a typical erythroblastic anæmia with enormous numbers of normoblasts in addition to oval red cells and target cells in the blood stream: also a leucocytosis. There is a yellowish pigmentation of the skin, enlargement of the spleen and liver and a peculiar Mongolian facial expression due to bony changes. *Etiology.*—Recently it has been shown that this condition is comparable to Sickle-Cell anæmia (§ 548). In both, in addition to the overt cases, there are many more people with a latent trait and red-cell deformity. It is believed that if both parents show the trait, then the child will show the overt disease. *Treatment.*—Splenectomy may prolong life, but the disease is slowly fatal; the children rarely living to the age of 10.

VII. **Hæmorrhagic Diseases of the New Born** (Syn. Melæna neonatorum).

Hæmorrhage in the new-born commences on the 2nd or 3rd day of life most commonly from the gastro-intestinal tract (melæna, hæmatemesis), but sometimes from the umbilical cord, mucous membranes, into the skin or the internal organs, or following circumcision. It is due to a deficiency of prothrombin in the blood which in turn is due to lack of vitamin K. *Treatment.*—Vitamin K by injection (0·5–1·0 mgm. daily) or by mouth (1–5 mgms. daily), are specific. Intramuscular

injections of whole blood to supply prothrombin (10–20 c.c., 2 hourly until the bleeding stops) may also be used. Blood transfusions may be necessary.

B. PALLOR OF THE SKIN IS MARKED, *but* ANÆMIA IS SLIGHT, *the drop in the hæmoglobin content of the blood relatively small, and is* NOT READILY AMENABLE TO IRON ADMINISTRATION. The common causes are :—

I. Syphilis (§ 552).
II. Lead poisoning (§ 553).
III. Acute and chronic renal diseases.
IV. Chronic hepatic disease.
V. Latent Tuberculosis.
VI. Gastro-intestinal conditions.
VII. Cardio-vascular conditions.
VIII. Addison's disease, myxœdema and other maladies of Groups II and III.

§ **552.** I. **Syphilis** leads to a degree of pallor which may simulate simple anæmia closely : some degree of secondary anæmia may develop at any stage, even quite late in the disease. It is caused by infection with *Treponema pallidum* (*Spirochæta pallida*) and is of especial importance in medicine by reason of its protean manifestations. If not recognised and treated early, it is liable to break out anew during the whole lifetime of the individual without fresh infection, even after many years of quiescence : in its later stages it may produce serious inflammatory and degenerative lesions in many different parts of the body, particularly in the cardio-vascular and central nervous systems. There are two chief forms: (A) Acquired, and (B) Congenital Syphilis.

(A). ACQUIRED SYPHILIS. *Symptoms.*—When no history of primary syphilis is obtainable and no physical signs discovered, the disease may be latent and unsuspected. For convenience the symptoms of syphilis are divided into four stages. There is a *period of incubation* which generally lasts about three weeks but which may vary between ten and forty-six days. *Primary Stage.*—(i.) At the end of the incubation period there generally appears at the site of inoculation a primary chancre. The initial manifestation is a flat, elevated, painless papule which slowly enlarges and later breaks down to form an indurated ulcer with a slight serous discharge (the hard or Hunterian chancre). It is usually single, and since the infection is commonly conveyed by sexual intercourse, it is likely to be found most commonly on the prepuce or glans penis of the male, and the labiæ and nymphæ of the female. Occasionally the initial papule desquamates without ulceration: and when the primary lesion occurs on the cervix or vagina it will often be entirely overlooked. (ii.) Within one to two weeks the lymphatic glands draining the primary focus become enlarged and hardened—especially in the groin. (iii.) The lesion after a time cicatrises and may leave behind a scar, or some slight coloration, but it often heals without leaving any mark.

The *Secondary* symptoms make their appearance when the infection becomes generalised about three to six weeks after the first appearance of the chancre (four to twelve weeks after inoculation). They comprise a

rash, sore throat, " mucous patches ", enlargement of the lymph glands, pyrexia and other constitutional symptoms. (i.) The early *rash* appears chiefly on the chest and abdomen as a faint generalised dusky macular eruption which takes about three weeks to mature and three weeks to decline (it may be brought out more clearly by a warm bath) (§ 610). Subsequently there are rashes of many different kinds—macular, papular, scaly, pustular and tubercular, but never eczematous or vesicular. They are generalised or symmetrical in distribution, with a preference for the forehead and flexor surfaces, and characteristically *reddish-brown* in colour, *polymorphic* in type, and *never irritate* (§ 645). (ii.) The *sore throat* may be slight, but on the other hand pain on swallowing may be marked. The accompanying erythema is described in § 158. (iii.) " *Mucous patches* " (or " snail-tracks ") are seen on the lips, tonsils, pillars of the fauces, soft palate and uvula, and tend to be symmetrical (§ 158) : similar lesions may also occur at the corners of the mouth and at other mucous orifices, and their secretions are all highly contagious. (iv.) The *lymph glands* near the primary focus become involved as a secondary manifestation, and generalised lymphadenopathy may persist for months or years : the hardness of the glands aids diagnosis. (v.) A low-grade *pyrexia* is often overlooked (§ 514), and may be accompanied by pain in the limbs. (vi.) There is loss of appetite, general malaise and some loss of weight. The hair may fall out and the nail-beds become affected by an indolent inflammation. The liver, spleen and thyroid may enlarge : the eyes may become affected by iritis or choroido-retinitis : the joints with synovitis and the bones with periostitis in which the pain is worse at night. Any of these symptoms may recur again and again in the ensuing months or years.

The *Tertiary* stage is the result of a syphilitic invasion of the various organs of the body during the secondary stage, and indicates that the patient has not been adequately treated in the primary or secondary stages. The organisms may lie dormant for many years before producing noticeable clinical disease. As may be seen by studying the preceding and succeeding pages, almost any organ may be affected. Some of the principal results are as follows : (i.) All the *skin* symptoms noted in the secondary stage may recur, but they are much less profuse and are apt to be asymmetrical in distribution, serpiginous in outline, lenticular or nodular in shape, with a greater tendency to deep ulceration, suppuration or scarring, and to be followed by more loss of tissue. (ii.) Nodular or infiltrating *gummatous deposits* are followed by scarring and perhaps by ulceration. These may involve the mucous membranes (especially in the mouth and naso-pharynx) : the liver, spleen and other abdominal organs : the lungs and mediastinum, the heart, brain and other vital structures. The bones are often attacked by gummatous periosteal deposits, leading in the hard palate and nasal septum to perforation, and in the other flat bones and the long bones to a diffuse periostitis or to more localised " nodes ". (iii.) In the cardio-vascular system, the wall of the thoracic

aorta and the aortic valves are infiltrated and weakened (§ 80) and aneurysm is common: the smaller arteries (especially of the brain) show an endarteritis and may become thrombosed (§ 90). (iv.) The *lymphatic glands* may remain enlarged. (v.) Intermitting *pyrexia* may accompany the formation of gummata (Fig. 122). (vi.) *Lardaceous disease* of the liver, spleen, kidneys or intestine may ensue. (vii.) The principal result of the invasion of the *central nervous system* is often the production 10–20 years later of tabes dorsalis (§ 817), general paralysis of the insane (§ 902), or both (tabo-paresis). (viii.) In a few untreated or malignant cases of syphilis there is cachexia which may be fatal.

Varieties.—In practice, it is convenient to recognise two main types of syphilis. Civilised communities are now more resistant to the disease than a hundred or more years ago, and most cases assume a benign type: in such the influence of adequate treatment during the primary and secondary stages will prevent any tertiary stage developing. In others, and particularly in primitive races not previously exposed to the disease, a more virulent form ensues: this may be enhanced by a debilitated state of the individual, and by inadequate treatment in the earlier stages causing the organisms to develop a resistance to subsequent remedies.

(B). Congenital or "Hereditary" Syphilis is now rare. Syphilis is no longer regarded as a cause of abortion for this is not more frequent in syphilitic than in normal women. On the other hand, it does cause the child to die within the uterus (Still-birth), or, being born alive to die within the first twelve or eighteen months of marasmus. Even so, the greater proportion of syphilitic children pass through infancy with few characteristic clinical lesions. There is no primary chancre in a congenital syphilitic child, and if symptoms arise they conform more or less to the secondary symptoms above described. In the small number of infants who do develop symptoms early in life, at the age of a few weeks "snuffles" occurs, and this so interferes with feeding that it contributes to marasmus. In these there is often a ham-coloured eruption on the buttocks, flexures, palms or soles: the general condition deteriorates, the cry is hoarse, the bones are tender and a fatal gastro-enteritis, bronchitis or pneumonia terminates the picture. Epiphysitis may cause pseudo-paralysis, but in a larger number of children, a symptomless epiphysitis or periostitis, demonstrated only by X-ray examination, is valuable evidence of active disease. About the seventh year the permanent incisors appear, and frequently present a "screw-driver" shape and a notched border described by Sir Jonathan Hutchinson (Fig. 3). At any later date, and even in early adult life, interstitial keratitis, periostitis, synovitis, or *sudden* eighth nerve deafness in one ear rapidly followed by similar deafness in the other ear, may appear. More rarely the skin, viscera and the nervous system are attacked (with the development of juvenile tabes or G.P.I.). Even without symptoms the C.S.F. is often pathological—Table XXXIX. The residual lesions of congenital syphilis persist throughout life as hall-marks (stigmata) of the disease, and are summarised in Table XL.

TABLE XXXIX.—HEREDITARY SYPHILIS.

A. MANIFESTATIONS IN INFANTS (three weeks to twelve months).

Stillbirths are common. When born alive are usually healthy. Soon a small number of infants develop symptoms resembling the secondary stage of acquired syphilis. The lesions are:

I. Mucous membranes { Snuffles and hoarse cry. / Condylomata around anus or mouth.

II. Marasmus, leading to wizened appearance: very marked wasting, often fatal.

III. Skin { Papular, Scaly, Pustular, Bullous, Polymorphic } Always symmetrical, transitory, ham-coloured, becoming deep-brown; on buttocks because of urine and fæces; in flexures because of perspiration. Patches of peeling erythema about the palms and soles, nates, etc. Circum-oral eczema produces rhagades on healing.

IV. Epiphysitis and Periostitis—Tenderness of bones, abscesses, or caries of long bones.

Skull: cranio-tabes, *i.e.*, thinning in one place, thickening in another (now rare).

In many, symptomless periostitis or epiphysitis can be demonstrated radiographically.

V. Large cirrhotic liver.

VI. "White" (interstitial) pneumonia—usually found only at autopsy.

B. MANIFESTATIONS IN CHILDREN AND YOUNG ADULTS— rare before five years of age.

I. Interstitial Keratitis—an acute painful inflammation first of one cornea, then the other. As it resolves appears like ground-glass. Between tenth and twentieth year. Clears up very slowly with treatment, but some scarring remains.

II. Deafness, often in association with interstitial keratitis. Characteristically comes on suddenly with noises in the ears, without pain or otorrhœa. Usually causes complete deafness within 24 hours, and within a week the opposite ear is similarly affected. Treatment has no effect.

III. Periostitis of long bones (rarely skull)—generally causes thickening (sabre-tibia), or nodes, occasionally suppuration.

IV. Synovitis is painless and persistent—knees or other large joints (Clutton's joints).

V. Skin, mucous membranes and viscera fairly often affected at this stage, with lesions of tertiary type.

VI. Nervous system rarely involved (juvenile tabes and G.P.I.). C.S.F. much more frequently gives positive W.R.

The *Diagnosis* of the Hunterian chancre has to be made from the non-Hunterian chancre or soft sore: in the latter pain is a marked feature, the ulceration is deeper, and the edges are softer and less deeply infiltrated. Identification of the *Treponema pallidum* under the dark-ground illumination of the microscope will settle the diagnosis (§ 133). The Wassermann, Kahn and other tests for syphilis (§ 924) are very helpful: they usually become positive in 2–3 weeks after the appearance of the chancre and are always positive in the secondary stage. Even in the presence of active disease, negative results may be met in the tertiary stage, especially in those who have had incomplete treatment earlier. In this case a positive result by examination of the cerebro-spinal fluid denoting neuro-syphilis

TABLE XL.—
HALL-MARKS OF PREVIOUS LESIONS OF CONGENITAL SYPHILIS.

NOTE.—Only a few of these may be present in any individual case. Signs may also be present in brothers and sisters.

I. *Tegumentary System.*	*Skin*—Peribuccal cicatrices radiating from mouth (rhagades). *Eruptions* (rare)—Lupoid ulceration, gradually spreading. *Mucous membranes*—Cicatrices of the throat and palate. Hole in nasal septum or palate.
II. *Bones and Joints.*	*Cranial malformations*—prominent frontal eminences, natiform cranium, asymmetry, hydrocephalus. *Nasal malformations*—" saddle nose," depressed septum. *Tibial deformities*—" sabre-blade " or nodular tibia. *Joint lesions*—Chronic painless effusions (Clutton's joints).
III. *Hutchinson's Triad.*	1. Eye—The remnants of interstitial keratitis (striæ in cornea), old iritis, or choroidal atrophy. 2. Ear—Total nerve deafness. 3. Teeth—Microdontism, "screw-driver" incisor teeth (§ Fig. 3), with central notch in the cutting edge.
IV. *Constitutional Effects.*	Infantile build. Retardation of growth and mental development, of dentition and of puberty.

may be helpful (§ 920). It must not be forgotten that " false positive " results of the Wassermann reaction may be met, especially in and following such conditions as benign tertian malaria, infectious mononucleosis, mumps, measles and other specific fevers of childhood, after the injection of tetanus toxoid and inoculation for small-pox. In these cases positive findings sooner or later revert to negative results without treatment. In any obscure clinical condition, the possibility of a syphilitic cause should be borne in mind : and in the absence of confirmation by examination of the blood or C.S.F., a therapeutic trial should be made with anti-syphilitic remedies.

Etiology.—The specific organism is a feebly staining spirochæte, *Treponema pallidum* (*Spirochæta pallida*). It can be obtained not only from the primary sore, but in abundance from condylomata, and also from the viscera in secondary, tertiary, and congenital syphilis. The organism has a corkscrew shape with from eight to twelve curves (Fig. 132). It is differentiated from a commonly occurring spirillum, the *Treponema refringens*, in that the latter has fewer and less delicate curves.

Syphilis resembles the specific fevers in having a period of incubation followed by a characteristic eruption. One attack renders a person immune to a second attack, except in cases where the first was cured by treatment. It differs from other specific fevers in the extreme length of its course, which may last many years, in the long intervals which may separate its various manifestations, and above all in its liability to recur without fresh infection. Inoculation can only take place through a minor abrasion of the skin or mucous membrane, and may occur in three ways : (*a*) Usually it is by direct contact with an infected person, generally during sexual intercourse ; and in some cases by suckling

(as in wet nurses), kissing, and (in doctors and midwives) as a result of examining diseased persons; (*b*) contaminated articles—*e.g.*, spoons, cups, pipes, towels, or surgical instruments have sometimes been suspected. In the first two stages the blood and the moist exudations of all the lesions are certainly contagious. In the later stages some difference of opinion exists as to the contagiousness of the blood and secretions, but generally speaking contagion is rare after five years from the onset of the disease. (*c*) In bygone days the use of human vaccine lymph, even when free from blood, for vaccination purposes from arm to arm was the occasional means of propagating syphilis, but the frequency of this was certainly exaggerated.



FIG. 133.—(TREPONEMA PALLIDUM) SPIROCHÆTA PALLIDA of SYPHILIS, magnified about 500 diameters. Illustration lent by the courtesy of Colonel Sir W. B. Leishman, R.A.M.C. The organism is of a spiral form like a long corkscrew. The wavy organism on the left is the *Spirillum refringens*.

In the young pregnant woman suffering from early syphilis, the chance of transmission to the child is high (approx. 90 per cent.). When she has had syphilis for five years or more, the infection is attenuated and transmission is much less frequent. It is probably true to say that a father never transmits the infection directly to the offspring and only does so by first infecting the mother who later develops a positive Wassermann reaction. When a pregnant woman with latent syphilis is found to have a repeatedly positive Wassermann test, it is imperative that she should be treated throughout pregnancy, thus ensuring that the child will be born healthy.

Prognosis.—The toxæmia of syphilis is particularly virulent in the fœtus: in infancy it may cause fatal marasmus. Following the secondary stage, untreated adults fall into four groups: (*a*) one-third remain symptomless throughout life, with a persistent positive W.R.; (*b*) one-third are likewise symptomless, with a W.R. which ultimately becomes negative; (*c*) one-sixth develop early tertiary symptoms which abort spontaneously, and (*d*) one-sixth develop and suffer from disease afflicting many different systems of the body. The first two groups are termed "latent syphilis." Unfortunately it is not possible to know beforehand whether the disease will remain latent, or whether it will cause future complications in some vital part. In general, when serial blood reactions become weaker and finally negative, such complications are very unlikely.

The severity and duration of an attack of syphilis are influenced by many circumstances, and particularly by the habits and mode of life (especially intemperance), age, occupation, exposure, privation and pre-existing disease (such as tuberculosis and nephritis). The factor which influences the prognosis of syphilis most is adequate and continuous treatment during its earlier stages.

Treatment is of two kinds, prophylactic and curative. *Prophylaxis* is best carried out within half an hour of exposure by thoroughly rubbing in to the exposed site calomel ointment (33 per cent.) : the ointment should be left in situ for 48 hours. *Curative treatment* should be begun as soon as possible after the diagnosis is made. The most successful results are obtained when treatment is commenced before the secondary symptoms appear, and while the Wassermann test is still negative.

The drugs which have been used are potassium iodide, metallic mercury and bismuth or their salts, the organic arsenical compounds and penicillin. *Potassium iodide* has no lethal effect on treponemata, and is only of value in the tertiary stages : its particular use is in promoting the absorption and healing of gummata in organs such as the skin, liver, spleen or bones. It has the advantage of producing no toxic reactions, other than iodism. Start with gr. 10–20 and rapidly increase to gr. 60 t.i.d. after meals, followed by a glass of milk. If iodide of potassium disagrees, try substituting the sodium, calcium or ammonium salts. *Mercury* was wholly relied on for the treatment of the primary and secondary stages of syphilis before the introduction of bismuth and the arseno-benzol remedies. Now its use has been almost entirely superseded by bismuth and other modern drugs. When used, it is administered by mouth as liq. hydrargyri perchloridi in doses of ℳ30 t.i.d. after meals, gradually increased to ℳ60 t.i.d. : or to children as hyd. c̄ cret. gr. 1–2 t.i.d. The precautions to be observed are : (i.) get rid of decayed teeth and see the gums are healthy at the outset : (ii.) the patient should be seen once a week, to watch for salivation, diarrhœa, gastric disturbance : (iii.) patients with renal or visceral disease require smaller doses and extra caution. *Bismuth* given intramuscularly has replaced mercury. Insoluble preparations are mostly employed as suspensions of metallic bismuth or of bismuth oxychloride : the doses are 0·1–0·2 G. once a week. Administer deeply into the muscles of the buttocks : in all cases insert the needle first, then slightly withdraw the piston of the syringe to make certain a vein has not been entered : finally inject the dose slowly. Bismuth, like mercury, shows with oral sepsis a deep purple line along the gums, and with poisoning a stomatitis with ulceration of the gums and buccal mucous membrane, with enlarged cervical glands. Albuminuria, loss of weight and sleeplessness may occur. Ehrlich introduced the *organic arsenical compounds* in 1910 when he discovered salvarsan or " 606 " (arsphenamine B.P.). This trivalent arsenical substance was soon replaced by another in the form of neosalvarsan (neoarsphenamine B.P.). These and related compounds are not directly bactericidal to the treponemata but are stored chiefly in the liver and, in the presence of oxygen, slowly liberate arsenoxides which are the lethal agents. Arsphenamine and neoarsphenamine are of curative value in all three stages of syphilis, and if given early the secondary stage may be prevented or aborted. Administered only by the intravenous route, great care must be taken that none of the solution enters the perivenous tissues as it can produce a severe chemical cellulitis and even necrosis. In any case, they can prove profoundly toxic to elderly and debilitated subjects. To prevent reactions after an injection, it is well to give a carbohydrate meal or glucose shortly before it, not to inject after a heavy meal or after ingestion of alcohol, nor when there is constipation. Great care must be taken when treating patients with diseases of the liver, kidney or cardio-vascular

system: in such cases first give potassium iodide for at least a month, and then use a course of intramuscular bismuth.

A course of neoarsphenamine usually consists in giving initially 0·15–0·30 G. to an adult, increased by weekly increments to a maximum single dose of 0·75 G., this being repeated until a total of 4–6 G. has been given. Certain untoward results of therapy may be mentioned here: (1) Within a few minutes after an injection symptoms resembling anaphylaxis may arise (nitritoid crisis); adrenalin ℳ 10 controls this condition. (2) Some cases react a few hours after an injection with fever and rigors: these symptoms are less common since using sterile redistilled water for dissolving the drug. (3) The Jarisch-Herxheimer reaction is due to an effect in the blood vessels at the site of the treponemata: there is believed to be a sudden liberation of their toxins, with œdema and local inflammation causing narrowing of the blood vessels, and even thrombosis. With this there is a sudden increase in symptoms (*e.g.*, of headache in cerebral syphilis) and with cardio-vascular syphilis involving the mouths of the coronary vessels, angina or left ventricular failure may occur. (4) Arsenical hepatitis is probably due more often to an associated virus than to the arsenical compound (§ 332). (5) Other toxic effects include erythema, dermatitis and exfoliation, acute encephalopathy, purpura and aplastic anæmia. When these toxic complications are met, give British-anti-lewisite (B.A.L.): start on the first day with 2 c.c. at four-hourly intervals, on the second and third days give the same dose twelve-hourly and on the two following days give the dose once each twenty-four hours. In children, and when intravenous therapy is difficult, sulpharsphenamine is given intramuscularly. In view of its greater permeability into the brain, a pentavalent compound (tryparsamide) is usually preferred in cerebral syphilis G.P.I.).

Since the arsenoxides are the active substances which destroy the treponemata, modern use has been made of *mapharside* (arsphenoxide B.P.) in the treatment of syphilis. On account of its rapid excretion, two or three doses of 40–60 mgm. have to be given each week. Experience is proving this substance equal in value to the arsphenamines. *Penicillin* by deep subcutaneous injection is likely to prove the most effective single curative agent. In primary and secondary syphilis it will cure most cases and render the Wassermann reaction negative, so long as it is given in adequate doses. In tertiary disease it is less effective but still of great value: in neuro-syphilis (tabes and G.P.I.) the results are encouraging if given in very large doses—not less than 10 mega-units in two weeks. Jarisch-Herxheimer reactions occur in 50 per cent. of cases in the primary and secondary stages and consist of increased signs of inflammation in the primary sore, accentuation of a secondary rash, pyrexia and adenitis—but these effects should be neglected unless very severe. In tertiary disease, the reactions may be more serious at the commencement of the first course of treatment, and should be guarded against by a preliminary course of intramuscular bismuth injections. When a patient is being treated for gonorrhœa by penicillin, the occurrence of such Herxheimer reaction with general symptoms should be regarded as suggestive of a coincident syphilitic infection.

In practice, treatment is most effective when at least two or three of these remedies are given simultaneously: these drugs then have a synergic therapeutic effect. The usual combination is of penicillin, with neoarsphenamine and bismuth, and the treatment advised by V. E. Lloyd is as follows. For *primary and secondary syphilis* he gives a two months' course of treatment: for the first eight days 600,000 units of penicillin in oil-wax, or of procaine penicillin once a day (total 4·8 mega units): on the second day 0·3 G. neoarsphenamine, on the sixth, ninth and twelfth days 0·45 G., followed by 6 weekly doses of 0·6 G. (total 5·25 G.): and in addition bismuth oxychloride 0·2 G. weekly for 8 weeks (total 1·6 G.).

He advises a Wassermann test once a month, and if it remains strongly positive at the end of 3 months, a repetition of this course of treatment.

In *tertiary syphilis* Lloyd points out that it is more important to treat the patient than the disease, and due regard must be had to the condition of the heart, liver, lungs and kidneys. Much harm may be done by injudicious intensive treatment. In general, he advises that when infection has probably been present for less than ten years, and the patient is otherwise healthy, there should be a four-week course of pot. iodide (gr. 10 t.i.d.) with a weekly dose of bismuth (0·2 G.), followed by a full course of treatment as laid down for the primary and secondary stages. When infection has been present for more than 10 years, and the patient is under 60 years and in good health otherwise, he uses a four-week course of pot. iodide and bismuth (as above), then 600,000 units of penicillin in oil-wax or procaine penicillin daily for 8 days, followed by 8 weekly injections of neoarsphenamine (0·45–0·6 G.), and then 8 weekly doses of 0·2 G. bismuth. For the benign lesions of the skin, mucous membranes and bones in later life, he advises pot. iodide (grs. 10–20 t.i.d.) and bismuth (0·1 G. weekly), for four weeks, and then pot. iodide and bismuth with penicillin 250,000 units in oil-wax or procaine penicillin once a week for a further 4 weeks. The positive serological tests in later life will remain unaffected by any form of treatment. Neuro-syphilis as indicated by positive C.S.F. findings needs still more intensive treatment.

It is very important to decide when marriage is permissible: V. E. Lloyd advises postponement until treatment has been completed, and all tests (including lumbar puncture) have been satisfactory for 3 years.

In *congenital syphilis*, a positive W.R. in the blood of the umbilical cord may reflect the state of the mother's blood, and if the strength of the reaction decreases in the course of the next few months, to become negative at the fourth–sixth months, active treatment is not required. But if the reaction remains positive, or becomes more so, or if symptoms of infection are present, then treatment is immediately necessary. Mercury with chalk (gr. ½–1 a day) and mercurial inunctions are being superseded by more modern remedies. Penicillin rarely causes troublesome Herxheimer reactions. Owing to the difficulty of intravenous treatment, neoarsphenamine is replaced by intramusc. injections of sulpharsphenamine (B.P.): and bismuth injections can also be given intramuscularly. In general, and after three months of age, the maximum weekly dose of sulpharsphenamine is 0·005 G. per lb. body-weight. *At birth*, Lloyd advises starting with penicillin (1,000 units per lb. body-weight in each injection) four-hourly for 2 weeks. He follows this by sulpharsphenamine (0·005 G. increasing the weekly doses to 0·050 G. at the end of 8 doses), and then by 8 weekly doses of metallic bismuth into the buttocks (0·020 G. per dose). *At one year*, for a child of average weight, penicillin injections are given in the same doses, but at three-hourly intervals, for 2 weeks. Then an eight-week course of sulpharsphenamine is given weekly in doses of 0·025 G. increased to 0·10 G. by the eighth dose. After a few weeks'

rest, 8 weekly doses of bismuth metal (0·04 G.) follow. At *5–6 years* the maximum dose of sulpharsphenamine is 0·30 G. and at *10–12 years* 0·45 G. The C.S.F. should be examined if the serological tests of the blood are still positive after two such courses of treatment.

§ **553. II. Plumbism** (Synonyms: Lead Poisoning, Saturnism). Chronic ill-health, usually associated with a number of other symptoms, results from the slow absorption of lead into the system.

Symptoms.—(1) Pallor is very marked, due to vascular spasm rather than to anæmia. The pale pasty appearance of lead workers who are insufficiently protected is well known: with this general asthenia and loss of weight are usual. (2) Attacks of severe intestinal colic are accompanied by obstinate constipation. An acute attack of colic may be associated with general malaise, a rise of temperature and some degree of abdominal tenderness and rigidity: during the attacks the pulse is slowed. The presence of recurrent attacks of colic is of diagnostic aid. (3) Nervous symptoms: lead has a special tendency to attack the peripheral nerves, and especially the motor cells and fibres: the musculo-spiral nerves are most commonly affected, leading to bilateral wrist-drop, but the supinator longus muscles escape. Sometimes the paralysis is generalised. Otherwise the brain cells may be involved (saturnine encephalopathy): neurasthenia is common and in typical cases, severe headache, acute mania, and convulsions occur. The mental condition may deteriorate until the patient is unable to look after himself. In others, optic neuritis and various degrees of amblyopia may occur: tremor is often present without paralysis, especially if the lead has entered the system by inhalation, as in glass-blowers. (4) Chronic renal changes may ensue, leading to chronic interstitial nephritis and uræmia. With this, an advanced degree of arteriosclerosis may be met, leading to fainting fits, and even hemiplegia. (5) In those with teeth, the gums show a punctate " blue line " close to the gum margin: this is much more marked when dental sepsis is present, permitting the formation of hydrogen sulphide, with the deposition of lead sulphide: a hand lens is of help in identifying the punctate character of the blue line. A similar line is met in those receiving bismuth injections: in copper poisoning the line is brighter and more greenish. (6) Punctate basophilia (§ 531) in the red cells is present in anyone in contact with lead: when the number of red cells showing this stippling shows a sharp and persistent rise, toxic symptoms are likely to ensue. (7) In chronic cases, and particularly in women, marked secondary anæmia, and a relative lymphocytosis of the white cells, may be found.

Diagnosis.—Vague ill-health, pallor, the blue line, recurring colic and nervous symptoms occurring in a lead worker are diagnostic. Lead poisoning due to cosmetics, toys, etc., has, in some cases, been unsuspected till too late.

Etiology.—Some appear to be immune; women are more susceptible than men. Alcoholism, anæmia, gout, renal disease and acute infections render individuals more liable. Lead may enter the system by the mouth, the lungs or the skin. (1) Epidemics have been caused by water contaminated with lead, when stored in lead cisterns; soft water and slightly acid water has dissolved some of the lead carbonate found inside leaden pipes. Poisoning has occurred with food contaminated with lead, when stored in lead-lined or pewter vessels, and with drink, such as beer or cider which has lain several hours in a pewter or leaden pipe. Other causes are lead used for abortion, sucking paint off toys, sleeping in newly painted rooms, hair dyes, grease paint and face powders containing lead. (2) Lead is still more poisonous when its dust or fumes are inhaled, as occurs with painters, pewterers and those exposed to fumes of melted lead, and lead glaze workers. (3) Lead can be absorbed through the skin, as with tetra-ethyl petrol. Possibly some of the cases due to face powders and grease paint enter by the skin.

Treatment.—The first indication is the avoidance of the cause. Those who are exposed to the poison by reason of their occupation should observe the greatest personal cleanliness; the face, hands, and teeth should be cleansed before meals.

The ventilation of the workroom should be supervised, and a respirator worn if the air contains much dust. In acute cases give rest in bed, warm external applications, castor oil and sulphate of magnesium. Sir Thomas Oliver recommended gr. ½ monosulphite of soda when abdominal pain is continuous and there is tenderness on pressure. Calcium gluconate given intravenously (10 c.c. of 20% solution) repeated in three and six hours will arrest severe lead colic. In the acute stage, one aims at removing the lead rapidly from the blood, and storing it in the bones as an insoluble salt. For this purpose utilise the antagonism between lead and calcium salts by giving a high calcium diet (milk, eggs, green vegetables, with calcium gluconate gr. 60 t.i.d.). After the acute toxic symptoms have subsided, try to eliminate the lead by giving a low calcium diet, together with ammonium chloride (gr. 120 to gr. 240 during the day) or parathormone injections (15–25 units t.d.s.).

III. **Acute and Chronic Renal Disease** are usually accompanied by a pallor which may readily be mistaken for primary anæmia. This is especially the case in parenchymatous nephritis, which is apt to affect young people. The pallor is of an ivory whiteness, is usually accompanied by a certain amount of dropsy, and the urine reveals a definite amount of albumen and tube casts. Chronic interstitial nephritis usually occurs in older people; it is generally attended by sallowness, but progressive asthenia is its more constant and striking symptom.

IV. **Chronic Liver Disease**, and especially cirrhosis of the liver, may be attended by an anæmic pallor; but it is usually attended also by dilatation of the venous capillaries in the face, which are very characteristic (§ 342).

V. **Latent Tuberculosis** is generally attended by pallor, weakness, and loss of weight (§ 131). The pallor is often very marked and gives rise to a diagnosis of anæmia which is not supported by the blood examination. If pallor and general debility in a young patient do not respond to iron and general tonic measures, tuberculosis should be suspected. The disease may be latent in the sense that there are no obvious clinical manifestations, *e.g.*, no abnormal physical signs to be found in the lungs or elsewhere. It is useful to remember that an active tuberculous process, no matter where it is situated, is nearly always attended by pyrexia, usually of an intermittent type; this fact is apt to be overlooked (see § 512).

VI. **Gastro-intestinal conditions** and especially simple dyspepsia, constipation, colitis and other disorders of the alimentary canal frequently come under our notice for pallor, and are often associated with secondary hypochromic or microcytic anæmia. Indeed, dyspepsia and confinement indoors are perhaps the commonest causes of pallor among town-dwellers. Focal sepsis, as in apical abscesses, sinus disease, and pyorrhœa alveolaris may cause intense anæmia. Deficient food, and particularly deficient nitrogenous food, may also act in a lesser degree. In colitis and other intestinal conditions with much intestinal dyspepsia, pallor may be noticeable. The pallor is of a peculiar kind, the skin loses its lustre, and there are dark rings under the eyes. In some of these patients the cæcum is loaded and abnormal intestinal flora are found. It is wonderful what an improvement in the appearance of such cases is made after a course

of treatment by intestinal antiseptics or *B. acidophilus*, combined with suitable diet, purgatives and colon douches.

VII. Certain **Cardio-vascular conditions.** Malignant endocarditis produces pallor, caused by the associated anæmia. Pallor also occurs in one type of hyperpiesis (§ 94), in acute pericarditis (§ 46) and endocarditis (§ 49) and in aortic valvular disease.

VIII. Finally, in various conditions referred to in Group II (Emaciation), or Group III (Debility) (p. 729), Pallor may be the symptom which first attracts our notice. ADDISON'S DISEASE is one of these: another is early MYXŒDEMA (§ 559) and the puffiness of the eyes, the failing memory, loss of hair, and bodily weakness may for a time escape observation, or be attributed to other causes. Septic foci, especially SEPTIC TONSILS, are often accompanied by a greyish pallor, even when there is no true anæmia. MULTIPLE MYELOMATOSIS (§ 598. X) also may first come under notice for anæmia.

GROUP II. EMACIATION

WASTING is a common sequence of nearly all acute and many chronic diseases, but when it is the leading or only symptom the following morbid conditions should be borne in mind; for fallacies see § 525.

COMMON CAUSES.

Young and Middle Age.	*After Middle Age.*
I. Tuberculosis.	X. Malignant disease.
II. Diabetes Mellitus.	XI. Senile decay and arterial disease.
III. Hyperthyroidism.	XII. Chronic nephritis.
IV. Digestive disorders.	
V. Focal sepsis.	
VI. Diseases of nervous system.	

RARER CAUSES.

VII. Simmonds' disease.
VIII. Drugs.
IX. Other rare causes described elsewhere include diabetes insipidus (§ 419), pancreatic diseases (§ 256), pernicious anæmia (§ 539), lymphadenoma (§ 572), cirrhosis of the liver (§ 342), syphilis (§ 552).

Marasmus in children may be caused by defective feeding, diarrhœa, constipation, persistent vomiting, hereditary syphilis, rickets, certain nervous states, tuberculosis of the lungs, lymphatic glands or of the peritoneum. These are discussed in §§ 556 *et seq.*

§ 554. I. TUBERCULOSIS is often first evidenced by an apparently causeless loss of weight. In latent tuberculosis the trunk and limbs may be wasted although the face be plump and rosy. There is a daily rise in temperature, tendency to night sweats, and careful search will usually reveal an active focus in the lungs (§ 131), peritoneum (§ 557), Fallopian tubes (§ 450), or in the lymphatic glands (§ 571).

II. DIABETES MELLITUS in the young may even impress the patient by the inconsistency of the ravenous appetite and constant thirst with

the loss of weight. On the other hand, some middle-aged patients with diabetes are well-nourished and even obese.

III. HYPERTHYROIDISM is usually recognised in the young when we meet the combination of exophthalmos, restlessness and nervousness, tachycardia and full thyroid; but in elderly and latent cases the diagnosis may be much more difficult (§ 186).

IV. DIGESTIVE DISORDERS and DEFECTIVE FEEDING. Perhaps the commonest causes of slight loss of flesh met with in practice are defective feeding and digestive disorders. Digestive disorders may, of course, exist without any wasting. In younger persons, loss of weight may be due to purposeful reduction by eating insufficient or non-nutritious food. Defective feeding, and particularly deficiency in the fats, carbohydrates, and in foods containing vitamins, may without any digestive disorder be attended by emaciation. *Defective teeth* frequently cause both digestive troubles and loss of flesh. Various INTESTINAL CONDITIONS are often attended by undue spareness of body. Among the latent causes of this may be mentioned obscure intestinal conditions such as *colitis*, *worms* and *amœbiasis*, which may long be overlooked. Vomiting or diarrhœa of any origin cause rapid wasting, especially in children.

V. FOCAL SEPSIS may cause emaciation due to long continued absorption of toxins from defective teeth, tonsils or other hidden foci, or by producing periodic blood stream infections causing bacteræmia or septicæmia.

VI. DISEASES of the NERVOUS SYSTEM may start with or present generalised wasting, as in bulbar paralysis and paralysis agitans. Wasting due to disappearance of muscular tissue alone, at first localised and later generalised, occurs in *motor-neurone disease*, and in *myopathy*. The psychological causes of emaciation are extremely varied and common. Wasting occurs in *anxiety neuroses*; such cases do not show the raised basal metabolic rate of hyperthyroidism. *Refusal of food* and consequent starvation is a symptom of hysteria and various psychoses. A variety of this condition is *selective food faddism*, where the patient believes herself incapable of digesting certain foods which are consequently eliminated from the diet. One such patient, troubled with flatulence, lived for years only on preserved ginger and peppermint creams, until scurvy developed. In *anorexia nervosa* (§§ 273, 888) the patient, usually an adolescent girl, refuses food because of lack of appetite. Amenorrhœa is present and emaciation may be extreme, but the signs of premature senility seen in Simmonds' disease are absent. The condition may be hysterical or a symptom of an oncoming psychosis such as *schizophrenia*. Some of the cases develop pulmonary tuberculosis. In the variety of manic-depressive psychosis known as *cyclothymia* the patient may lose weight during the periods of excitement, whilst in the alternating depressed phases menstruation is abolished and the patient's weight increases.

RARER CAUSES

VII. **Simmonds' disease** is caused by lack of development or atrophy of the anterior lobe of the pituitary, with deficiency of hormones necessary for nutrition and

growth, and of the gonadotropic and thyrotropic hormones. It is more common in females, especially after post-partum hæmorrhage. *Symptoms.*—There is extreme emaciation with a wizened appearance, little subcutaneous fat and a dry wrinkled skin. In an *adult* there is often asthenia, amenorrhœa, loss of pubic and axillary hair, and atrophy of the breasts, ovaries and uterus. In a *child* puberty is absent or much delayed, growth in height ceases and wasting occurs. In both types there is usually an increased sugar tolerance with signs of myxœdema, such as cold extremities, constipation, and a slow pulse rate with a low basal metabolic rate. Achlorhydria and anæmia of secondary or pernicious types may occur. *Progeria* is an extreme condition of Simmonds' disease occurring in a child or young adult, associated with premature arterio-sclerosis and loss of hair, teeth and nails. After several years death usually occurs, with lethargy and coma. *Treatment* by anterior pituitary injections should be pushed.

VIII. DRUGS may act by depressing the mental or physical faculties or by impairing digestion. Drug addicts (*e.g.*, morphia, heroin, cocaine) are almost invariably emaciated. Recently drug addiction to thyroid and amphetamine have been recorded. The later stages of chronic alcoholism may produce marked loss of weight.

IX. Other rare causes such as DIABETES INSIPIDUS, PANCREATIC DISEASES, PERNICIOUS ANÆMIA, LYMPHADENOMA, CIRRHOSIS of the liver and SYPHILIS, are described in their respective paragraphs.

Localised absence of fat occurs in the rare condition called PROGRESSIVE LIPODYSTROPHY. Fat disappears from the face, arms and upper body and accumulates over the lower trunk, hips and lower extremities (§ 16).

In elderly subjects, although any of the above causes may be in operation, suspect especially :—

§ 555. X. **Malignant Disease** (Carcinoma and Sarcoma) is a cause of emaciation which should always be considered when the patient is at or past middle age. The essential features are the tendency of the growths to recur after removal, to invade neighbouring parts and to metastasise in distant parts. *Carcinomata* may arise from any epithelial or mucous surface: common primary sites are the skin, tongue, œsophagus, colon, rectum, breast, uterus and penis. Carcinoma may be latent when it arises in the thyroid, prostate, testicle, pancreas, stomach or colon. *Sarcomata* arise from tissues of mesodermal origin, especially from lymph glands, fascia, bones, the corium, ovary, kidney in children, brain, spinal cord, retina, and in the fibrous structures of the muscles, breast and testicle. A carcinoma usually starts after the age of 40, whereas a sarcoma may attack children or young adults. Malignant growths vary considerably in their malignancy: with carcinomata, the softer or encephaloid types are rapidly growing, while the harder or scirrhous types are much slower. Among sarcomata the melanotic and round celled forms are very malignant, whereas fibro-sarcomata are the slowest.

Symptoms.—(1) The initial symptoms may depend on the *primary site of the disease.* Thus the patient may first seek advice for (i.) a lump (as in the breast); (ii.) pain (from local invasion); (iii.) a continuous blood stained discharge (*e.g.*, from the uterus): or (iv.) symptoms and signs of obstruction (*e.g.*, of the colon or pylorus). The diagnosis of these has been considered already (the abdomen, § 263; the chest, §§ 81, 138). (2) In

other cases the first evidence of the disorder may be from the *disorganisation produced by metastases.* Carcinomata spread *via* the lymphatic system. They tend to invade (i.) neighbouring glands; (ii.) liver, often with hard, tender nodules and producing obstructive jaundice; (iii.) the serous sacs, producing serous effusions (often bloodstained) in the peritoneum or pleura, or (iv.) bones. Cancer of the breast, thyroid, kidney, prostate or ovary also invade the blood stream and may first be recognised by a constant pain in the back from metastases in the vertebrae, or by a spontaneous fracture. (v.) Carcinoma of a bronchus often first declares itself by the occurrence of a metastasis in the brain. Sarcomata spread by the blood stream, with metastases especially in the lungs, liver or bones. (3) Or the patient may first seek advice for the *symptoms of general debility.* In any case this develops sooner or later. Common symptoms are (i.) loss of general energy and strength, (ii.) loss of appetite, (iii.) loss of weight. With this the appearances of cancerous cachexia occur: the patient looks ill, the skin assumes a yellowish, earthy or sallow hue and becomes inelastic. Sooner or later anæmia develops, and often an irregular temperature, especially when the liver is involved.

Diagnosis.—Malignant disease may have to be diagnosed from all the other conditions which give rise to emaciation. (1) A thorough clinical examination must be carried out in every case, including the rectum and pelvic organs. A malignant nodule may have to be diagnosed from syphilitic gumma (compare, for instance, syphilis of the tongue, skin, etc.), but the latter is usually attended by less pain and constitutional disturbance, and disappears on giving potassium iodide. (2) In any case of doubt, exploration or a biopsy must be carried out in order to ensure early diagnosis and treatment.

The *Prognosis,* if the case is untreated, is always very grave, the course rarely lasting more than one, or, at the outside, two years. A few cases of undoubted malignant disease have undergone spontaneous involution. The prognosis largely depends upon the stage at which the true nature of the case is detected. On this depends very largely both the prospect of arrest or removal. In general terms the prognosis also depends on (1) the position and accessibility of the growth, how far vital structures are involved, and whether it is on or near the surface; (2) the structure of the tumour (*vide supra*); and (3) the age of the patient, for growth is more rapid in the young.

Etiology.—(1) In carcinoma the age of the patient is nearly always over forty, though I have seen cases of scirrhus of the pylorus in persons aged twenty-eight and thirty-three. Sarcoma, on the other hand, may affect children or adults of any age. Sarcoma is the commonest malignant growth of the kidney that is met under the age of nine. The cause of cancer has so far defied investigation. The Cancer Committee summarised the various lines of inquiry and their results. (1) The virus theory. (2) The embryonic theory, which holds that small islands of cells in fœtal life are isolated and in later life take on independent

growth. (3) The theory that cancer cells revert to the type of division peculiar to sex cells. (4) Virchow's view that chronic irritation plays a large part in the causation of cancer. In experimental work the last-named has produced positive results. The irritants known to lead to cancer are (i.) certain animal parasites are often followed by cancer of the area they infect; (ii.) chemical irritants such as gas tar, arsenious acid, unrefined paraffin oil, soot. Malignant growths share the characteristic of young and embryonic tissues of possessing a higher potassium and a lower calcium salt content. Experiments with vitamins and various forms of diet have been disappointing.

Treatment.—Early removal is still the most reliable method. Intensive X-ray therapy and radium claim cures in early cases, even of deep-seated tumours. Methods which have succeeded in individual cases are: injection of Coley's fluid, colloidal copper, gold, lead and selenium. Metallic injections have aided where leucopenia was not marked. In cancer of the prostate, intensive œstrogen therapy can abolish pain and apparently hold the disease in check: similar treatment has proved beneficial in some cases of cancer of the breast. The injection of radio-active substances is on trial.

XI. SENILE DECAY and ARTERIAL SCLEROSIS. Many people in their later years lose flesh, especially as their mental and physical faculties become impaired. Others gain flesh as a result of enforced idleness and immoderate appetite (and see § 558).

XII. CHRONIC NEPHRITIS may be an unsuspected cause in an elderly man until the urine is examined (§ 399). The associated nausea and vomiting of the later stages add to the wasting, and the rapidity of the loss of weight is a fair guide to the prognosis.

§ 556. **Marasmus in Children.**—Infants and children emaciate with almost any disorder and with surprising rapidity. An attack of diarrhœa may give rise to loss of much weight in twenty-four hours. The principal causes are:

(*a*) Those which occur chiefly under two years of age: Defective or improper food or feeding; those associated with diarrhœa or constipation; those associated with persistent vomiting; hereditary syphilis; and rickets.

(*b*) Those which are met with chiefly after two years of age: Nervous causes, certain constitutional conditions, and tuberculosis of the lungs, peritoneum, or lymphatic glands.

I. DEFECTIVE FEEDING is a common cause of emaciation. Such children are always fretful, underweight, late in sitting up and teething, with poor muscular tone and often anæmia. The bowels are irregular, constipation often alternating with diarrhœa is common, and the stools furnish a good index as to the chief defect in assimilation present—undigested "curds" of protein, greasy masses of soaps and fats, or green and explosive stools due to excess of carbohydrate. Breast milk may be too plentiful or too little, or when breast feeding is continued beyond the

ninth month iron and other deficiencies occur. About the fifth–sixth months it is usual to commence mixed feeding to supplement breast or artificial milk foods. For further information special books on the feeding of infants must be consulted. In older children the diet is often unbalanced : thus the poorer child usually has too much sugar, white bread and biscuits and too little milk. The wealthy child has often too much fat (butter, milk and cream) and too little carbohydrate.

II. Diarrhœa and Constipation are potent causes of wasting in infancy and childhood ; they are due to dietetic errors or defective cleanliness in the nursery and may be associated with intestinal worms. Infantile diarrhœa is fully discussed in § 307. Chronic constipation undoubtedly causes marasmus. In a family with which I was well acquainted two children died of marasmus associated with obstinate constipation ; the third child who, the mother stated, exactly resembled the others in all particulars, was following the same fatal course until systematic treatment by an aperient mixture restored the health.

III. Persistent Vomiting may be due to errors of diet, especially too frequent or over-feeding, or to gastro-intestinal catarrh. Careful dieting cures most cases. The reflex and other causes of vomiting (§ 271) must be considered when simple treatment is unavailing. In intractable cases feeding by a nasal catheter has been resorted to. Hypertrophic stenosis of the pylorus is a rare cause of vomiting in infants (§ 271).

IV. Hereditary Syphilis is recognised by snuffles and other signs which appear generally during the first six months (see § 552).

V. Rickets (§ 596) may be accompanied by wasting, but is more common in children who are overweight. It appears between the sixth and the eighteenth month of life ; rarely after two years of age, unless associated with renal or cœliac disease (§ 307).

In *older children*, in addition to the above-mentioned causes which may continue to operate, wasting is due to VI. nervous causes. A child in an unhappy environment, or who inherits a nervous disposition, is usually thin, restless, and subject to gastro-intestinal attacks. Dr. Cameron has drawn attention to a well-known type—the child full of energy, soon exhausted, and subject to attacks of prostration with pallor, irritability, even vomiting and fever. These children apparently have a defective fat metabolism and are in better health when the fat in their diet is reduced and the sugar increased. Cyclical vomiting is a closely allied condition (§ 271).

VII. Constitutional Conditions such as asthma (§ 127), cœliac disease (§ 307), recurrent bronchitis, hereditary syphilis (§ 552), as well as congenital abnormalities, often cause wasting.

VIII. Pulmonary Tuberculosis is usually insidious in onset as in adults. It more commonly affects the bases than the apices and causes a chronic cough, persistent pyrexia and wasting. Tubercle bacilli can usually be detected in sputum, from stomach washings or in the stools.

§ 557. IX. **Tuberculous Peritonitis** is a wasting disorder occurring for the most part in children of two years and upwards, due to tuberculosis of the peritoneum and the mesenteric glands. This form was formerly known as tabes mesenterica.

Symptoms.—The onset is very insidious, and may extend over many months. Gradually the limbs and face become shrunken, and there are anæmia, listlessness, attacks of pyrexia, and sometimes abdominal cramps. The leading physical sign is the enlarged abdomen, which is generally tympanitic on percussion. There are three main types: (i.) the ascitic, (ii.) the fibro-caseous adhesive, and (iii.) the loculated type, which is a combination of the first two. (i.) In the ascitic variety, the patient complains of little pain; gastro-intestinal symptoms may be absent, but ascites is present. Ascites unaccompanied by anasarca in a young adult is usually due to tuberculous peritonitis. A sample of fluid may be withdrawn with a needle, and shows an excess of protein and of lymphocytes, and guinea-pig inoculations often confirm the diagnosis. (ii.) In the adhesive variety, there is matting together of the peritoneum and intestines; this may be localised or generalised. Attacks of diarrhœa or constipation occur, perhaps with signs of intestinal obstruction. Pain and tenderness may be marked features, and localised thickenings and masses with a doughy feeling can be palpated. The rolled-up omentum often forms a tumour stretching across the upper abdomen below the edge of the liver. (iii.) In the loculated variety, matting occurs, with encysted fluid in the centre. The hectic fever so common in tuberculosis may be present, and sometimes the disease runs a more acute course with pyrexia, resembling typhoid fever, from which it can only be differentiated by the Widal reaction. (iv.) Tuberculosis of the ileo-caecal group of lymph glands causes general ill-health, and may cause local pain and swelling often confused with appendicitis.

Diagnosis.—In addition to the diseases just mentioned, tuberculous peritonitis may have to be distinguished from the distension of the bowels due to improper feeding, in which there is generally no pyrexia, no resistant masses, and disappearance on regulating the diet. X-ray may reveal calcification of the tuberculous masses in chronic cases. The Mantoux test is strongly positive. *Rickets* (§ 596) may show a distended abdomen, but the characteristic rachitic changes in the skeleton differentiate it. In *Hirschsprung's disease* large enemata bring away the masses palpable through the abdominal wall (§ 317). *Cœliac disease* (see § 307).

Prognosis.—The course of the malady is apt to be irregular, with intervals of apparent recovery, followed by relapses. The ascitic type usually does best. Sometimes the glands undergo a fibroid change, and what appear to be the most unlikely cases recover. Among the untoward symptoms are acute local pain and tenderness, indicating peritonitis; constant diarrhœa, indicating ulceration of the bowels; and evidences of tubercle elsewhere. The complications are numerous—ulceration of the intestine, with pyrexia and intractable diarrhœa; general tuberculosis; abscesses bursting in various situations, such as into the peritoneal cavity or from the umbilicus, the latter forming a chronic fistula. Intestinal obstruction may result at any time from the formation of bands of adhesion.

Etiology.—Tuberculosis of the mesenteric glands is usually due to the ingestion of infected milk: it may occur at almost any age, but is rare under two. Males are affected more than females. If the mucous membrane of the alimentary canal is diseased, the risk of infection is greater. The miliary type with ascites may be of systemic origin. Other varieties arise by extension from tuberculous enteritis, salpingitis, ileo-cæcal or mesenteric gland tuberculosis.

Treatment.—Prophylactic measures consist in sterilising or pasteurising the milk, and regulating the supply whence it is obtained. The disease is now much less common in Britain than formerly, since pasteurisation of milk has been generally adopted. Many cases do well with absolute rest, open-air, a regulated diet and properly applied heliotherapy. Dr. A. Rollier's work on this subject must be studied. Sir Henry Gauvain gives the following instructions for the sunlight available in this country:—

1st day	.. .. Expose legs to knees for	5	minutes hourly for three successive hours.
2nd „	.. .. „ „ „ „ „	10	
3rd „	.. .. „ „ „ groins „	10	
4th „	.. .. Expose legs and buttocks for	20	
5th „	In addition one aspect of the trunk for five minutes.		
8th „	Total exposure for 20 minutes hourly for three successive hours.		

Uncomplicated cases do well if they pigment well. X-ray treatment does good in the fibrous adhesive types. In the ascitic variety, open operation and swabbing out the fluid is often curative.

GROUP III. DEBILITY ONLY (ASTHENIA)

The causes of debility not necessarily accompanied either by pallor or emaciation are as numerous as those of the two preceding groups, and it must be remembered that all the disorders in both of those groups may commence with weakness only; in short, the majority of chronic disorders begin with debility. After any acute illness debility is present; this is usual after fevers, and is especially marked after influenza. The fallacies (§ 525) and methods of examination have already been given.

It should be remembered that profound fatigue may be complained of for a few days to a week before the onset of any acute disease. The same symptom may be complained of, before any sign is evident, in cases of tonsillitis or even of the "common cold."

COMMONER CAUSES

I. Senile decay and arterial disease.
II. Disease of the Nervous System, functional or organic.
III. Chronic infections. Focal sepsis, tuberculosis and syphilis.
IV. Chronic dyspepsia and obscure abdominal diseases.
V. Chronic interstitial nephritis.
VI. Conditions referred to in Groups I and II, and especially the early stages of Diabetes Mellitus, Hyperthyroidism, Anæmia of any origin (especially Pernicious Anæmia) and Leukæmia.
VII. After shock or operation, or after the specific fevers.

RARER CAUSES

VIII. Myxœdema.
IX. Addison's disease.
X. Hæmochromatosis.
XI. Diseases of the pancreas, acromegaly, myelopathic albuminuria, beri-beri, pellagra, alkalosis, hypoglycæmia.
XII. Carbon monoxide poisoning.

When a patient is suffering from debility or loss of vigour of mind and body without any very marked pallor or obvious loss of flesh, and without any marked physical signs or other evidences of disease, in the *first half* of life one would suspect neurasthenia, chronic dyspepsia or gastro-intestinal disorders, incipient or latent tuberculosis, diabetes.

In the *second half* of life one would suspect senile decay, chronic interstitial nephritis, focal sepsis, cardiac valvular or aortic disease, diabetes, myxœdema, Addison's disease. And failing these, some of the conditions previously mentioned among the anæmic or wasting disorders (Groups I and II).

§ 558. I. Senile Decay and Arterial Disease.—As we advance in years the power both of body and mind notably declines. This should not be

very obvious under sixty, but the age at which it appears differs considerably in different persons, and still more in different families, for the onset of decay in human beings, as in plants and animals, is largely a question of heredity plus the previous habits of the individual. It is also contributed to by chronic alcoholism, gout, syphilis, hypothyroidism, chronic nephritis, focal sepsis and chronic lead poisoning. Structurally there is a universal tendency to atrophy or degeneration of the parenchyma or functionally active tissues, and slight increase in the lower forms of tissue (such as fibrous and supporting tissues) in all the organs and structures of the body. This is seen particularly in the cardio-vascular system, where the muscular coat (the functionally active tissue of the arteries) first shows signs of senile degeneration (see § 93).

Symptoms.—Consequent on the changes above mentioned there is a universal lowering of vitality and nutrition, and the general enfeeblement of thought, word and act which results in the mumbling, fumbling and stumbling of old age. Physical weakness comes on so slowly that even the patient himself is hardly aware of it. In the second half of life, and especially in those with an alcoholic history, atheroma of the blood vessels, often with accompanying myocardial degeneration, may cause no other symptom than debility. *Endocarditis* (rheumatic or infective) should be remembered. It is not sufficiently recognised that widespread disease of the arteries alone may give rise to progressive mental and bodily enfeeblement at whatever age it comes on.

The following case may be quoted by way of illustration: Jessie T—— (æt. 49) first began to complain of muscular and mental weakness four years previously. This gradually increased, so that at the time of admission she could only walk by pushing a chair before her, and the case was thought at first to be some kind of paraplegia. There were no physical signs in any organ, no evidences of disease in the nervous system at any time, and the urine was always normal. She became progressively more and more enfeebled in body and mind, gradually took to bed, and died, ten years after admission, of progressive asthenia. At the autopsy all the organs were normal, both macro- and microscopically, with the exception of atrophy; but there was extreme and widespread disease of all the arteries of the body and of the brain, the main change being granular degeneration of the muscular coat of the heart and arteries, with consequent yielding and great dilatation of the arteries.

The condition of the heart and aorta should be carefully noted. The blood pressure should be recorded at intervals, and the walls of the superficial arteries carefully investigated (§§ 91 *et seq.*). Among the later symptoms associated with senile decay of the cardio-vascular and other tissues, perhaps vertigo is the commonest. A large number of other cerebral vascular symptoms may be experienced, and even syncope may occur (§§ 35, 719). The urine should be carefully and repeatedly examined so that senile decay may not be confused with other causes of debility (*infra*), particularly chronic interstitial nephritis.

The *Prognosis* depends upon the amenability of the cardio-vascular system to treatment. The diseases to which old age is most liable are of a chronic and degenerative nature, the arterial—*i.e.*, the nutritive system

being responsible for this, and itself showing the most definite and widespread signs of degeneration. The immediate cause of death in old age is usually some pulmonary complication. An analysis of 409 fatal cases in persons of sixty years of age and upwards, who died consecutively in Paddington Infirmary showed that 121, or 30 per cent., died of some pulmonary condition other than tubercle (pneumonia, bronchitis and hypostatic congestion). The next most fatal disease was cancer, 62 cases (15·5 per cent.), then simple senile decay, 35 cases (9 per cent.), then chronic interstitial nephritis, 24 cases (6 per cent.), then pulmonary tuberculosis, 22 cases (5·5 per cent.).

The *Treatment* should be mainly directed to the cardio-vascular system, and especially to the raising of low blood-pressure and the lowering of high blood-pressure (§§ 87 and 88). Stimulants are nearly always called for in the treatment of disease in the aged. The food should be light, nutritious, and easily assimilable, and small in quantity; it is remarkable how small a quantity of food the aged require, and it has been reckoned that 12 ounces of solid food per diem are sufficient. It is not only useless but harmful to over-feed the aged; keep them warm and prevent chill, but do not over-feed. Strychnine is *par excellence* the tonic of the aged.

II. DISEASES OF THE NERVOUS SYSTEM, FUNCTIONAL OR ORGANIC. In various *functional and degenerative conditions of the nervous system* general weakness may be the chief complaint. *Sleeplessness, worry and mental strain* are potent causes in modern life. This is especially true of functional disorders, such as neurasthenia and hysteria, where the weakness may amount to complete prostration. Such cases are usually seen in the first half of life or middle age. *Toxic* or *infective* causes may be present, such as addiction to alcohol, tobacco or morphine, and bromide intoxication. *Polyneuritis* is often described by the patient as causing general weakness, in which the limbs are especially involved. Among the gross lesions which may develop insidiously, with weakness, are *paralysis agitans, bulbar paralysis,* and frontal *cerebral tumour*—diseases more often met with in the second half of life. *Myasthenia gravis* is a rare condition, usually evidenced by local and generalised weakness (§ 808).

III. FOCAL SEPSIS should be looked for in long-continued debility without any definite cause. Fleeting or constant " rheumatic " pains and low grade pyrexia may also be present. Pus in the antrum, sinuses or tonsil is often unsuspected. A dead tooth, with a granuloma at its root not always visible to X-ray, may cause in time serious illness. Uterine or cervical discharge may be overlooked. EARLY TUBERCULOSIS of the lungs and other areas should always be remembered in cases of unexplained general debility, especially in younger subjects.

IV. CHRONIC DYSPEPSIA, gastritis, visceroptosis, and other obscure DISEASES WITHIN THE ABDOMEN may be attended for long by debility only. Gastro-intestinal troubles produce debility by causing toxæmia and malassimilation of food. In *B. coli* infections, before the development of urinary signs, there is often a history of many months of readily induced

fatigue. In women there is often, but not always, a concurrent history of frequent micturition; in children this may be absent. *Mucous colitis* may be especially mentioned, also *chronic appendicitis, abdominal cancer*, and many of the other conditions mentioned in Chapter IX.

V. Chronic Interstitial Nephritis (§ 401) is a cause of progressive enfeeblement coming on at or past middle life. It is apt to be mistaken for senility; failing vigour is the leading symptom for which the patient seeks advice in a large proportion of both these conditions. Sometimes this weakness is accompanied by generalised muscular wasting, but quite as often there is none. The complexion is generally sallow, but there is no definite pallor till late in the disease. Headache is common, chronic interstitial nephritis being one of the commonest causes of headache coming on after middle life.

RARER CAUSES OF DEBILITY

§ 559. VIII. **Myxœdema** (μύξα, mucus; οἴδημα, swelling) is an insidious disease evidenced by weakness, lethargy, and other manifestations of deficiency in the metabolic processes of the body, due to diminished thyroid function. It was so named by the late Dr. Ord, who at first believed it constituted a new and till then undescribed form of generalised œdema. Connective tissue develops in the skin round the hair follicles, sweat and sebaceous glands, and subcutaneous fat deposits occur; this does not allow pitting on pressure.

Symptoms.—(1) The weakness is associated with a characteristic slowness of action, thought and speech. It usually comes on very gradually, but occasionally is of acute onset and is much more common in women at the menopause, in whom the milder degrees are often overlooked. (2) The aspect is so characteristic that when the doctor has once seen a case he recognises it again directly (Fig. 1). The skin has a yellow tint which contrasts with a flush on the cheeks. The face is smooth and expressionless, is slightly puffy, especially around the eyes, the eyebrows are thin, especially in the outer parts, and the hair of the scalp is coarse, dry and unruly. (3) The tongue is broad and flabby. (4) There is a deposit of connective tissue in the skin as a whole, often with pads above the clavicles, back of the neck and over the deltoids. The hands become puffy and spade-like. The skin is dry and inelastic, and rarely sweats: it looks œdematous but does not pit on pressure except around the ankles. (5) The mental processes are dulled, the speech is slowed and often husky, and at times wandering of thought and action may give rise to hallucinations or to mild dementia. (6) The temperature is subnormal, the hands and feet cold and blue, and intolerance of cold is usual. (7) The pulse rate is slowed to 50-60 per minute, but in advanced cases myocardial degeneration ensues, with a dilated heart, poor heart sounds, and a more rapid pulse rate. The vessels become prematurely sclerotic, often with a raised blood pressure. (8) Digestive processes are slowed and obstinate constipation is the rule. (9) Anæmia of secondary type is often aggravated by menorrhagia, except at the menopause; the activity of the bone marrow is also deficient. (10) The basal metabolic rate is lowered, and a considerable gain in weight is usual. (11) The thyroid gland is commonly diminished in size, but may be larger than normal. In rare instances the condition is not primarily due to under-action of the thyroid gland but to diminution of the *thyrotropic hormone of the pituitary*, when amenorrhœa, *loss* of weight, and other evidences of anterior pituitary deficiency accompany the myxœdema (§ 554. VII).

Diagnosis.—Myxœdema may be mistaken in its earlier stages for *anæmia* and the other disorders mentioned in Group I, also for other causes of *debility*. A subnormal temperature, and pads above the clavicles or back of the neck, may be the only signs of slight thyroid insufficiency. It may be diagnosed from chronic inter-

stitial nephritis and other forms of chronic renal disease by the absence of pitting on pressure and of the urinary changes of renal disease. In obscure cases the lowered basal metabolism is of value (§ 925).

Prognosis.—Before the introduction of the thyroid treatment advanced cases rarely lived more than a few years, dying usually of cardio-vascular changes or some intercurrent malady.

Etiology.—The disease is much more frequent in women, in whom it supervenes usually about middle life. It is due to a deficiency of thyroid function. Sometimes a toxæmia, by exhausting the thyroid, leads to hypothyroidism ; it may arise during treatment with thiouracil. See also juvenile myxœdema (§ 191).

Treatment.—The treatment by the internal administration of thyroid is so certain and efficacious that this may be used as a means of diagnosis. Thyroideum (B.P.) ¼ to ½ gr., very cautiously increased, may be administered twice or thrice daily after meals: eventually some cases can take over 5 grains daily. The drug must never be pushed to the point of producing tachycardia ; the basal metabolism test shows when enough thyroid has been taken. Complete recovery may ensue after a few weeks' or few months' treatment but the patient is usually obliged to continue treatment indefinitely : for the effect of treatment see Fig. 1. In cases of urgency intravenous thyroxin can be given. To combat the anæmia, iron must be prescribed in addition.

§ 560. IX. **Addison's Disease** is a rare malady, described by Dr. Addison in 1855, characterised by progressive loss of strength and general pigmentation of the skin, due to disease of the suprarenal glands.

The *Symptoms* come under five categories: (1) Progressive *general weakness* is its most marked feature and may appear long before any other symptom. It is unaccompanied, as a rule, either by anæmia or marked emaciation until perhaps towards the end. Uncomplicated cases present a subnormal temperature throughout, but in those of tuberculous origin a low-grade temperature is common. (2) *Pigmentation* of the skin ensues sooner or later in most people, but may be slight or absent in the very fair skinned. The colour begins with a yellowish tint, which gradually deepens into a bronze mahogany colour in those of dark complexion. The localities most affected are the exposed parts (the face, neck, and hands), those where pigmentation is normally present, such as the axillæ and nipples, and sites of pressure (*e.g.*, waist). The edge of a patch of colour shades gradually into the healthy skin around, which makes it difficult to discover such a patch in its early stage. The mucous membranes of the tongue, gums, lips, cheeks and throat frequently show pigmented patches. (3) *Gastric* symptoms generally occur at some time, such as vomiting, hiccough, and cramp-like pains in the abdomen and loins. Pains in the limbs may also be complained of. There is often constipation, but sometimes there is intractable diarrhœa, which may be fatal. (4) *Cardio-vascular symptoms* may be present—palpitation, dyspnœa, sighing, yawning, a poor peripheral circulation, and later a tendency to collapse. A marked feature is the low systolic and diastolic pressure (*e.g.*, 70S—50D). (5) *Nervous symptoms* are less common, but may consist of headache, vertigo and nervousness. The mind is clear, except towards the end, when delirium, convulsions, or coma may set in. (6) *Crises* in the symptoms and signs are precipitated by infection, over-excitement and cessation of treatment: in them there is collapse, dehydration and hypoglycæmia, a rapid feeble pulse and a very low blood pressure: irritability or stupor may precede death. (7) The *blood* is concentrated, as shown by some degree of polycythæmia and a raised blood urea (up to 80 mgms. per cent.). The sodium and chlorine content of the plasma are usually low (below 300 and 330 mgm. per cent. respectively), whereas the potassium may be excessive. These groups of symptoms vary in their predominance, but asthenia is always present, and pigmentation nearly always. An acute variety may occur.

The *Diagnosis* is often difficult on account of the vagueness of the symptoms, the absence of physical signs, and the resemblance of the pigmentation to that seen

with other cachectic states, especially cancer. The low blood pressure and the low serum values of the sodium and chlorine are distinctive. *Cancer of the pylorus* is accompanied by sallowness, which is often mistaken for the pigmentation of Addison's disease. Both, moreover, are accompanied by enfeeblement, gastric pain and vomiting. Other *pigmentary conditions* are mentioned under pigmentation (§ 652); slight jaundice, the pigmentation of malaria, chloasma and arsenical pigmentation are the chief causes of error in diagnosis. Test the hair for arsenic. *Chronic nephritis*, neurasthenia, and other conditions attended by asthenia have been mistaken for the disease. X-ray may reveal calcareous deposits in the suprarenals when tuberculous disease is present, and yields negative findings when the disease is due to atrophy.

Prognosis.—The course of the disease is progressive, and usually prolonged; it may last one to ten years. There are frequent relapses, with intermissions of comparative health, but it always ends in death. It may end suddenly with syncope, severe vomiting, and diarrhœa, convulsions, or coma, or it may terminate gradually by asthenia. Intercurrent infections are a frequent cause of death. Occasionally cases run an acute course, death occurring in a few weeks.

Etiology.—The disease is due to insufficient production of the various suprarenal cortical hormones: this causes a deficiency of blood sodium chloride with dehydration and hypotension, and also hypoglycæmia. Patients are usually about middle life, and by far the larger number are males. The destruction of the suprarenal glands is a result of (i.) tuberculosis or (ii.) slow progressive atrophy. In a few cases the glands have been normal at autopsy, but their nerve supply has been destroyed.

Treatment.—Extracts of suprarenal cortex (eucortone and eschatin) and desoxycorticosterone are effective. In *acute relapses* (often with an infection) 15–50 c.c. of eucortone are given (intravenously or intramuscularly) daily, and should be combined with a litre of 5 per cent. dextrose in N-saline: the dose is gradually reduced to 3–5 c.c. daily, subcutaneously. For *maintenance* desoxycorticosterone acetate is frequently effective: this substance, believed to be one of the essential constituents of the suprarenal cortex, has now been synthesised and is usually given in an oily solution intramuscularly (5 mgm. is equivalent to 10 c.c. of cortical extract). Sterile pellets have been implanted subcutaneously, but need replacing after 4–6 months: for each ½ mgm. given as a daily maintenance dose 60–100 mgms. should be implanted, with a maximum of 1,000 mgms. When cortical extracts are used for maintenance, sodium chloride 5–15 G. daily in food, porridge, milk or in keratin-coated capsules should be administered. With desoxycorticosterone full doses of sodium chloride may be dangerous by producing excessive salt retention with œdema, hypertension and even heart failure, and the dose of sodium chloride should at the most be 3–5 G. daily. Symptomatic treatment consists in rest, supporting the strength and avoiding cold and over-exertion.

§ **561.** X. **Hæmochromatosis** (Bronzed Diabetes) is a rare disease, often showing a hereditary tendency, almost confined to the male sex. It seems to depend on some disorder of normal metabolism of iron, whereby the iron-containing pigment is not excreted but retained in the tissues. The liver, pancreas and skin suffer most, and the three cardinal symptoms of the disease depend on this fact. They are: (1) cirrhosis of the liver, with ascites; (2) glycosuria, usually permanent and leading to coma, but in some cases appearing late as a subsidiary feature; (3) pigmentation of the skin, giving rise to a slaty colour, occurring chiefly on exposed areas and not on the oral mucous membrane. The course is from a few months to one or two years, and treatment, except for the alleviation of diabetic symptoms or ascites, is unavailing.

XI. **Disease of the Pancreas, acromegaly, myelopathic albumosuria, beri-beri, pellagra, alkalosis, and other conditions mentioned in Groups I and II** (*q.v.*), may come on with debility only, or the patient may seek relief for debility. *Hypoglycæmia* (§ 418) is often overlooked; the exhaustion in such cases is relieved by taking food.

XII. **Carbon monoxide poisoning** should be borne in mind when lassitude, slight giddiness or headache are often present. Other symptoms are nausea, vomiting, palpitation, inability to move the limbs. Slight gas leaks, imperceptible to smell,

can cause malaise. Petrol fumes in closed cars, escape of noxious coal and anthracite fumes from ill-ventilated chimneys or through overheated metal stove casings, are often unsuspected causes of poor health. In large doses this gas causes collapse or coma without any warning. (See § 717. IX.) The diagnosis is made by removing 10 c.c. of blood from a vein and examining its spectrum.

CHAPTER XVII

THE EXTREMITIES

IN the preceding pages we have seen on several occasions that so-called local diseases, such as pneumonia and endocarditis, have by scientific research been shown to be only local manifestations of a general bacterial infection. This principle will here again be illustrated, for a gouty joint is only the local evidence of disordered metabolism, and acute rheumatism is probably microbic in origin. Probably all joint diseases (other than traumatic) are local manifestations of some toxic, septic, or infective blood condition. In conformity, however, with the scheme of this work, whereby all diseases are approached from a symptomatic standpoint, certain diseases, the symptoms and physical signs of which are referable mainly or entirely to the upper or lower extremities, will now be considered.

PART A. SYMPTOMATOLOGY

The CARDINAL SYMPTOM referable to the extremities is **pain** (or painful sensations of some kind), which may or may not be accompanied by some **physical change.**

§ **567. Pain in the Limbs** should be investigated, like **pain** in other situations, as to its *position*, *character*, *degree*, *constancy*, and *duration*. Its position may be localised to the skin, or to a joint or any other structure, or be generalised, as in sheer exhaustion ; its character may be sharp and shooting (as in tabes) or dull and heavy (as in vascular lesions), or like pins and needles (as in nerve and neuro-vascular lesions). The skin, subcutaneous tissues, nerves, muscles, and vessels must be examined for a local cause ; but it must be remembered that pains in the limbs, especially in the legs, may be due to a generalised infection which may not be evident for some time after the onset of the pain. So also disease of the brain, spinal cord, chest, or abdomen may be the causal condition ; hence a thorough examination, including investigation of the urine, blood and even lumbar puncture, may be necessary in obscure cases. Pain in the limbs may come on *acutely* or *insidiously*.

(*a*) *Acute pain in the limbs* coming on more or less SUDDENLY may herald influenza, tonsillitis, typhoid fever, malignant endocarditis, variola, scarlatina, or some other specific fever. In many cases of influenza this pain and pyrexia are the only symptoms. *Acute rheumatism* also comes on rapidly with pains referable to the muscles, bones or joints, and so does *dengue* ("break-bone" fever). In *Trench fever* there is great pain and tenderness in the legs, especially in the shins. *Trichinosis* is attended by excruciating muscular pain in the second stage of the disease, when the parasite begins to migrate. *Scurvy*, *osteomyelitis* and *epiphysitis* are

other causes of pain in the limbs associated with pyrexia. A sudden sharp pain in one spot in the limb is felt when *embolism* of an artery occurs: so also in *thrombosis* of a vein. In either case pyrexia may be absent.

(*b*) *Subacute and chronic pains in the limbs* coming on more or less *insidiously* may be due to (1) affections of the *nerves*, of central or local origin and especially to sciatica and polyneuritis. Carcinoma of the prostate may produce pains in the legs with demonstrable deposits in the pelvic bones. The same pains may occur in neurasthenia; the pathological condition in this disease, as in alcoholism, neuritis, lead poisoning, and the acute specific fevers, may be due to toxæmia. Other causes of *nerve* origin are tabes dorsalis, cervical rib, cerebral tumour and meningitis, disease of the spinal cord or vertebræ, neuralgia, and acroparæsthesia. Severe pain in the foot should lead us to suspect flat foot or metatarsalgia. Metatarsalgia (Morton's disease) is a neuralgia of the foot due to lateral displacement of the heads of the metatarsal bones which press upon the nerves, and may also produce a corn (for which, indeed, the patient may seek advice). (2) Pains in the *joints* or *muscles* are characteristic of chronic rheumatism, rheumatoid arthritis, osteoarthritis, gout and all forms of *synovitis*. (3) *Vascular* affections are such as thrombosis, embolism, intermittent claudication, erythromelalgia, aneurysm, varicose veins, phlegmasia alba dolens, Raynaud's disease, periarteritis nodosa and gangrene. "Numbness" and tingling are characteristic of vascular affections, and may indicate a vasomotor disorder, or may be due to a *neurological* cause such as neuritis, the incipient stage of tabes, disseminated sclerosis, or other organic nerve disease. (4) Certain *skin* diseases are accompanied by pains in the limbs. (5) *Growing pains* (so-called) in children may be of serious import, as the first evidence of subacute rheumatism, which may produce endocarditis with permanent damage unless the condition is recognised and dealt with early. (6) Pains in the legs may be due to orthopædic and postural defects of the feet and spine. (7) Various diseases of the *bones* (§ 595) begin insidiously, with indefinite, vague pain in the limb or limbs. This must be especially remembered in children. Acute or chronic inflammation may arise, and unless the bone be superficial there may be no surface indications. Osteomyelitis is very serious, and requires prompt recognition. Syphilis causes pain in the bones, worse at night. Other causes are Paget's disease, multiple myeloma, tumours, rickets and blood diseases. (8) A *muscular* strain or rupture of muscular fibres may leave a chronic pain and partial loss of function unattended by any physical sign. Other causes of muscle pain are acute myositis, rheumatic fibrositis, trichinosis, tumours, and myositis ossificans. (9) *Local injury* or pressure, such as injury from a crutch, or sleeping in a cramped position, or lymphatic glands or other tumours in the axilla, neck, or pelvis. Heavy rings may cause ulnar neuralgia. Shooting pains down the arms, especially the left, occur in aneurysm of the aorta and angina (see also § 800). (10) A careful examination of the *chest* should be made, for pain down the arm may indicate disease in that region; *e.g.*, apical pleurisy, cardiac disease,

aneurysm or other mediastinal tumour. (11) Disease of the *pelvis*, vertebræ and hip joint are frequently overlooked causes of pain in the legs.

PART B. PHYSICAL EXAMINATION

The physical signs referable to the extremities consist mainly of some visible or tangible alteration in the skin and general contour of the limbs, the joints, the muscles, the bones, or the vessels and nerves.

§ 568. **Inspection of the Limb** may reveal generalised redness or alteration of colour, œdema, varicose veins, or some other diffuse or localised swelling. Eruptions are dealt with in Chapter XVIII. Œdema of both legs is dealt with under Dropsy.

Even without the skill of a palmist or the acumen of a Sherlock Holmes a great deal concerning the temperament, habits, and diseases of a patient may be learned by a careful **inspection of the hands.** The long, thin, dextrous *fingers*, perpetually on the move, almost surely indicate a nervous temperament and imaginative disposition, just as the short, thick, almost clumsy fingers and hands of another bespeak slowness, deliberation, and doggedness. The occupation of a patient may often be learned from a glance at the palms. Some people habitually have cold, damp, clammy hands; these generally suffer from alcoholic habits, rheumatism, rheumatoid arthritis, or other condition causing a defective vaso-motor tone. Finger pads, fibrous thickenings over the proximal finger joints, occur with rheumatic or other chronic infection. The *nails* and nail-beds afford information. They are dusky with impaired circulation, and pale in anæmia; compression on the tip of the nail should not completely empty the capillaries, as it does in anæmia. In aortic regurgitation compression of the nail tip reveals capillary pulsation. A transverse ridge or white mark in the nails indicates injury or arrested growth, and may mark the date of an illness of even so slight a nature as sea-sickness. It is useful to remember that the nail takes five to six months to grow from root to tip. Atrophic and hollowed nails (Koilonychia, Spoon nails) may be congenital; when acquired, hypochromic anæmia is usually present. Various distortions of the nail occur in Raynaud's disease, neuritis and injury, as with manicuring. Pitted, dark, and discoloured nails may be due to eczema, psoriasis, ringworm or monilial (thrush) infection. *Clubbed fingers—i.e.*, fingers with a bulbous end and great convexity of the nails (filbert-shaped nails), are characteristic of congenital cardiac disease or valvular disease in early life; malignant endocarditis and long-standing biliary cirrhosis are other causes. Clubbed fingers are also seen with pulmonary osteo-arthropathy, empyema, fibroid lung, chronic phthisis, bronchiectasis and lung abscess. *Glossy fingers* (fingers with smooth, thin skin) are the result of a neuritic dystrophy, and are associated with destructive and paralytic lesions of the nerve trunks; they also occur in scleroderma. *Dactylitis* is a thickening of one phalanx due to disease of the bone, with infiltration of the tissues of the fingers, resulting in a deformity known as the "champagne bottle finger." It is met with chiefly in tuberculous, and sometimes syphilitic, children. Heberden's nodes, lipping and distortion of the terminal phalangeal joints, are in reality osteoarthritis of the fingers. Gouty nodules of urate of soda form white masses near the joints, just beneath the skin, and have an external resemblance to Heberden's nodes. Scars may be due to painless injuries, as in syringomyelia or tabes, and from chronic sinuses, as in gout and in tertiary ulcers of the leg. The bone ends of the wrists are enlarged in rickets, syphilis, and pulmonary osteo-arthropathy. "Spade-shaped" hands, with thickened tissues, suggest myxœdema; large, flat hands with osseous enlargement occur in acromegaly and pulmonary osteo-arthropathy. The "claw-hand" (*main en griffe*) occurs as the result of injury or neuritis of the ulnar and median nerves; it is also seen in progressive muscular atrophy, syringomyelia, and cervical pachymeningitis. Wrist-drop is characteristic of musculo-spiral nerve paralysis, as in lead palsy.

§ 569. **Varicose Veins** consist of dilatation and tortuosity of the superficial veins, and occur chiefly in the legs, where their tortuous elevations are characteristic. They occur chiefly in those who stand a great deal, especially in women who have borne children. Varicose veins predispose to œdema, eczema and ulceration, and severe hæmorrhage may ensue if they rupture. Injection treatment has no danger if correctly performed, and as the patient can walk immediately after it, there is no interference with his work. A sclerosing solution leads to clotting, then obliteration of the vein. Contra-indications are pregnancy, phlebitis, deep thrombosis, renal, liver or cardiac disease.

Technique.—For anterior veins the patient should sit with the leg on a stool; for posterior or very large veins, he may stand or lie supine. Use the ordinary Record syringe with a number 15 or 16 needle. Hold the vein firmly with the left hand and push the needle into the vein. Take care that none of the solution enters the tissues outside the vein. Inject very slowly and begin with the lower veins. The solutions most used are: quinine urethane, dose ½ to 3 c.cm.; monoethanolamine oleate (ethamolin), 5 per cent.; sodium morrhuate, 5 to 10 per cent.; and for very small veins, salt 20 per cent. with 1 per cent. tutocaine.

Gas gangrene is due to infection and necrosis of muscles, usually by anærobic organisms such as *Cl. welchii*, with which streptococci and staphylococci are often associated. If untreated, the infection spreads, producing swelling, with crepitations and a dark-brown discoloration: on incision, bubbles of gas escape and there is a musty odour. The condition often responds to antitoxin, penicillin and sulphathiazole or sulphadiazine therapy: but as these drugs do not penetrate into devitalised tissues, surgical excision and drainage are still of major importance.

§ 570. **Œdema of one limb** (localised dropsy) is a swelling which pits on pressure. The swelling due to elephantiasis lymphangiectodes (see below) is usually much more solid. *Œdema localised to one area* of a limb indicates a local cause, usually inflammatory œdema (*e.g.*, cellulitis), osteomyelitis, angio-neurotic œdema or local thrombosis. *Generalised œdema* of the greater part or whole of a limb points to obstruction of the main vein by thrombosis within, or pressure on the vein or lymphatics from without. (1) *Simple venous thrombosis* indicates a clot within the vein, and apart from œdema is often symptomless, with no local pain or tenderness. *Thrombo-phlebitis* indicates thrombosis with an inflammatory reaction in the vein wall, and often follows simple thrombosis: it causes local pain, tenderness and often swelling at the seat of obstruction, with some irregular fever. The site of disease in the leg may be the superficial or deep veins of the calf, the superficial or deep femoral or the iliac veins: in the arm it is usually in the brachial or axillary vein. The *causes* in any given case are often multiple: pressure from without (as in the calf veins on an operating table), venous stasis (due to bed rest), dehydration, increased coagulability of the blood and focal sepsis (especially apical infection of the teeth or tinea tarsi) all contribute: carcinoma of the stomach is often an unsuspected cause. A common example is *phlegmasia alba dolens* (white leg) which may follow confinement, partly as a result of the previous pressure on the pelvic veins. In *thrombosis migrans*, thrombosis occurs in several arteries and veins in various parts of the body at different times. A femoral, coronary, pulmonary and cerebral thrombosis may all occur in the same individual: a septic focus is believed to be causal in many cases: the prognosis is often grave. The commonest *complication* of venous thrombosis is a pulmonary embolism, which may recur (§ 105): a sudden, unexpected and sometimes fatal embolism may be the first evidence of unsuspected thrombosis in the deep veins of the leg or pelvis. *Treatment.*—Prophylaxis demands, after childbirth or abdominal operations, early movements of the legs, breathing exercises, a copious fluid intake, and raising the calves from the operating table. When thrombosis is especially to be feared, dicoumarol may be given. Once thrombosis has occurred instead of complete rest as formerly advised, it is now usual to administer drugs which prevent the spread of the clot and hasten its resolution. Heparin acts rapidly and Bauer advises four-hourly intravenous

doses of 150 mgm. (1 mgm. = 80 units) for 24–36 hours, followed by 100 mgm. three or four times a day until symptoms subside: he combines this with active movements of the affected limb on the first day. Others use a continuous drip transfusion with 10–30 units of heparin per minute. Dicoumarol acts by lengthening the prothrombin time of the blood (§ 533) but has little effect for the first 24 hours: it is given by mouth in an initial dose of 200 mgm. the first day, followed by 100–200 mgm. a day with a view to keeping the prothrombin index between 35 and 50 per cent. of normal. (It should only be used in hospital where daily prothrombin estimations can be carried out). Some advise also ligaturing the common femoral or iliac veins. (2) Œdema may also be due to *pressure upon a vein* by a tumour such as enlarged glands in the axilla or elsewhere, aneurysm, or other intra-thoracic growth pressing upon the veins coming from the arm; pelvic cellulitis, carcinoma of the prostate bladder or uterus, bands of adhesion, or other intrapelvic growth pressing on the veins of the leg.



Fig. 134.—Elephantiasis Lymphangiectodes in a man about forty years of age who had never been abroad.

Elephantiasis Lymphangiectodes (Fig. 134) is a solid œdema, not pitting on pressure in any notable degree, affecting one leg, occasionally one arm, or the scrotum, due to a blocking of the lymphatics of the limb. It may occur after blockage of the lymphatics by carcinoma, or removal of a tumour. It is met with chiefly in tropical countries in persons whose blood contains the filaria embryos. The adult worm is believed to block the lymphatics, and so produce the disease. It is occasionally seen in temperate climates in persons whose blood does not reveal the parasite, and the cause in these cases is obscure. Bilateral localised œdema occurs in **Milroy's hereditary Œdema** (Meigo's disease). The condition affects several members of a family. The œdema has an abrupt line of demarcation at the level of the hip, knee or ankle. There may be a history of attacks of fever with increase of swelling. The *cause* is unknown. The *diagnosis* rests on the history, and similar œdema in other members of the family. *Treatment* consists in adequate support for the limbs.

§ 571. Swelling of the Lymphatic Glands in the neck, axillæ, groins, or elsewhere on the surface of the body or limbs may be due to: I. injury, septic or infective processes; II. tuberculosis; III. cancer; IV. lymphosarcoma; V. syphilis; VI. climatic bubo; VII. chancroid; VIII. acute specific fevers; IX. leukæmia; X. lymphadenoma, § 572; XI. glandular fever, § 499; XII. plague, § 500; XIII. trypanosomiasis, § 518; XIV. Japanese river fever, § 486. In I, II, III, VII and XIV the glands enlarge adjacent to some focus of mischief, and the glandular swelling usually remains localised; in the remainder all the lymphatic glands tend to become affected.

I. *Local* injuries, septic sores, infected tonsillar crypts and abscesses give rise to enlargement of the neighbouring lymphatic glands. Pain and enlargement of the glands in the groin, for instance, may be due to direct injury to those glands; but more usually to a sore on the foot or around the toe-nails, through which infection has occurred. *Post-mortem scratches* or inoculation from septicæmia cases are of a

much more virulent nature. Red streaks along the course of the lymphatics indicate lymphangitis. The glands at the elbow and axilla become acutely painful and tender, and may rapidly suppurate. In the absence of chemo-therapy general septicæmia and death ensue in a day or two.

II. *Tuberculous disease* occurs most often in children. (*a*) The commoner type affects the upper cervical glands, causing tenderness and enlargement, usually on one side only in the earlier stages. At first discrete, the capsules of the glands are later broken through by caseous material, causing matting together and finally a sinus to the skin. (*b*) A rare form (endothelial tuberculosis) is more common in adults: there is general enlargement of the lymphatic glands and often splenic enlargement. The glands rarely mat together and constitutional disturbance is slight.

III. *Cancer* gives rise first to enlargement of the adjacent glands (§ 555). These glands are known by their hardness and their tendency to invade, and become fixed to adjacent tissues.

IV. *Lymphosarcoma* is a sarcomatous growth *starting in* the lymphatic glands. Lymphosarcoma may resemble lymphadenoma; the glands form greater masses and tend to infiltrate the neighbouring structures; this causes more marked pressure signs in the chest and abdomen (see § 81). The diagnosis may be possible only after excision and examination of a gland.

V. *Syphilis* first affects the lymphatic glands in the neighbourhood of the chancre (and therefore usually in the groin). They are small, hard (shotty), painless, and only perceptible on palpation. In the secondary stage these become larger but do not suppurate as with a soft chancre; at the same time there is universal lymphadenitis, and careful palpation for many years afterwards will still reveal this (§ 552).

VI. **Lymphogranuloma inguinale** (Syn., Climatic or Tropical Bubo) is a venereal disease caused by a filterable virus which is common in Oriental countries and America and is now appearing in Europe.

Symptoms.—The incubation period varies from a few days to three weeks. The *Primary* lesion consists of small herpetiform ulcers on the penis. The *Secondary* phase shows swelling of the medial group of inguinal glands in from one to six weeks later. Both sides may be involved. Discomfort, tenderness and groin pain accompanied by fever may first suggest the condition. The glands at first are hard to the touch and somewhat tender; the surrounding skin becomes red, then bluish-violet. Subsequently they remain indolent, or fluctuation occurs and sinuses form in about half the cases. Generally the iliac glands are palpable as a hard mass above Poupart's ligament. General features include fever of remittent type which may last many weeks, anorexia and loss of weight. In women a chronic elephantoid condition of the vulva may ensue, also stricture of the rectum.

Diagnosis.—Herpes genitalis resembles the primary lesion, while filarial involvement of the lymph glands, septic and tuberculous adenitis, venereal buboes due to chancroid, gonorrhœa and syphilis, and buboes due to plague, rat-bite fever and tularæmia may need differentiation. A history of cohabitation with native women is an important aid to diagnosis. This is confirmed by Frei's intradermal test, which is performed with sterilised diluted pus or mouse-brain virus. A reddish, infiltrated papule of from 7·5–20 mm. diameter appearing within forty-eight hours is evidence of infection with the virus of climatic bubo.

Prognosis.—The disease is rarely fatal, but enlarged glands, sinuses or ulcers in the groin may take eighteen months to heal.

Treatment.—The patient is put to bed on a nutritious diet and sulphonamide therapy instituted without delay (Table XXVIII). Intramuscular injections of anthiomaline aid healing: streptomycin is on trial, but penicillin is disappointing. In early cases excision often leads to rapid cure, but the femoral chain of glands should not be unduly interfered with, lest elephantiasis of the limb result. Pus, where present, should be aspirated under aseptic conditions, the needle being inserted into healthy skin some distance from the bubo. At this late stage excision does not give good results.

VII. **Chancroid** is due to Ducrés bacillus, and has an incubation period of only a few days. It gives a soft sore on the genitalia, and the inguinal buboes often suppurate.

VIII. In most of the *acute specific fevers* there is, as in syphilis, a slight generalised glandular enlargement. After *Whooping cough* isolated glandular enlargements are not uncommon. In those fevers which have a local manifestation—the throat in scarlet fever and diphtheria, for instance—the adjacent glands are first and chiefly affected. In German measles the occipital glands are especially noticeable. In bubonic plague the enlargement is great; in milder cases of plague only slight glandular swelling and fever occur (pestis minor). *Rheumatoid arthritis* is accompanied by enlargement of the glands and spleen, especially in children.

IX. In *Leukæmia* there is a generalised enlargement, and the blood changes are characteristic (§ 543).

§ 572. X. **Lymphadenoma** (Synonym: Hodgkin's disease).—This disease is characterised by progressive enlargement of the lymphatic glands; enlargement of the spleen; and later progressive anæmia without other important blood changes.

Symptoms.—(1) Enlargement of lymphatic glands. Those in the neck are often first affected, but in other cases the primary glandular enlargement may occur in the mediastinal, abdominal or groin glands. Sooner or later other groups are affected, until widespread adenopathy is present. The glands are sometimes painful and tender: are freely movable over each other and over adjacent structures; of rubbery consistency; without tendency to break down. (2) Enlargement of mediastinal and mesenteric glands may produce special symptoms, due to pressure upon important structures, such as the veins in the thorax and abdomen, the trachea and bronchi, and œsophagus. (3) A peribronchial invasion produces the symptoms and signs of a mediastinal tumour. (4) Enlargement of the spleen occurs later, and is sometimes very marked. (5) Fever, either a low irregular fever, or an undulating fever, the waves varying in length from a week or ten days to a month or more. This undulating fever, in lymphadenoma, is known as the Pel-Ebstein syndrome. (6) Symptoms of anæmia, slight at first, but later becoming more severe. The blood changes are not characteristic, apart from the anæmia, though there is often a high polymorph leucocytosis, and an eosinophilia in 15 per cent. of cases. (7) General pruritus, sometimes severe, and various eruptions, including herpes zoster. (8) Bronzing of the skin in some cases. (9) Spinal deposits cause spinal compression. (10) The blood sedimentation rate sooner or later reaches very high figures.

Diagnosis.—The disease must be distinguished from others giving rise to enlarged lymphatic glands. It is distinguished from *lymphatic leukæmia* by the absence of the blood changes of that disease; from *glandular fever, secondary syphilis, rubella,* secondary deposits of *malignant disease,* etc., by the size, consistency, and distribution of the glands, together with the progress of the case, and the absence of evidence of the other diseases. Occasionally it may be difficult to distinguish between lymphadenoma affecting only one set of glands and some other diseases, especially *lymphosarcoma* and *tuberculosis*; it may be necessary to excise a gland, under local anæsthesia, to establish the diagnosis by histological examination. Mervyn Gordon has established a biological test; after the intracerebral inoculation of an emulsion of a fresh gland into a rabbit, encephalitis with ataxia and spasticity of the hind limbs, and convulsions, may prove fatal. A similar encephalitogenic agent is present also in normal bone marrow and spleen and is believed by some to be a reaction to eosinophils.

Etiology.—Males are more frequently affected than females, and most often about middle age. The cause is undecided: Mervyn Gordon has described certain "elementary bodies" as being possibly causal. The morbid changes are of the nature of a granulomatous process which causes enlargement of the lymphatic structures, but in which almost all the organs are involved. The morbid anatomical changes are those of enlargement of the lymphatic structures of the body; the changes also occur in almost all the organs of the body, including the bone marrow. On microscopical examination there is found increase in the reticulum of the affected organ in the more chronic cases passing on to fibrosis; an increase in the lymphocytes; the presence of many endo-

thelial cells, some of which have more than one nucleus, giving rise to the characteristic Dorothy Reed giant cells; and often also an increase in eosinophil cells.

The *prognosis* is bad. The patient becomes progressively weaker and more anæmic, and pressure upon important structures by the enlarged glands may hasten the end. The more chronic cases may live for ten years after the diagnosis is made; febrile cases usually die within a year.

Treatment.—Complete excision of the enlarged glands, when they occur in one region only, if the diagnosis is made early, arrests somewhat the progress of the disease. Apart from this, arsenic is a most useful drug given in full doses: antimony compounds often aid. Deep X-ray also delays progress. General measures must be taken to improve the health and strength. Recently urethane (G. 1–2 daily) and courses of nitrogen mustard (0·4–0·6 mgm. per kilo) have given beneficial results, but are not curative.

§ 573. The JOINTS, MUSCLES, BONES, VESSELS, NERVES, and CONSTITUTIONAL SYMPTOMS should be next investigated.

The **joints** may need investigation for tenderness, pain, heat, swelling, or redness, and for loss of function or range of movement. The affected and the unaffected sides should be carefully compared. Slight degrees of fluid in a joint are often difficult to detect. The active movements (those which the patient can make) and the passive movements (those made by the doctor) should, with due consideration and caution, be tested. Among the *fallacies*, paralysis, or muscular weakness is often simulated by chronic joint diseases, and *vice versa*, and pain in the limbs from various causes will often simulate a stiffness of the joint. Disease near a joint may be mistaken for a diseased joint. Pain may be referred, *e.g.*, in hip-joint disease pain is often complained of in the knee. In neuritis pain may be referred to the joint supplied by the affected nerve. X-rays may help diagnosis. In acute joint disease the fallacies of epiphysitis and acute osteomyelitis must be avoided. The presence of associated symptoms may aid; for example, tophi suggest gout; subcutaneous nodules, rheumatism.

The **muscles** may be investigated for tenderness, stiffness, or swelling. The investigation of paralysis, tonic or clonic spasm, or wasting, is given under diseases of the nervous system (Chapter XIX). We are here concerned only with pain, tenderness, or swelling localised in the muscles (§§ 592, 594); it is the presence of these localised symptoms which helps us to differentiate muscular diseases from paralysis and other diseases of the nervous system. To decide that the lesion is not in the bones or ligaments may be difficult; if it be in the muscle, the pain is greater during active than passive movement of the affected muscle; if in the ligaments or joints, the pain is about equal.

The examination of the **bones** belongs especially to the surgeon, but disease situated in the bones may be evidenced by pain, tenderness, swelling, or deformity. They often first come under the notice of the physician when pain is the only symptom, and the diagnosis presents considerable difficulty. X-ray examination is of great value.

In the diagnosis of SWELLINGS CONNECTED WITH BONES it is well to remember the following data. Symptoms come on *acutely* with trauma, periostitis and osteomyelitis; slowly and *chronically* with caries, necrosis, chronic periostitis and osteitis, rickets, syphilis and tumour. In regard to physical signs the *diaphysis* is mainly affected in acute and chronic inflammation, in sarcomatous and other tumours; the *epiphysis* in rickets, syphilis, and central sarcoma. The consistency of the swelling is *soft* in abscess and vascular sarcomata, *hard* in chronic inflammation. As regards the mode and rate of growth, the swelling *progressively enlarges* in inflammatory and malignant tumours, and is *relatively slow* or *stationary* in chronic inflammation and benign tumours; *receding* swellings are always inflammatory.

The **vessels** and **nerves** need examination when any of the symptoms indicate their implication, as in erythromelalgia and some other conditions in Group I below. Pressure along their course may elicit tenderness, indicative of inflammation. For

symptoms of peripheral neuritis see §§ 793 *et seq.*; thrombosis of a vein, embolism of an artery, § 570 and § 577, and periarteritis, § 94.

The **viscera** should be examined, particularly in acute joint diseases, which are almost always the product of some blood disorder—*e.g.*, the heart must always be examined in rheumatic conditions, the kidneys in gouty disorders.

Pyrexia and **Constitutional Symptoms** are present in a considerable number of diseases of the extremities, particularly in the acute joint and bone disorders, and they may be investigated on the lines laid down in Chapter XV. Rigors and sweating indicate a pyogenic process. Characteristic blood changes are found in several diseases, notably glandular and septic processes.

PART C. DIAGNOSIS, PROGNOSIS, AND TREATMENT OF DISEASES CAUSING SYMPTOMS REFERABLE TO THE EXTREMITIES

§ **574. Routine Examination and Classification.**—As a matter of routine, as in other cases, investigate—

First, the LEADING SYMPTOM, which in this instance is very often as visible or palpable to the patient as to the physician.

Secondly, the HISTORY of the case, its mode of onset (acute or chronic), and evolution in chronological order.

Thirdly, examine the AFFECTED LIMB or limbs, their colour and contour, the joints, muscles, bones, vessels, or nerves, as may be indicated; and, finally, examine the VISCERA and the TEMPERATURE. The movements, reflexes and sensation, should be tested in cases where nervous disease is suspected. An X-ray examination is often essential.

If there is any visible abnormality in the COLOUR of the hands or limbs, turn to Group I, below.

If the symptoms point to JOINT disease, acute or chronic, turn to Group II, § 582 (Acute), or § 585 (Chronic).

If the symptoms point to disease of the MUSCLES (rare), turn to § 592.

If the symptoms point to disease of the BONES, turn to § 595.

If the symptoms point to disease of the NERVOUS SYSTEM, turn to § 691.

GROUP I. ALTERATIONS IN COLOUR OF THE EXTREMITIES

This group comprises only the following morbid conditions which may be considered medical. Other alterations in colour or contour, such as œdema of one limb, clubbed fingers and varicose veins, have been referred to in §§ 568 to 570. Pigment alterations are described in § 652. There remain—

I. Erythromelalgia and Acroteric Scleroderma, § 575.
II. Cyanosis, § 576.
III. Gangrene, § 577.
IV. Trench-foot and Frost-bite, § 578.
V. Raynaud's disease and Dead hands, § 579.
VI. Intermittent claudication, § 580.
VII. Pink Disease, § 581.

§ 575. I. **Erythromelalgia** is a painful redness and swelling occurring in paroxysms, and symmetrically affecting both hands, sometimes the feet, and sometimes spreading to the arms and legs. One side may be more affected than the other, but in all cases both sides are involved to some extent. The disorder starts intermittently with tingling and numbness in the extremities (*acroparæsthesia*), and later on a painful redness supervenes. The paroxysms are often determined, and always aggravated by hanging the limbs down, and also by placing them in very hot or very cold water. They are often worse when the patient lies down and goes to sleep, and thus the night may be badly disturbed. The pain and swelling are lessened by holding the hands over the head, or raising the feet. There is no paralysis, but the fingers cannot easily be bent, as thickening of the subcutaneous tissues may ensue, and the paroxysms are apt to return even after long intervals. The swelling and redness affect the whole hand (Figs. 135*a* and *b*)—not in patches, as in chilblains, lupus, or erythema.



Fig. 135*a*.—Erythromelalgia in a woman aged about thirty.



Fig. 135*b*.—Erythromelalgia, showing maximum closure of the hand.

Thus the *Diagnosis* is simple. In the cyanotic form of *Raynaud's disease* the symptoms start and prevail in one or two finger-tips; in erythromelalgia all the fingers and the whole hand are about equally involved. It is a prolonged and very painful disorder, never fatal, and is to some extent amenable to treatment.

Etiology.—Women are more prone to the disease in the proportion of 20 to 1, and chiefly between eighteen and twenty-five, and at the climacteric. The determining cause is probably a sympathetic-parasympathetic imbalance—the symptoms resemble those of cholinergic overactivity. Several cases have exhibited, concurrently with a severe paroxysm of the erythromelalgic symptoms, erythematous blotches on other parts of the body, and severe " rheumatic " pains in the limbs.

Treatment.—Bromides invariably relieve the condition for a time ; arsenic, strychnine, quinine, and other tonics are useful. The general health should be attended to, and particularly the digestion. Septic foci must be removed. A weak descending galvanic or a sinusoidal current is helpful. Severe cases need rest in bed, and sedatives such as bromides and a medium-acting barbiturate. Anti-histamine drugs may help.

Acroteric Scleroderma (Hutchinson) or **Sclerodactylia** is a scleroderma affecting the hands and feet, and sometimes the nose, in which the skin is bluish and thickened at first, white and atrophic afterwards.

§ **576.** II. **Cyanosis** (Blueness) **of the extremities.**—Blueness and redness of the extremities appear to be due to a vaso-motor condition or to polycythæmia (§ 31). After infantile paralysis the affected limbs are blue and cold, with defective circulation. In ACROCYANOSIS the fingers, hands and wrists, even the forearms are cold, blue and swollen. This malady is seen chiefly in young women. A similar condition affecting the ankles and legs is sometimes seen in girls—ERYTHROCYANOSIS CRURUM PUELLARUM. The feet often escape; occasionally the blueness extends as far up as the buttocks. These patients are usually stout, and chilblains are often present. The cause is obscure. Bazin's disease may accompany the blue state of the legs, but it is a separate entity (see § 647). In some cases there appears to be pituitary deficiency, in others thyroid dysfunction. In girls, thin stockings and undergarments may cause and aggravate the condition. Blueness of the extensor surface of the upper arms also occurs with pituitary deficiency and with infective foci, especially in middle-aged women. Cervical ribs have been a causal factor. Treatment consists in removing the cause, giving lime salts and endocrine preparations, and a systematic course of exercises, massage and a sinusoidal current. Acetylcholine injections also aid. In severe cases bilateral sympathetic ganglionectomy should be considered.

§ **577.** III. Of **Gangrene,** necrosis, or death of part of an extremity, there are two kinds:

(*a*) In DRY GANGRENE the extremity becomes white and cold, then of an ashy and black colour; the part shrivels up, becomes dry and mummified. It is *arterial* in origin due to gradual obliteration of the lumen by arteriosclerosis combined with more or less cardiac enfeeblement. It usually occurs as senile gangrene, but may be met in younger patients in Raynaud's disease, claudication and thrombo-angiitis obliterans, in ergot poisoning and in cases of embolic blocking of an artery. The artery is tender at the seat of the embolism, and ceases to pulsate below (§ 580).

(*b*) In MOIST GANGRENE the part becomes cold, purple, or mottled, and engorged with blood. Blebs then form on the surface, and a bright red line separates the dead from the living tissues. The dead part ultimately sloughs off, and leaves an ulcer. This gangrene is due to *venous* obstruction, the result of thrombosis, pressure, injury, or inflammation. The gangrene occurring in diabetes is of the moist variety and is usually associated with arteriosclerosis and infection.

The *treatment* of both of these conditions belongs to surgery: the pain and line of demarcation of dry gangrene are facilitated by cold air from a fan, or by surrounding the part with ice. Nicotinic acid (25 mgm. each 1–2 hours) improves the circulation by dilating the arterioles. A powder for local application containing penicillin (1,000 units) in sulphanilamide or sulphathiazole (G. 1), together with the systemic administration of these drugs, will often control infection. Some cases do well with periarterial sympathectomy. This is performed when, after paralysis of the vaso-motor nerves, the vessels can still dilate. This can be tested before operation by (*a*) giving a spinal anæsthetic or (*b*) causing an artificial temperature and recording the skin temperatures.

§ **578.** IV. **Trench Foot** is an affection of which the etiology is uncertain, but in which cold, wet, and more especially blood stasis appear to be causative factors.

Symptoms.—The onset is usually slow, following exposure. Then development of (1) numbness and later great pain and hyperæsthesia, confined as a rule to the feet but sometimes affecting the hands also; still later, (2) œdema of the feet, with, as a rule, blebs, ulcers or other cutaneous lesions; (3) these may eventuate in moist gangrene.

Frost-bite is due to more severe cold, especially at a high altitude and when combined with exposure to a high wind.

Symptoms.—(1) The onset is often sudden, with loss of sensation, including that of cold. (2) The skin becomes waxy pale, and the affected joints are stiffened. (3) Complete recovery may follow effective treatment, but the nails may be lost, some permanent anæsthesia may remain, and (4) dry or moist gangrene may eventuate. Recurrences are common on re-exposure.

Treatment of Trench-foot and Frost-bite. Prophylactic treatment consists in wearing loose warm dry clothing, with Balaclava helmets and warm gloves: two pairs of socks should be worn in well-fitting boots: all clothing must be windproof. Curative treatment must avoid the application of hot bottles or other warmth, and avoid rubbing with snow. The feet must be kept at rest, cleansed and wrapped in dry sterile dressings with plenty of cotton wool: hot drinks and sleep are essential, but alcohol avoided. Amputation must be performed for spreading gangrene or extensive sepsis if other measures fail.

§ 579. V. **Raynaud's Disease** (Synonyms: Symmetrical Gangrene, Local Asphyxia of the Extremities).—This disease, which was first described in 1862 by Raynaud, is characterised by local vascular changes in one or more of the fingers, for the most part symmetrically on the two sides of the body, resulting very often in gangrene. Occasionally the toes are involved. Three types or stages of the disease have been described—a syncopal type, due to vascular spasm; an asphyxial type, due to spasm of the arterioles and dilatation of the capillaries; and a gangrenous type.

Symptoms.—(1) First is noticed pallor (*local syncope*), coldness and numbness of one or more of the fingers or toes, usually the corresponding finger or toe on both sides, coming on in attacks, lasting an hour or more. This pale stage is generally followed by (2) coldness and local asphyxia, with cyanosis. (3) In the third or reactionary stage, there is considerable pain and swelling, and the tip of one or more of the fingers or toes, or the ears, may be dark purple. Sometimes the pale stage is very definite, sometimes it is absent, or so transient as to be unobserved. Occasionally the entire hands are involved. (4) After a number of these attacks, local ulcers and *gangrene* occur at the area affected; the dead becomes separated from the living part, and the ulcer that is left heals normally, but slowly. Cases have been recorded of extensive multiple gangrene in which the patient has lost entire limbs. The attacks described may be the only symptom, but in most cases other symptoms of considerable pathological interest may be observed. In some there is a generalised scleroderma, the skin appearing to be stretched and smooth, or sometimes cracked; in such cases all the fingers are pale and dead-looking, their entire substance becomes wasted and the nails may be lost. X-ray reveals atrophic changes in the bones. In other cases erythematous blotches occur from time to time in different parts of the body, leaving bruise-like stains. The patients are usually nervous, and prone to emotional attacks. Transient attacks of hemiplegia and aphasia have been observed, also of paroxysmal hæmoglobinuria, all pointing to vaso-motor irregularities. Effusion into the phalangeal and other joints may supervene, and may result in ankylosis.

The *Diagnosis* is usually simple. The earlier stages are allied to *erythromelalgia*, *sclerodactylia*, and to "dead hands," but these affections are not so localised to the fingers' *ends*, are less severe, and never go on to gangrene. Local vaso-motor symptoms, affecting usually only one arm, may be due to a *cervical rib*. *Phenobarbitone* poisoning produces similar symptoms.

Prognosis.—The disease runs a prolonged course of many years. The attacks become more prolonged and frequent, and the patient gradually becomes more and more helpless. There are many degrees of severity, ranging from a small localised syncope or asphyxia to gangrene of the entire segment of a limb. It is a curious circumstance that, in most cases, once a finger has become gangrenous the stump does not become similarly affected later on. The subjects of this malady in a marked form rarely reach old age, but usually die of some intercurrent malady.

Etiology.—The disease is more common in women, and especially those of a nervous temperament. It usually starts between the ages of fifteen and thirty. Attacks may be brought on by chill or mental disturbances. The condition is due to vaso-

motor spasm affecting the arterioles and capillaries. Later, permanent narrowing occurs, making treatment less successful.

Treatment.—The affected limbs must be kept warm, constipation corrected, and the patient protected from exposure to cold. Electricity should be tried; the best methods are a strong descending galvanic or a surging sinusoidal current. Histamine ionisation and massage are useful. Thyroid is beneficial and nitroglycerine has been used in the syncopal type: priscol tablets produce still better vaso-dilatation. The pain if intense may require morphia, which acts in a double way in asphyxial cases by giving tone to the vessels. Stellate ganglionectomy and lumbar sympathectomy are performed in severe cases with complete relief of symptoms. Operations must be done early, before the vessels become permanently narrowed, the most suitable cases being those with a good response to the thermal test (§ 577).

Dead Hands (Pallor of the Fingers).—Many patients—but particularly those who present other evidences of an inherent vaso-motor instability, and are subjects of the gouty or rheumatic diathesis—complain that the hands or finger-tips "go dead," or white, like those of a corpse, and feel numb and cold. These attacks, which rarely last very long, may happen in warm summer weather, without any obvious cause. This vascular disorder resembles the slight or early phase of Raynaud's disease. These attacks are not as a rule serious. As they often depend upon oral or gastro-intestinal sepsis, treatment is directed to the underlying cause, and massage and electricity are given locally.

§ 580. VI. **Intermittent Claudication** (Intermittent Limping) is a condition occurring chiefly in men. It is due to spasm or sclerosis of the arteries supplying the affected leg, usually of the smaller branches, but sometimes of the main trunks. X-ray shows if calcification is present and its site (§ 91). The symptoms come on when an extra local supply of blood is required, such as during walking: after a certain distance a cramp-like pain in one or both calves makes it difficult or impossible to proceed. The pain disappears on resting, but recurs when walking a similar distance; the legs may go cold, numb, and powerless. The pulse of distal vessels (*e.g.*, the dorsalis pedis) may cease to beat especially when an arterial thrombus complicates. Sometimes dry gangrene supervenes. Different causes occur: in many there is already considerable arterio-sclerosis: in others syphilitic arteritis or diabetes mellitus are contributory: tobacco has been blamed. *Thrombo-angiitis obliterans* (Buerger's disease) affects both veins and arteries, and is probably an allied toxic condition. When dependent, the leg and foot become purple; when elevated, they become white. *Prognosis:* The patient may live for years with periods of freedom, and die of intercurrent disease. *Treatment:* (i.) Treat the arterial sclerosis. This is only effectual in syphilis. Protein shock with typhoid vaccine has cured some cases. (ii.) Avoid undue exercise or fixation of the affected limb; (iii.) avoid cold and tobacco; (iv.) increase the peripheral blood-supply by vaso-dilators such as acetyl-choline and muscle extract (myosoton, lacarnol), warmth, contrast baths, diathermy, sinusoidal current and Buerger's passive exercises. Others benefit by placing the limb in an air-tight chamber and applying alternate pressure and suction by means of the Pavaex apparatus. Lumbar or periarterial sympathectomy and other forms of surgical intervention are called for when medical treatment fails and thermal tests show the vessels are capable of vaso-dilatation.

§ 581. VII. **Pink Disease** (syn. Erythrœdema) is a rare disease which runs a course of several months, with characteristic swelling and redness of the hands and feet, affecting children under four years of age.

Symptoms: (i.) There is often a preliminary stage with fever (100° to 102° F.), malaise, muscular weakness and loss of weight. For about a month irritability and sleeplessness, bouts of profuse sweating and severe itching of the skin are prominent. (ii.) In the second stage, also lasting about a month, the hands and feet swell, become pink or cyanosed and cold. Photophobia, rapid pulse, coryza, sweating, stomatitis and often vesicles with staphylococcal complications, are common. (iii.) About the

third month, recovery begins, but it is many months before the child becomes normal.

Diagnosis.—*Infantile paralysis* is usually suspected and the condition may not be recognised until the development of pink swelling of the extremities.

Etiology.—The disease affects children between 1½ and 3½ years old, the majority being between 9 and 18 months. Cases were recorded in London in 1898; most of the cases have occurred in Australia. The cause is unknown; vitamin deficiency or an infection have been suspected.

Prognosis.—There are no sequelæ and one attack confers immunity. Children nursed at home usually recover, but if taken to hospital there is great risk of infection, such as broncho-pneumonia and gastro-enteritis, which may prove fatal. Secondary infections of the skin such as pustules and onychia are common.

Treatment.—Nursing in the open air is recommended, with light clothing and sun bathing. Talc should be dusted over the extremities; painting with spirit prevents the development of pustules. For mouth lesions, the child likes to chew a swab soaked in hydrogen peroxide 1 in 4; a string ensures that this is not swallowed. The diet must have adequate vitamin content: raw liver is often valuable.

GROUP II. JOINT DISEASES

The methods of examination and exclusion of fallacies have already been described. Arthritic disorders may conveniently be grouped into acute and chronic:

Acute (§ 582).

I. Acute rheumatism.
II. Acute gout.
III. Acute gonorrhœal arthritis.
IV. Acute rheumatoid arthritis.
V. Septicæmia and Pyæmia.
VI. Acute specific fevers.
VII. Purpura, scurvy, hæmophilia, leukæmia.
VIII. Traumatism.
IX. Extension from adjacent bone.

Chronic (§ 585).

I. Subacute and Chronic infective arthritis.
II. Rheumatoid arthritis.
III. Osteo-arthritis.
IV. Chronic gout.
V. Spondylitis deformans.
VI. Gonorrhœal arthritis.
VII. Tuberculous synovitis.
VIII. Syphilitic arthritis.
IX. Hysterical joint disorder.
X. Neuropathic joint disease.

(*a*) *Acute Joint Diseases*

Acute joint diseases come on more or less abruptly, and are as a rule attended by the local and general signs of inflammation. Acute rheumatism is essentially an erratic polyarthritis from the commencement; acute gout usually affects a single joint; most of the other causes start in one joint, but (excepting VIII and IX) tend to a progressive involvement of others. It is worth noting that all the acute joint disorders (traumatism being excluded) are due either to some microbic process or to some other systemic disorder. These facts emphasise the necessity of investigating the constitutional symptoms, the viscera, and the blood.

§ **582. I. Acute Rheumatism** (Rheumatic Fever) is an acute febrile disease, with erratic painful swellings of the joints and a marked tendency to disease of the heart; running a prolonged course of many weeks if untreated, and followed by a great tendency to relapse. It is a disease

especially of childhood, when it is capable of many manifestations. Rheumatic fever tends to affect not only the joints, but also all the fibrous, serous and muscular tissues. The serous membranes of the joints, endocardium, and pericardium (which, it will be observed, histologically resemble each other) are the favourite situations of the inflammation. Acute rheumatism, unlike acute gout, attacks several joints, usually the larger ones—*e.g.*, the knees, ankles, shoulders. In adults it occurs only in a modified form, with polysynovitis as its most distinctive feature.

Symptoms.—(1) The fever, which may have been preceded by *tonsillitis* one to three weeks previously, comes on in the course of twenty-four hours, setting in before or at the same time as the joints are inflamed. It is of a continued type (Fig. 136), usually remaining about 102° or 103° F. for

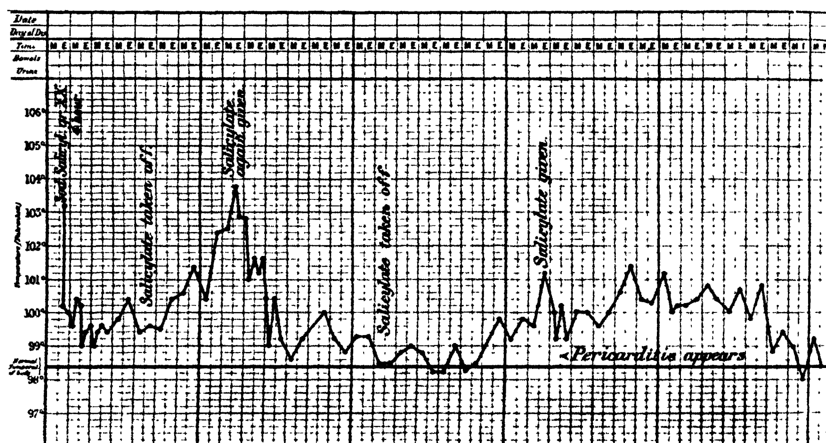


FIG. 136.—RHEUMATIC FEVER.—Henry H——, æt. twenty-two; the chart shows efficacy of salicylates in reducing the temperature until pericarditis appears; then the controlling power of the drug is less.

some days. The onset of any inflammatory complication in the pericardium or elsewhere is marked by renewed fever, pain, and sometimes delirium, the latter being otherwise *extremely rare* in acute rheumatism; in uncomplicated cases the mind remains quite clear throughout. The usual accompaniments of pyrexia are present—viz., the urine is scanty and highly coloured; the tongue is coated, the pulse quick and bounding, usually over 100. Hyperpyrexia occasionally occurs. (2) In adults there is a profuse *perspiration* with a sour disagreeable odour and an acid reaction, but in children this is unusual; later on sudaminal vesicles are frequently seen. Erythematous, purpuric, and other *rashes* occasionally appear. (3) The two distinguishing features of the *joint lesions* of acute rheumatism are their wandering or metastatic character, and the absence of suppuration. The effusion into a joint is not very great; first one joint is affected, but within a day or so, another is involved, the first joint having almost recovered;

finally several may be affected together. The joints are hot and swollen, and though not tender to the touch are often acutely painful on the slightest movement. The skin over the joints is either unaltered in colour or shows a faint flush. (4) *Peri-* or *endo-carditis* are other manifestations of the disease; the pericardium may be the first serous membrane to be affected. In 150 fatal cases analysed by Dr. F. J. Poynton, evidence of mitral endocarditis existed in 149. There is always some *dilatation of the heart* in rheumatic fever; *myocarditis* is commonly present, but rarely occurs as the sole cardiac lesion. In children the heart is chiefly affected and in comparison the joints but little. (5) *Rheumatic nodules* occasionally occur. They are small movable bodies, usually fibrinous, but may become fibrous. They are generally symmetrically placed on opposite sides of the body and appear on bony prominences and prominent tendons. The commonest places to find them are about the elbows, knees, malleoli, occipital curved lines, posterior spinous processes of the vertebræ, and knuckles. (6) *Chorea* may be the first and only sign of rheumatism, but is frequently followed by arthritis and endocarditis. (7) Pneumonia, pleurisy, and peritonitis, all occur rarely. (8) In untreated cases the fever and local inflammation may subside gradually in four to six weeks, and return again after an interval lasting, perhaps, a few days to a fortnight. Even after recovery the liability to recurrence is very great, and special care is needed. (9) Anæmia develops rapidly during the stage of fever, and may be severe in convalescence; in no other acute specific disease, excepting perhaps diphtheria, does the blood deteriorate in so short a time.

Two *variations* of the above symptom-group are met with clinically. In *subacute rheumatism* all the symptoms are milder, and may drag on for months. "Growing pains" may be the only symptom complained of by the child, and the cardiac infection may not reveal itself till years later. *Malignant rheumatic fever* is a very serious form in which the heart is mainly involved, the joints little if at all. An eruption something like typhus may appear, and after a few days the temperature rapidly rises and the patient dies.

The *Diagnosis* of rheumatic fever in the adult is not as a rule difficult. *Acute gout* is distinguished by its sudden onset, and by the other features mentioned in Table XL. *Acute rheumatoid arthritis* affects chiefly the larger joints or the fingers (§ 586); the swelling is fusiform, and does not subside under treatment by salicylates. *Septicæmia*, especially streptococcal, and *pyæmia* may closely resemble rheumatic fever. In these pyrexia shows a marked diurnal variation, often with rigors and delirium, skin rashes may be present, and leucocytosis is often higher than in rheumatic fever (in which it rarely exceeds 15,000). In any doubtful case, blood culture must be performed early. *Osteomyelitis* starting near the epiphysis may be mistaken for rheumatic fever. *Meningococcal septicæmia* causes confusion at first, as in the septicæmic stage flitting pains in the joints and muscles occur. The presence of marked headache, facial herpes and characteristic skin rashes help to distinguish this condition, and

a post-nasal culture usually grows meningococci. *Acute poliomyelitis* in children causes pains in limbs before paralysis sets in, pains in the neck with some stiffness, and the lymphocytic response in the C.S.F. helps to distinguish this condition. *Gonorrhœal arthritis* usually affects the knees or the small tarsal or carpal joints (see § 583. III and § 590); the condition is more chronic, and there is a history of gleet. Among the other diseases which sometimes have to be diagnosed are *dengue*, which has a characteristic eruption; *trichinosis*, in which the pain and swelling are referable rather to the muscles; in *infective endocarditis* the temperature is intermittent, salicylates are of little use, and a positive blood culture is obtained; *lymphatic leukæmia* may be difficult to distinguish until a blood count is performed. The secondary arthritis of *cerebro-spinal fever* and *dysentery* must be borne in mind. *Infantile scurvy* and *syphilitic epiphysitis* rarely occur after the age of two, whereas acute rheumatism is rare before three.

Prognosis.—The disease is not dangerous to life when it attacks the joints only, but when the heart is severely affected the prognosis is more grave. One attack predisposes to future attacks. Other untoward symptoms are hyperpyrexia and cerebral symptoms. The younger the patient the more likely is relapse to occur. An attack is grave in proportion to the height of the temperature, the involvement of the heart, and the presence of cerebral symptoms. The latter, happily rare, are of the gravest import unless accounted for by salicylates. The visceral manifestations of rheumatic fever are more serious than the disease itself. The chief of these relate to the heart, which should in all cases be examined daily as a matter of routine. Nodules are always of grave import.

Etiology.—Age is the most important predisposing factor, acute rheumatism being almost confined to persons under twenty-five, the commonest age being between seven and twenty. It does not occur under the age of two, and is extremely rare in advanced life. Dr. F. Langmead's investigations among school children revealed the fact that one in fifteen of those over seven years was rheumatic, and in 87 per cent. a cardiac lesion developed. Females seem slightly more prone to the disease, and heredity plays a considerable part. It is especially prevalent amongst the poor and the artisan class. Among the determining causes are exposure to cold or chill, and fatigue. Acute rheumatism is apt to follow tonsillitis, scarlatina, or chorea.

The occurrence of localised outbreaks of acute rheumatism in those living and sleeping in confined conditions, and especially following an epidemic of acute streptococcal sore throat, supports the view of its being a specific infective disease. In 1900 F. J. Poynton and Alexander Paine isolated a diplococcus from the blood exudates, and cardiac valves of rheumatic cases, which answered the tests of specificity. This has been corroborated by others, but still awaits general acceptance. Poynton believed the infection entered by the throat: but routine tonsillectomy is not a preventive.

Treatment.—Absolute rest in bed is essential: the patient should be between blankets to absorb perspiration, and allowed one pillow if it adds to his comfort. If severe carditis supervenes, the Fowler position may be the only one possible. Copious fluids must be taken as for pyrexia (§ 524). Sodium salicylate combined with at least an equal amount of sodium bicarbonate will relieve the joint pains and cause the temperature to fall almost to normal within 72 hours, provided large doses are adminis-

tered: usual doses for an adult are 20 gr. every two hours during the first two days, then smaller doses till the temperature is normal or toxic symptoms of the drug ensue—viz., headache, deafness and buzzing in the ears, vomiting, albuminuria or delirium. When salicylates fail to control the fever, either the diagnosis is incorrect or severe carditis (often with pericarditis) is present (Fig. 136). If the patient is in great pain and a rapid relief required, 1 gr. of sodium salicylate in 1 c.c. water for each year of a child's age (maximum dose 15 c.c. in adults) may be injected intravenously or intramuscularly once a day. Occasionally a case of undoubted acute rheumatism does fail to respond to salicylate therapy, when concentrated antiscarlatinal serum in 15 c.c. doses, repeated in 36 hours, may be given intramuscularly. Penicillin and sulphonamides are useless. Local remedies to relieve pain consist of wrapping the joints in cotton wool, or applying lead and opium lotion, oil of wintergreen or compound liniment of menthol (B.P.C.). To relieve the pain of pericarditis a leech or a blister with liq. iodi fort. over the precordium is of service. With hyperpyrexia a graduated hot bath or an ice pack may be required. Once the acute phase has subsided, before allowing extra activity it is important that there is no evidence of active carditis. The best guides are the absence of pyrexia or tachycardia, the heart should be normal in size and the murmurs at a stationary stage, the patient should be gaining ½–1 lb. in weight each week and the blood sedimentation rate should have returned to normal. Then slowly graduated extra activity is allowed; 6–9 months in bed are often necessary. During convalescence treatment is required to avoid relapses and second attacks. As *prophylaxis* the patient should wear flannel and avoid exposure: by preventing streptococcal throats, good results have been recorded with sulphadiazine G. 1 daily in the winter and spring months. When school children suffer with "growing-pains" or tonsillitis, a strict watch should be kept on them, and rest in bed ordered if the heart shows any suspicious signs.

§ 583. II. **Acute Gout.**—Gout is a diseased or disordered metabolism associated with excess of uric acid in the blood, and characterised by recurrent attacks of acute inflammation of the joints with deposition of sodium biurate. It is one of the oldest known diseases, but is at the present time rare. Gout occurs in acute, chronic, and irregular forms.

The *Symptoms* of an attack of acute gout are usually preceded by dyspepsia, heartburn, flatulence, and weariness after food, indentation of the tongue, a bad taste in the mouth, and scanty, high-coloured urine constantly depositing urates, cardiac irregularities, restlessness at night, and a tendency to catarrh of the mucous membranes on the slightest exposure. The temper is proverbially irritable. The onset of an attack is usually *very sudden*, often in the middle of the night. It affects preferably one of the smaller joints, and especially the metatarso-phalangeal joint of the big toe. The swelling is tense, shining, bluish red, pits on pressure, and is acutely tender, but suppuration never occurs. Other joints may become affected, but the inflammation does not shift from

one joint to another as in acute rheumatism. Mild constitutional symptoms are present with pyrexia (102°); there may be muttering delirium at night. The urine contains less uric and phosphoric acid before the attack, and more during it, and may contain a trace of albumen. An attack lasts two to three days or two to three weeks, and is usually followed by improved health; but the intervals between the attacks gradually become shorter, at first two or three years, then one year, then six months; finally the disease becomes chronic, and permanent changes take place in the joint. For *chronic gout* and its associated symptoms, see § 588.

Varieties.—(1) In *Subacute gout* the attack is milder, and there are wandering pains resembling fibrositis, without joint involvement. So-called *irregular gout* consists chiefly of a variety of symptoms referable to various organs, supposed to be due to a deposit of sodium biurate. These are now considered to be due to septic foci occurring in gouty subjects. (2) *Suppressed gout* was a term applied to cases where the joint mischief suddenly improves coincident with internal symptoms affecting the digestive tract, the heart or the brain. There may be vomiting and diarrhœa, dyspnœa, arrhythmia, pericarditis, delirium and coma, or cerebral hæmorrhage. These are now regarded as due to uræmia or cardiac failure.

The *Diagnosis* is not difficult except sometimes from acute rheumatism. There is increase of uric acid in the blood, especially just before the acute attack, up to 6–9 mgm. per cent. (normal, 1 to 4 mgm.). The blood sedimentation rate is raised in both diseases.

TABLE XLA.—DIAGNOSIS BETWEEN ACUTE GOUT AND ACUTE RHEUMATISM.

Acute Gout.	*Acute Rheumatism.*
In typical cases: Middle age; male sex.	In typical cases: Youth; either sex.
Preference for smaller joints; never wandering from joint to joint.	Preference for larger joints; usually wandering from joint to joint.
Swelling is usually red, tense, pitting on pressure, acutely tender. Pain persists during rest.	Swelling is hot, but pale; pain only on pressure or movement of joint.
Ears may show tophi.	No tophi.
Fever may be slight or transient.	Fever always marked and continuous.

Prognosis.—The duration of an attack—which is never fatal in itself—depends upon the age and constitutional condition of the patient. Attacks tend to recur in the same joint. Gout tends to shorten life mainly by the resulting kidney disease and cardio-vascular changes, and the ultimate prognosis largely depends upon the condition of the

urine, which should be of good specific gravity and free from albumen. Among the complications of gout (1) chronic renal disease is the most important. During an attack there is generally a certain amount of albuminuria, but this passes off. Gradually, however, after repeated attacks, an interstitial fibrosis takes place in the kidney giving rise to the gouty kidney. Glycosuria occasionally occurs. Renal calculus may occur in persons of the gouty diathesis who may have escaped joint symptoms. (2) Cardiac and cardio-vascular diseases come next in frequency. The "gouty" heart is "irritable," acts irregularly, causing palpitation, tachycardia, pain, and often great distress. Various valvular lesions and arterial disease may develop. (3) Chronic bronchitis is frequent, and in treating this the gouty condition must not be forgotten. (4) Various gastric and hepatic derangements are frequent. (5) Eczema, hyperkeratoses and fissured nails. Hot, itchy eyeballs, migraine, and episcleritis; glaucoma and iritis also occur.

Etiology. Among the *predisposing* causes of gout age, habits, sex and heredity are important. (1) The disease is more frequent between thirty and fifty; it is rare under thirty, and it is uncommon to see it start after fifty. (2) Gout is almost confined to men; if it occurs in women, the attacks are generally slight. (3) Out of 520 cases collected by Sir Alfred Garrod, 332 were distinctly hereditary. The predisposition is transmitted mainly through the male line; but rarely it may be transmitted by an unaffected female, and reappears in the sons. (4) Lead in the system; painters, glaziers, etc., are prone to gout and gouty kidney. (5) Attacks are more frequent in the changeable weather of spring and autumn. *Exciting* causes are (1) alcohol, especially forms containing a high percentage of sugar and alcohol, such as port wine, brown sherry, Madeira, sweet wines generally, and malt liquor. (2) Nitrogenous food in excess, especially the purin-containing foods; gout is commoner in meat eaters and in those who over-eat. (3) Too little exercise and fresh air reinforce these two causes. (4) Gout is very rare among Scottish artisans; a possible explanation is that the beverage of the Scottish artisan is whisky, while that of the English workman is beer, which contains over a grain per pint of purin bodies. An attack may be determined by (1) a debauch of alcohol; (2) indigestion; (3) chill; (4) severe mental or bodily fatigue; (5) injury may determine the particular part affected; (6) attacks are often precipitated by catarrhal infections of the nose and throat.

Treatment during an attack.—(1) A low diet of milk and farinaceous food, and complete abstinence from alcohol should be enjoined, unless the heart be weak, when well-diluted matured whisky is the only form permissible. (2) A brisk cathartic with one or more grains of calomel should be given at the onset, followed by frequent doses of saline purgatives, such as Hunyadi Janos or Carlsbad water. (3) Alkaline carbonates (potassium, lithium, sodium) promote the solution of uric acid. (4) Colchicum (combined with alkaline carbonate) is a specific, and it may be given every four hours as the tincture (♏ 40 for the first dose, then ♏ 12)

until the pain has gone; then it should be stopped. If taken too long it acts as an irritant poison. (5) Aspirin in full and repeated doses relieves pain and promotes elimination of uric acid: otherwise morphia may be needed. (6) Local treatment consists of complete rest, wrapping the joint in cotton-wool, and the application of sedatives, such as lotions of sodium bicarbonate gr. 240, with tinct. opii ℳ 120 in fl. oz. 10 of water. A very comforting lotion consists of sp. vin. rect.; liq. ammon. acet.; aq. rosæ āā ℳ 180; aq. ad. fl. oz. 12. When symptoms of suppressed gout come on, employ eliminatory treatment promptly, stimulate if symptoms of collapse follow, and apply counter-irritation (mustard, turpentine stupes) and hot fomentations to the chest or abdomen as the case demands.

Treatment between the attacks—i.e., preventive treatment—resolves itself mainly into a question of diet (see § 297. XVI). Open-air exercise is advisable. A valuable drug is cinchophen B.P. (atophan). It is useless for pain during an attack, but it increases elimination of uric acid in the urine later. 7½-grain doses thrice daily for two consecutive days in the week do as much as continuous doses over a longer period; and toxic effects are avoided. Alkalies and glucose should be taken at the same time. Stop it if dyspepsia or a rash appear, as toxic hepatitis may develop. An occasional dose of mercury followed by a saline purge is useful. Colchicum is of use in the subacute exacerbations, when it may be given with large doses of potassium iodide until pain is relieved. Mineral waters (Carlsbad, Vichy, Hunyadi Janos, Friedrichshall) can be freely taken. Lithium and potassium urate are more soluble than sodium urate, and some remain free of gouty symptoms when taking potassium chloride instead of common salt with meals. Visits to Bath, Harrogate, Buxton, Strathpeffer, Carlsbad, Royat, and Aix-les-Bains are undoubtedly beneficial.

III. **Acute Gonorrhœal Arthritis** (Gonorrhœal Rheumatism) is an acute arthritis resembling "rheumatic fever," due to infection by the gonococcus from the urethra during the acute stage of gonorrhœa. It is more frequently met with in the chronic form described in § 590. There is a discharge or a history of it. If the disease arises in the acute stage of gonorrhœa, the joint mischief resembles acute rheumatism in all respects excepting: (1) Although the inflammation spreads from joint to joint, those first involved do not get better as the others become affected; (2) the temperature is more intermittent than with ordinary acute rheumatism; (3) it does not yield to salicylates, but rapidly responds to penicillin injections; and (4) there is much less tendency to heart complications. The joints rarely suppurate, but in the absence of chemotherapy extensive adhesions and deformities may arise. It occurs even in children. Treatment is discussed in § 590.

Reiter's disease is an acute polyarthritis and urethritis, often with conjunctivitis. Its cause is unknown but it is often confused with acute gonococcal arthritis: it is not amenable to treatment by sulphonamides or penicillin.

IV. **Acute Rheumatoid Arthritis** may start in a manner indistinguish-

able from acute rheumatism, but the joint swellings persist and become more typically those of rheumatoid arthritis later (§ 586).

V. **Septicæmia** and **Pyæmia** have been described in § 515. In some cases of acute general infection the joints are not involved, but in others there is a marked tendency to inflammation in and around the joints. This is not infrequent with infective endocarditis. It is differentiated from other joint lesions by: (1) the swelling does not shift its position, as it does in rheumatism; (2) the joint may be red and show evidences of suppuration; (3) the constitutional symptoms are characteristic, especially the wide and irregular temperature, often rigors and sweatings, and the leucocytosis is usually above 15,000; (4) a cause may be revealed in the shape of an internal or external pyogenic focus.

VI. Other **acute specific diseases** may lead to arthritis. The joint disease can be identified only by the presence or history of the disease which it complicates. In *adults* pneumonia and typhoid fever may be complicated or followed by a suppurative affection of the joints, often with a fatal issue. Less often other acute specific fevers are so complicated. In dengue joint swelling is often part of the disease; in cerebro-spinal meningitis and Mediterranean fever the joints are often affected. In *children* the joints may be affected in the early stages of acute poliomyelitis: measles, typhoid, mumps, and influenza are rarer causes. Synovitis sometimes follows the administration of antitoxins, *i.e.*, serum disease (§ 521).

VII. There are four remaining generalised disorders associated with joint trouble—viz., Purpura Rheumatica, Scurvy, Hæmophilia, and Leukæmia.

§ **584. Purpura Rheumatica** (Syn., Peliosis Rheumatica, Schönlein's Disease).—In this disease pain in the joints is not due to rheumatic fever but to an infective condition associated with an eruption of the skin (§ 653). It is chiefly a disease of male children, between 3 and 12 years of age, but can occur in adults. Relapse is common.

Symptoms.—(1) There is an initial pyrexia, rarely above 100°, accompanied in most cases by a hæmolytic streptococcal throat infection. (2) Within the next 7 days joint symptoms occur. These vary from transient swelling around one joint to an acute painful swelling of many joints. (3) The eruption is often the first symptom: it is present as a maculo-papular rash on the extensor surfaces of the elbows and forearms, or of the lower legs, and may affect the buttocks. It is roughly symmetrical, itching at first, and may leave brown staining. Petechiæ may occur. (4) Acute nephritis, often hæmorrhagic, is common and may lead to subacute and chronic nephritis. (5) True rheumatic fever with carditis may ensue. (6) Henoch's purpura may co-exist and give acute abdominal symptoms. (7) The blood may show an increase in polymorphs and/or eosinophils, but unlike purpura hæmorrhagica, the blood platelets are normal, as are the bleeding and clotting times. *Etiology.*—There is an aseptic inflammation of the smaller blood vessels of the affected areas, usually caused by a hæmolytic streptococcus. *Treatment.*—Intravenous calcium gluconate and subcut. adrenalin may be tried. Penicillin, the sulphonamides and salicylates seem to be valueless.

HENOCH'S PURPURA ABDOMINALIS is closely allied to purpura rheumatica, and all or any of the symptoms of this disease may be present. The chief *Symptom* is severe colic, occurring independently of diet, perhaps associated with diarrhœa and blood or blood-stained stools. Vomiting may be severe, with or without blood, and a tumour

due to hæmorrhage into the submucous coats of the intestine may occur—especially in the lower ileum. The condition is apt to be mistaken for intussusception—this complication or sometimes perforation may occur. The attacks recur at intervals for years. For *treatment*, see purpura rheumatica.

In SCURVY (§ 545) tender non-suppurative swellings occur beneath the periosteum near the joints, but the joints themselves are not often affected. The disease is recognised by the spongy bleeding gums, anæmia, and other symptoms of scurvy.

In HÆMOPHILIA (§ 550) the larger joints are usually affected. The joint lesion is probably always due to the extravasation of blood into the joint cavities, and usually supervenes suddenly on a slight blow or exposure to chill. It not infrequently recurs, and may ultimately lead to ankylosis. It is diagnosed mainly by the history of hæmorrhages in the patient and by the family history. The condition usually starts between the ages of seven and fourteen and only affects males.

In ACUTE LEUKÆMIA joint symptoms may predominate in the earlier stages. Swelling is usually slight, but there is considerable pain, with limitation of movement and some pyrexia, which make the distinction from acute rheumatism difficult. The pain may be due to small purpuric hæmorrhages into the joints. Sooner or later purpuric retinitis, generalised purpura, enlargement of lymphatic glands and of the spleen aid diagnosis. The ends of the bones may present a frosted glass appearance on X-Ray examination, and a blood count is usually diagnostic.

VIII. **Acute Traumatic Synovitis** is recognised by the history of an injury, though one must bear in mind (1) that many constitutional processes, especially gout, are lighted up by a very slight injury, and (2) that in childhood the history of traumatism may be wanting.

IX. **Extension** from epiphysitis or osteomyelitis (§ 595) or other bone disease in childhood—set up very likely by injury—may produce acute inflammation in a joint, and the serious nature of the condition may be overlooked unless the correct meaning of the pyrexia and constitutional disturbance is appreciated.

(*b*) *Chronic Joint Diseases*

Joint disorders which may be chronic *ab initio* come clinically under ten headings.

I. Infective arthritis. § 585.
II. Rheumatoid arthritis. § 586.
III. Osteo-arthritis. § 587.
IV. Chronic gout. § 588.
V. Spondylitis deformans. § 589.
VI. Chronic gonorrhœal arthritis. § 590.
VII. Tuberculous joint disease. § 591.
VIII. Syphilitic arthritis.
IX. Hysterical joint affection.
X. Neuropathic arthritis (*e.g.*, Tabes, Syringomyelia, and Raynaud's disease)

Clinically, many of these joint diseases resemble each other very closely, both in their physical signs and their history, and many cases are met with which it is almost impossible to place definitely under one or other classification. Moreover, in their pathology we find the same resemblance, for with the possible exception of hysterical and neuropathic arthritis they are all of microbic or metabolic origin.

§ **585.** I. **Infective Arthritis** is a common affection of the larger joints and occurs equally in men and women. It is often called "chronic rheumatism." The disease may follow one or more acute attacks, or, as is more usual, come on insidiously as a chronic affection from the beginning. It is met with especially in persons past middle life. If it begins acutely, it is often accompanied by muscular and neuritic pains. Usually one joint is affected first, and others are involved later. In the acute phases, the joint is more or less swollen, tender, and painful on movement. Then

it becomes stiff, due to adhesions in it. In the acute cases there is a mild degree of pyrexia and sometimes other evidence of toxæmia, such as anæmia. The disease is never fatal.

The *diagnosis* from *chronic gout* is sometimes difficult; there are no tophi nor any constitutional evidences of gout. It is distinguished from *osteo-arthritis* by the absence of lipping and other characteristic bony changes. In *rheumatoid arthritis* there is a greater tendency for multiple involvement of the smaller joints, and the changes are periarticular. In obscure cases, X-rays aid the diagnosis. X-rays show a condition of general absorption of lime salts in the ends of the bones adjoining the infected joints and loss of definition in the joint surfaces; in advanced cases osteo-arthritic changes supervene.

Etiology.—The condition is due to toxic changes resulting from a septic focus; the streptococcus viridans is the usual causal organism. At the climacteric the lowered resistance is a predisposing factor. During convalescence from dysentery, arthritis may develop.

Treatment is discussed under the treatment of arthritis (§ 587).

§ **586.** II. **Rheumatoid Arthritis** is a general disease, producing synovitis and peri-arthritis, as shown by swelling and pain in the joints. It tends to get well, often after a protracted course, but leaves, not uncommonly, considerable deformity and crippling. It manifests also symptoms of constitutional disturbance.



Fig. 137.—Rheumatoid Arthritis in a woman, showing deformity of wrists and fingers.

Symptoms.—The onset may be acute, subacute, or chronic. In the first form the condition closely resembles acute rheumatism, but the joints prove intractable to the action of salicylates, and later assume the typical characters. In the subacute variety the joints are rapidly affected, but show only slight swelling at first, whilst the temperature is but little raised. The chronic form begins insidiously in one joint, and spreads slowly.

1. The joints usually affected first are the proximal row of the inter-phalangeal joints of the fingers, and the metacarpo-phalangeal joints (Fig.

137); next the wrists, ankles, and knees; then the shoulders, and last of all the hips, so that the progression of the disease in the joints is from the periphery. The lesions are usually symmetrical. The distal interphalangeal joints are usually spared. During the active stage the joints are painful, tender, and swollen, and somewhat limited in movement. The swelling is fusiform, due to the fact that the lesion is a combination of synovitis and peri-arthritis; and there are neither lipping of bone nor osteophytes to be felt, nor can grating be elicited. If the active stage is of long duration, this may be followed later by very marked limitation of movement and deformity, due to the formation of adhesions around the joint, and to the secondary contracture of muscles. In severe cases partial dislocation or ankylosis may occur. The most common displacement is that of ulnar deviation of the fingers. The muscles above and below the affected joints are conspicuously atrophied, to a much greater extent than could be explained by disuse. The tendon reflexes are increased.

2. Subcutaneous nodules are sometimes present. Usually these are in the form of flat masses in bursæ, especially the olecranon bursa; but more rarely they resemble the nodules of rheumatism, differing from them in being more permanent and occasionally tender.

TABLE XLI.—TABLE OF DIAGNOSIS.

Infective Arthritis.	*Chronic Gout.*	*Rheumatoid Arthritis.*	*Osteo-arthritis.*
Either sex; middle life or over.	Generally male sex; over forty.	Chiefly female sex; usually twenty to forty.	Either sex: the hips in men, the knees in women; forty to sixty
Poor and debilitated. Insidious onset.	Rich and plethoric generally. History of sudden onset and acute attacks with severe pain. Skin over joints red, swollen, and œdematous.	More common in the poor. Onset acute, subacute, or insidious. Constitutional symptoms present with pyrexia, anæmia, loss of weight.	Onset, insidious; course, progressive. No constitutional symptoms.
Tends to affect the larger joints: hips, knees, wrists, elbows and shoulders.	Only one joint affected at first; usually the metatarso-phalangeal of the great toe. Usually asymmetrical.	Generally polyarticular. Temporo-maxillary joint may be affected. Spreads from the smaller joints to the larger; proximal interphalangeal joints usually affected. Usually symmetrical.	Polyarticular or mono articular. Temporo-maxillary joint rarely, if ever, affected.
Thickening of synovial membrane; marked rarefaction of bones and irregular overgrowth around the joints.	Deposits of urate of soda round the joints. Blood uric acid increased.	Spindle-shaped enlargement with ulnar deviation and later some fixation. No lipping or osteophytes in early stages.	Radial deviation of terminal phalanges. Lipping and osteophytes marked. Cartilage and bone absorbed.

3. In the early stages the skin of the hands and feet is often cold and clammy; later it becomes glossy, atrophic, and often parchment-like on the backs of the hands and fingers. Pigmentation is common, occurring

as circumscribed spots like freckles, or as diffuse spreading patches, especially on the face and neck, and on the backs of the wrists and fore-arms, but it may be general. The forehead may shine like burnished bronze and various tints of yellow and brown are seen by reflected light at different angles. A brawny œdema of feet and legs may be present, independent of cardiac or renal disease.

4. The axillary, epitrochlear and inguinal glands are not uncommonly swollen.

5. Constitutional symptoms. There is usually fever during the active stage, the temperature varying from normal to as high as 102° or 103° F. The pulse is nearly always quickened, and may be from 90 to 100 for years. The general nutrition is impaired. There is marked loss of weight. Patients with this disease are usually delicate from childhood, with frequent "bilious attacks"; their glucose tolerance is low.

The *Diagnosis* is considered in Table XLI. X-rays show narrowing of the joint space with rarefaction of the bones adjoining, due to absorption of lime salts (Fig. 138*d*). The blood sedimentation rate is often raised to a high level in rheumatoid arthritis, but is rarely raised in osteo-arthritis.

Climacteric (menopausal) **arthritis** occurs in women between forty and fifty-five years of age, and involves principally the knees, and to a lesser extent the wrists and fingers. In the early stage there is swelling, due to synovitis, and pain; in the knee this is usually on the inner side. Later, osteo-arthritic changes may supervene. Obesity is usually present, with signs of hypothyroidism; in some cases an infective element co-exists.

In *children* certain forms of arthritis occur which resemble the lesions of rheumatoid arthritis in adults (E. C. Warner, *The Practitioner*, Feb. 1935, p. 170). The multiple arthritis described by Dr. G. F. Still (Still's disease), associated with pallor, fever, wasting, and enlargement of the lymphatic glands and spleen, differs from the description given above only in the frequency of affection of the glands and the splenic enlargement. It is probably rheumatoid arthritis modified by the age at which it occurs.

Prognosis.—The prognosis of rheumatoid arthritis has become more hopeful of recent years. This is especially true if the case is seen early and an infecting focus can be traced and dealt with. In many cases no source of infection can be traced. When the glucose tolerance is greatly lowered and the source of infection cannot be removed, the outlook is serious; this may occur when the mucous membrane of the intestine is involved. The course is slow, with many relapses, and there is much crippling and deformity unless adequate local treatment can be secured early. The disease is rarely fatal.

Etiology.—The disease may occur at any age, but is most common between twenty and forty. Females are more often affected than males, in the proportion of about three to one. A lowered resistance frequently precedes an attack, which may in this way follow acute infections, especially influenza, or overwork and anxiety. Apical abscesses, pyorrhœa alveolaris, septic conditions of the nose, antrum, sinuses, ears and throat, tonsils,

gall-bladder or digestive tract, pelvic disease, cystitis, colitis, and ulcerating piles may act as contributory causes, but that they are not the real cause is shown by the fact that dealing with such does not necessarily bring about lasting improvement.

Treatment is discussed in § 587.

§ **587.** III. **Osteo-arthritis** is a chronic degenerative disease of joints, progressive in character, and occurring chiefly in the elderly.

Symptoms.—The special features of the joints are as follows: The articular cartilage proliferates and degenerates; the ends of the bones are thickened and lipped. Bony outgrowths or osteophytes are formed, often in great quantity, so that if the joint is moved, scrunching or grating is audible, and by their interlocking, movement is much restricted. True ankylosis rarely occurs. The synovial membrane is affected later; thickened fringes can be felt, in some of which cartilaginous bodies are recognisable. These may be pedunculated or free, forming the so-called melon seed bodies. The joint is often distended with fluid, as are also the bursæ around it. Sometimes the encysted collections of fluid near the joint are unconnected with bursæ, but are lying in spaces bounded by muscles and areolar tissue. Pain is not usually severe, but the joints often feel hot and tingling, and occasionally numb. In severe cases considerable deformity results from absorption of the ends of the bones, so that shortening or displacement is produced. There is no constitutional disturbance in this disease; the patient often looks robust. Muscular atrophy is less marked than in rheumatoid arthritis, and does not lead to the same crippling by contracture. X-ray shows characteristic lipping of the bone edges and in advanced stages gross destruction of the joint (Fig. 138c).

The disease may affect one or many joints.

1. *Heberden's Nodes* form the commonest and best known variety. These are bony growths, which occur in symmetrical fashion at the sides of the distal interphalangeal joints of the hands. They are usually painless, but may be painful, and produce numbness and tingling in the fingers. Little bursal swellings occasionally accompany them. In advanced cases the terminal phalanges are bent acutely toward the radial side. This condition is much more common in women, may exist alone as evidence of osteo-arthritis, but often accompanies other varieties.

2. *The Carpo-metacarpal Joints of the Thumbs* are not infrequently affected alone, or with Heberden's nodes. The joints are loose and grate, and the bones can be felt to be lipped.

3. *The Knees* are frequently affected in women at the menopause. Some regard this condition as an infective arthritis. Pain and stiffness are noticed on walking or going downstairs, and the knees give way

4. *The Temporo-maxillary Joint* is rarely affected, and then generally only after trauma, such as fracture of the mandible.

5. *The Hip-Joint of Elderly Men.*—This is the most important local form of the disease, since it leads to considerable crippling. It is often unilateral. There are pain and rigidity of one hip-joint with difficulty in

abduction. The pain is felt most severely in the groin, but may radiate down the outer side of the thigh to the knee. From sciatica, with which it is often confused, it is distinguished by the position of the pain, and the fixity of the joint. Wasting may occur later, but is limited to the buttock and thigh. The limb may be shortened. It occurs chiefly in men over fifty, often as a result of previous minor injuries.

6. *The Generalised Form.*—In this condition most of the joints in the body may be attacked, including those of the spine. In the hands, the distal interphalangeal joints and the carpo-metacarpal joints of the thumbs are usually selected, and show the characteristic grating and lipping, not the fusiform swelling of rheumatoid arthritis.

Prognosis.—If treated early, temporary improvement may occur, but speaking broadly, the disease is progressive. The form occurring in the hip-joint of old men is very intractable, but that in the knees of women at the menopause more remediable. The crippling is not great, but patients with the joints of the lower extremities affected will often be afraid to get about, because of the fear of the knees giving way.

Etiology.—It occurs most often in men, and between forty and sixty years of age. It is doubtful whether the joint lesions are in any way specific, since similar changes occur as the result of traumatism, fractures, or of prolonged pressure as by a tight boot, in hæmophilia from repeated hæmorrhages, and in tabes dorsalis and syringomyelia.

Treatment of Arthritis.—Certain principles of treatment apply to all types of arthritis, but must be modified according to the clinical condition. (*a*) The *relief of pain* is first attempted. In infective, rheumatoid and gouty arthritis the patient should be kept in bed in the acute stages. It is unwise to do so with osteo-arthritis unless there is acute pain and swelling of the joint, perhaps due to the nipping of a synovial fringe. Whenever possible, patients with osteo-arthritis should be allowed daily exercise. Drugs of value for the relief of pain include salicylates, guaiacol carbonate, and for gout, colchicum. Potassium iodide or liq. iodi aquosus can be taken over a long period; the latter is given in 1-drop doses in milk, thrice daily, cautiously increased to 30 or 40 drops, then decreased, and again increased. Local applications such as Scott's dressing, a kaolin poultice, and liniments containing menthol, camphor, and methyl salicylate may be used. Heat is very welcome, and may be given as radiant heat, diathermy, paraffin wax baths or infra-red rays. Radiant heat baths do so much good to chronic stiffened joints that it is probable the benefit is due to the light as much as the heat. The pyretic couch treatment has also many advocates. Dr. Luff's iodine vapour bath relieves. The joint is washed and dried, the skin painted with liq. iodi mitis, and covered with a thin layer of butter-muslin; a linseed poultice is placed over this, and the part smothered in cotton-wool. Ionisation with sodium salicylate softens adhesions. Histamine ionisation is valuable; it should not be given in infective or acute rheumatoid arthritis, nor where there is cardiac weakness. Deep X-ray therapy is useful for osteo-arthritis, especially

of the knees, hips and spine. (*b*) *Prevention of deformities* is accomplished by suitable splints. The upper arms should be at a right angle; the elbow at a little less than a right angle; the wrist supinated and dorsiflexed; the fingers nearly extended. The hips and knees should be extended, the feet dorsiflexed. When pain has diminished, massage and weak doses of galvanism are indicated to prevent wasting and fixation. (*c*) The *diet* should be nourishing and abundant, especially in infective and rheumatoid arthritis. In the latter, wasting is often a prominent feature, and a high calorie diet is essential, such as 2,500 calories to a 10-stone person. The meat is limited to ½ gramme per lb. per day, and normal amounts of carbohydrate allowed; fats make up the remaining calories. Large quantities of fats, milk, eggs, fruit and green vegetables should be given. To help the metabolism of sugar, insulin in doses of 5 to 10 units twice a day may be given. In gout the diet (§ 297. XVI) must be sparing and contain little protein and carbohydrate, and no sweetbreads, liver or kidneys. Alcohol is forbidden, especially in the form of heavy or sweet wines, beers and spirits. (*d*) Abundant *fluids* must be taken, but no large quantities of very hard waters allowed. Spa treatment as at Bath, Buxton, Harrogate, Aix-les-Bains, etc., is partly of value for the water drunk, and also for the regulation of diet and exercise. Adequate excretion must also be attended to, and colon lavage or suitable aperients given as required. (*e*) Cases do best in a warm and dry *climate*; such is obtained by residence on the South Coast or Egypt. (*f*) An *infective focus* must always be sought for in infective arthritis. Common foci are the teeth, tonsils, sinuses, cervix or urethra, prostate, or occasionally in the alimentary tract. Two or three large doses of penicillin are given over the period of extraction of teeth or removal of tonsils, to avoid reactions in the joints (§ 209). Vaccines in small doses may be beneficial in raising the general resistance to a particular organism. In osteo-arthritis rheumatoid arthritis and gout, the removal of a septic focus will be of value in raising the general resistance, but wholesale removal of apparently normal teeth, tonsils, etc., is of no value unless special investigations by radiological and bacteriological examinations have implicated them. (*g*) In rheumatoid arthritis and infective arthritis, *intravenous medication* with protein shock (typhoid bacilli 80 to 150 million), or other preparations, such as sodium thiosulphate, sulfosin or colloidal iodine is advocated by some. (*h*) *Thyroid* and *stilbœstrol* help menopausal arthritis. (*i*) After the active stage has passed in rheumatoid and infective arthritis, *mobilisation* of the joints and correction of deformities by manipulation under an anæsthetic, followed by vigorous exercises and massage, is often essential This should never be done without a preliminary X-ray examination. (*j*) Tonics such as iron, arsenic or thyroid will hasten convalescence. (*k*) In cases of rheumatoid and infective arthritis with anæmia, a blood transfusion is often valuable. (*l*) Gold salts (myocrisin, solganal) especially in oily solutions, can be given intramuscularly for infective and rheumatoid arthritis, after foci of infection have been dealt with. Doses at weekly

intervals are increased from 0·01 to ·10 G., until a total of 1·0 G. has been given. Second and third courses are often required after an interval of 6–8 weeks, especially when the blood sedimentation rate remains high. The patient's condition is aggravated if a fresh dose is given before the reaction due to the preceding one has subsided: before each dose, toxic phenomena such as stomatitis, skin rashes, albuminuria and symptoms of agranulocytosis should be looked for. The simultaneous administration of 10 c.c. of calcium gluconate helps to avoid reactions. (*m*) The remarkable value of injections of cortisone (Compound E) in rheumatoid arthritis, is being further investigated.

§ **588.** IV. **Chronic Gout** usually supervenes upon a succession of acute attacks (§ 583); occasionally it is chronic or subacute from the beginning. The joint is stiff and painful on movement, is very tender, sometimes red; sometimes urate of soda (chalk stones) can be seen under the skin and may discharge through it, leaving chronic scars or sinuses. Secondary osteo-arthritis commonly ensues. The patient, usually a male over middle age, suffers from gouty dyspepsia, irritability of temper, and frequent subacute exacerbations of joint trouble. Tophi are usually present. They consist of nodules of sodium biurate, analogous to the deposits in the joints, and are commonly situated in the cartilage of the ear, near the helix, and in bursal sacs. The urine may contain a little albumen from time to time. Arterio-sclerosis and hypertension are usual.

The *Diagnosis* between chronic rheumatism and chronic gout is by no means easy. In general terms, chronic gout attacks the smaller joints, the patient is of a plethoric type, and there are concurrent symptoms such as tophi, arterio-sclerotic kidney, or the history of paroxysms of acute gout. A gouty diathesis is diagnosed when the blood uric acid is over 4 mg. per cent. (see also table, § 586). X-ray shows a general rarefaction of the bones near the joints, with small translucent areas where the bone is replaced by urate crystals. Gouty deposits may occur in joints affected by rheumatoid and infective arthritis. Later, osteo-arthritic changes are added (Fig. 138*a*).

The *Prognosis* of chronic gout is more serious than that of chronic rheumatism, though in both the same crippling of the joints occurs. Arterio-sclerotic kidney is almost sure to supervene sooner or later, and the prognosis mainly depends on three factors: (i.) the condition of the kidneys; (ii.) the degree of hypertension; and (iii.) the condition of the myocardium. The complications in addition to those mentioned under Acute Gout are bronchitis, iritis and scleritis; and deposits of urates in many tissues, and sometimes glycosuria are seen. The patient may eventually die with uræmia, pericarditis, pleurisy, or cerebral hæmorrhage. *Treatment* is described under Acute Gout and in § 587.

§ **589.** V. **Spondylitis Deformans** (Synonym: Spondylitis rhizo-mélique of Marie) is a disease formerly classed under rheumatoid arthritis, but now recognised as a separate morbid entity, first involving the sacro-iliac joints. The vertebral column and the shoulder and hip joints are most often affected.

(a)

(b)

(c)

(d)

FIG. 138 (a).—Advanced gout, with large areas of decalcification at the ends of the metacarpals and phalanges. Note the soft tissue swelling of the fingers. (b) Gonorrhœal arthritis of wrist and carpal joints. Decalcification and loss of definition of the joint surfaces are well seen. Note the new periosteal bone on the shaft of the radius. (c) Osteoarthritis, especially between the interphalangeal joints, and at the base of the first metacarpal. (d) Typical Rheumatoid (atrophic) Arthritis showing decalcification of the bones: the outlines of the joints are as yet unaffected.

Symptoms.—The spine becomes quite rigid usually from below upwards; the name "poker back" is aptly applied. This is due to a synostosis of the vertebræ and ossification of the intervertebral ligaments. A similar change at the hips and shoulders may produce fixation, partial or complete, of these joints also. There is marked kyphosis of the upper part of the spine. The chest is flattened, and the breathing becomes entirely abdominal, due to fixation of the costovertebral joints. Nipping of the nerves at their exit between the vertebræ may lead to referred pains around the chest or abdomen, areas of impaired sensation, paræsthesiæ, and local atrophy of muscle. The X-ray appearances are characteristic and the erythrocyte sedimentation rate is high at a very early stage.

Etiology.—It occurs chiefly in males between the ages of twenty and thirty-five, but has been described in children. Three children in one family have been attacked.

Treatment is symptomatic, and on the same lines as that for rheumatoid arthritis. Breathing exercises are necessary to keep the chest wall mobile. Pain is relieved especially with infra-red rays; wide-angled deep X-ray therapy helps certain cases (Gilbert Scott).

§ 590. VI. **Gonorrhœal Arthritis** is a *synovitis* following a gonorrhœal discharge and resembling chronic rheumatism in some respects, chronic pyæmia in others. The acute form is described in § 583. III.

Symptoms. Often only one joint is affected, the disease coming on insidiously during the gleet (often about the fourth or fifth week). The knees and wrists are especially liable to be involved. It is particularly apt to supervene in those cases of gonorrhœa in which the prostatic portion of the urethra is affected. When the joint becomes involved, the gleet sometimes disappears, a circumstance which may give rise to an error in diagnosis. In women a cervical or urethral discharge can usually be traced. Occasionally there is high fever; usually it is low, of the intermittent type. The general health is always affected; the patient may be anæmic and emaciated. The onset is usually sudden, one large joint being attacked. In some cases many and any joints may be affected. In the absence of chemo-therapy the joints become swollen, stiff, tender, and gradually ankylosed. The fibrous tissues also are often affected, especially the plantar fascia, producing flat feet. Periostitis may also occur, especially of the os calcis. Pain in these positions or in the tendo Achillis is an important diagnostic feature of the disease.

The *Diagnosis* may closely simulate that of acute or chronic infective arthritis: and is suggested when joints such as the sterno-clavicular, temporo-mandibular or hip joints are involved in young adults. (This is unusual in rheumatoid or infective arthritis.) Coincident iritis always suggests a gonococcal cause. The complement fixation test shows that at some time the patient has had gonorrhœa: when the test is negative in the blood, the joint fluid may give a positive result. The history of infection, the absence of response to salicylates and the good effect of penicillin will confirm (Fig. 138*b*).

The *Prognosis* is more hopeful in younger people, in attacks of recent date and especially with penicillin treatment. There may be pleurisy or iritis, and in rare cases the heart and the meninges have become affected, with fatal results.

Etiology.—Both sexes may be affected. Fluid aspirated from the joint rarely may show gonococci. Special exposure to chill during gonorrhœa may determine the onset of the arthritis.

Treatment.—The first indication is to put the patient to bed. The gonococcus is susceptible to penicillin (50,000 units four-hourly for 7 days), and this should be combined with a sulphonamide (Tables XXVIII and XXX). For the joints, use Scott's dressing, or short-wave diathermy (the gonococcus cannot exist at the temperature to which the joint is raised). Some prefer T.A.B. vaccine intravenously. The patient should avoid any possibility of a fresh attack of gonorrhœa.

§ 591. **Tuberculosis, Syphilis, Hysteria, Tabes Dorsalis,** and other **nervous disorders** also affect the joints.

VII. **Tuberculous Joint Disease.**—Tuberculosis affects chiefly the synovial membrane, but it may commence in the articular ends of the bones. This is *par excellence* the monoarticular joint disease of children.

Symptoms.—The onset is insidious, though not infrequently the symptoms date from an injury. The favourite situations are hip and knee-joint, though any joint may be affected. The child may complain of slight pain, which gives rise to limping, for weeks or months before anything else is apparent. Generally the disease is in the knee, but sometimes it is in the hip, although the pain may still be referred to the knee through the obturator nerve. The affected joint swells; it is pale, and has a pulpy or doughy feel beneath the finger, and fluctuation may be felt. If untreated, an abscess develops. The constitutional symptoms consist of an intermitting pyrexia and general debility which are present even from the very beginning.

The *Cause* is usually infection with bovine tuberculosis. In a chronic synovitis in adults tubercle bacilli can often be identified in aspirated joint fluid. The symptoms may date from or be first noted after an injury. The disease nearly always attacks children, though a more destructive form may occur in advanced life. It may last for many years, and the prospect of recovery depends upon when treatment begins. If neglected, extensive destruction of the joint follows, and frequently tuberculous disease in other organs. *Treatment* lies mainly with the surgeon, but much can be done in the early stages by rest, artificial and real sunlight, fresh air, and cod-liver oil. Rollier in Switzerland has excellent results with rest and sunshine.

VIII. **Syphilitic Joint Disease.**—In the secondary stage of syphilis there may be (i.) a subacute arthritis with redness and pain, or (ii.) an indolent hydrarthrosis, with little pain. In the tertiary stage of syphilis the differential features are: (1) One or several joints may be affected. The synovial membrane may be attacked, leading to a doughy swelling; or the ligaments or cartilage. (2) The joint manifests no signs of acute inflammation, but there is occasionally some effusion. (3) The pain is very moderate during the day, but subject to nocturnal exacerbations. (4) Other evidences of syphilis are generally present. (5) The condition is very chronic, and only partially responds to iodides. It may occur in children (§ 552).

A Pseudo-Paralysis of Syphilitic Origin occurs in infants, due to epiphysitis and later even a separation of the cartilage from the diaphysis, and may be mistaken for joint disease or for infantile paralysis. The affected part is acutely tender. The condition may be readily recognised by X-ray examination—indeed similar changes may be seen in the epiphyses with periostitis even in the absence of symptoms.

IX. **Hysterical Joint Disorder** is often a muscular stiffening and immobility. It usually affects the hip or the knee, and often dates from some trifling injury. The joint is fixed, tender (often more tender to light touches than to deep pressure), sometimes swollen, and the local temperature may be raised. Sometimes there are no physical signs referable to the joint at all. The loss of function may

be entirely due to muscular rigidity and in the case of the hip-joint the condition may very precisely mimic tuberculous disease of this joint. This *Diagnosis*, which is often difficult, rests mainly on (1) the absence of evidence of serious disease in the affected joint when examined under anæsthesia; (2) the disproportionate loss of function; (3) absence of X-ray changes.

The *Treatment* should be directed to the hysteria. See § 888.

X. **Neuropathic Arthritis** (Synonyms: Neuro-Arthropathy, Tabetic Arthropathy).—Two diseases of the spinal cord are sometimes, though comparatively rarely, attended with chronic mischief in the joints—viz., *Tabes Dorsalis* and *Syringomyelia.* In both arthritis may occur in an early stage of the disease, when nervous symptoms are few, and extensive disintegration of the joint may take place, without pain, heat, or redness, and without giving rise to much inconvenience. X-ray shows gross destruction of the bone, and even dislocation. In *tabes dorsalis* the associated joint lesion is known as tabetic arthropathy, or **Charcot's joint disease** (see § 817), which especially occurs in the lower limbs. Occupation chiefly determines which joints are involved; thus in a blacksmith the arms, and in a soldier the legs are affected. This lesion may occur without the patient suffering any pain; it may go on to extensive disorganisation with increased mobility and new bony formations before the patient seeks advice. In all such cases the pupils and ankle and knee-jerks should be examined.

Syringomyelia is characterised by muscular atrophy and dissociated anæsthesia. The arthritis is limited almost entirely to the upper limbs (§ 818).

In *Raynaud's Disease* (§ 579) a subacute or chronic synovitis sometimes occurs, associated with an atrophic condition of the bones.

Intermittent Hydrarthrosis possibly comes under this heading. The joints swell at periodic intervals which the patient can foretell almost to a day. Nothing accurate is known of its etiology. There is no fever. It has been ascribed to allergy; some cases have been associated with angioneurotic œdema.

GROUP III. MUSCULAR DISEASES

We are here concerned with lesions situated in the muscular substance as evidenced by pain in the muscle (myalgia) and tenderness, accompanied, perhaps, by some swelling. The causes of pain in the limbs were discussed in § 567. The causes of muscular weakness will be dealt with in the chapter on nervous diseases.

I. Muscular rheumatism. § 592.
II. Tumours. § 592.
III. Trichinosis. § 593.
IV. Idiopathic myositis. § 594.

§ 592. I. **Muscular Rheumatism** (fibrositis) is certainly the most frequent cause of muscular pain and tenderness in this country. It is very incapacitating, and prone to recur.

Symptoms.—(1) The pain usually comes on so suddenly that in the case of lumbago, it is often mistaken for a sprain or rupture of muscular fibres. Pain is accompanied by a complaint of stiffness: these are relieved by rest, and much more marked when movements are recommenced, *e.g.*, on first getting out of bed. (2) Acutely tender areas occur in the affected muscles, due to fibrous nodules which can often be felt in the muscles, tendons and fascia; they swell and become more painful in cold, damp weather. (3) At the onset there is usually a furred tongue and disordered digestion, with constipation. There may be slight pyrexia.

The commonest *variety* of muscular rheumatism is **lumbago,** where the pain is situated in the muscles and fasciæ of the small of the back: it may cause referred pain in the distribution of the sciatic nerve. It is usually of very sudden onset, often when in the act of stooping. Rheumatic torticollis affects the sterno-mastoid, and occurs chiefly in children Intercostal rheumatism (pleurodynia) is a similar affection of the inter costal muscles. Intractable headache may be caused by occipital fibrositis. Lumbago has to be *diagnosed* from other causes of lumbar pain (§§ 370 and 457). In aneurysm of the thoracic aorta the pain is more continuous, not so easily relieved by muscular rest. In myelitis and meningitis there are other symptoms referable to the nerve trunks, sensory, or motor. **Panniculitis** is a variety seen in middle-aged and elderly subjects, especially stout women, particularly across the back of the shoulders and round the knees, where there is diffuse involvement of the subcutaneous tissues. Tender areas are easily made out on pressure and the skin dimples on being picked up. **Epidemic fibrositis** has recently been recorded.

Etiology.—Muscular rheumatism occurs much more frequently in middle-aged and elderly adults. It is predisposed to by fatigue (with which is often associated a bad posture), a muscular strain, a "chill" (especially profuse perspiration and then sitting in a draught), and by errors of diet: sugar, rich foods, and sweet heavy wines are most harmful. Some cases are due to a subacute attack of gout (§ 583). Focal infection (*e.g.*, apical infection of one or more teeth) can cause generalised fibrositis, often with a low grade temperature and other constitutional effects. In some, intestinal toxæmia appears to be causal. The significance of the rheumatic nodule is not yet clear: two theories are (*a*) that it is due to local muscle spasm (Elliott), and (*b*) that there is local œdema of fatty areas in relation to the insertion of the paravertebral muscles (Copeman).

Treatment.—For an *acute attack* rest in bed and warmth are essential. The diet must be simple: carbohydrates are limited because the sugar tolerance is low: alcohol is often harmful (§ 587). The bowels should be regulated but vigorous aperients avoided. Relief from pain is secured with analgesic drugs—aspirin, tab. codein co. or full doses of sodium salicylate. Locally counter-irritants and cataplasma kaolini should be used. Sometimes an attack may be aborted by a Turkish bath. For more *chronic cases*, radiant heat, infra-red rays and diathermy allay pain: nodules may be injected with procaine hydrochlor. (0·5 per cent.) and then massaged: in other cases intensive doses of galvanism or diathermy with a small localising electrode applied to the tender point do good Warm underclothing should be worn. In cases of recurring lumbago, B. acidophilus taken over a long period has proved useful. Especially with *generalised fibrositis*, search should be made for an infecting focus in the teeth, tonsils, nasal sinuses or in the bowel. For the treatment of subacute gout, see § 583.

II. **Tumours** in the substance of the muscles may give rise to pain and tenderness usually associated with swelling. The pain and tenderness are at first strictly localised to the seat of the disease, and there is a thickening or tumour discoverable on careful palpation. In some cases—*e.g.*, syphilitic and malignant growths—the lymphatic glands in the neighbourhood are enlarged. The chief tumours affecting muscles are (*a*) innocent—syphilitic gumma; abscess, which may arise from a gumma, or be of inflammatory origin, especially after typhoid and influenza; innocent neoplasms such as fibroma, lipoma, angioma, hydatid cysts and hæmatoma. (*b*) Malignant growths, sarcoma, and carcinoma (by extension). First determine whether the swelling is inflammatory or non-inflammatory, malignant (and rapidly growing) or non-malignant, by an investigation of the swelling, the glands, the history, and the concurrent symptoms. The diagnosis and treatment is mainly surgical.

§ **593.** III. **Trichiniasis** is a rare disease, due to the presence of a nematode worm (the trichina spiralis) in the intestinal canal, and the dissemination of the embryos in the blood and the muscular system. The female *adult* or intestinal worm measures about ⅛ inch, the male slightly less. In fæcal examinations for the parasite it should be remembered that the characteristic feature is the "cell body" at the anterior part of the intestine of the parasite. The *larvæ* (Fig. 139) or muscle trichinæ are found in infected muscle, where they are visible by the aid of a ½ or 1 inch lens. Each consists of an ovoid capsule (translucent, or infiltrated with lime salts, according to the length of time it has existed) containing two or more embryos coiled up within it. The *embryos* are 0·6 to 1 mm. long, with pointed head and rounded tail. The presence of these larvæ gives to pork or other infected meat a characteristic "measly" appearance visible to the naked eye. Trichina is chiefly conveyed to man by "measly" pork, insufficiently cooked; the capsules are digested, and the embryos set free in the intestinal canal. During the ensuing week the embryos attain sexual maturity; each female can produce several hundred embryos. After fecundation the female worm penetrates the walls of the intestine; hundreds of embryos reach the lymph spaces, blood and muscles, where after 2–3 weeks they become encysted. They have been found alive and capable of developing 10 years after their entrance.

Symptoms.—The disease runs a course of several weeks, and shows three stages. The *first* stage lasts 7–10 days, with symptoms of gastro-intestinal disturbance, abdominal pain, diarrhœa and vomiting, urticaria and other eruptions. The *second* stage, lasting 2–3 weeks, coincides with the migration of the embryos. This gives rise to headache, œdema of the eyelids, stiffness and aching of the limbs, obstinate constipation, and to mental symptoms with lethargy and confusion, meningitic or encephalitic symptoms, and melancholia: monoplegia, cerebellar signs, oculogyric crises and extensor plantar responses are met. The wandering of the embyros in the muscles produces shortening and rigidity, especially of the biceps. In severe cases, movement of the affected muscles—*e.g.*, turning the eyeball, chewing, swallowing, etc.—aggravates the pain. Remitting or intermitting pyrexia is present with profuse perspiration and insomnia. Extreme dyspnœa arises if the diaphragm is implicated. There may be general œdema and later on emaciation. In slight cases the muscular and other symptoms are insignificant and overlooked. In the *third* stage the acute symptoms gradually subside, great muscular weakness ensues, and recovery is slow. Various complications (see below) may interrupt this stage.

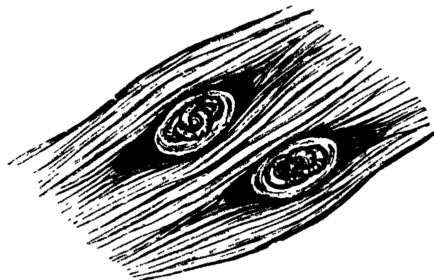


FIG. 139.—Larvæ of the TRICHINA SPIRALIS encysted in muscle.

Diagnosis.—In slighter cases the distinction from muscular rheumatism or other diseases in this group may be difficult,

though the headache, puffiness of the eyelids, the widespread muscular tenderness, the history of gastro-intestinal symytoms, and the epidemic occurrence in a whole family should aid us. The stools after a large dose of calomel may be searched for the adult worm. Some cases are mistaken for typhoid fever, and *vice versa*. In trichiniasis there is marked leucocytosis—reaching 30,000 per cu. mm. or more—due mainly to an enormous increase in the eosinophil cells which may amount to 50 per cent. of all the leucocytes. The embryos may be found in the blood in the third week, and later, a biopsy of the affected deltoid may reveal encysting larvæ. In after years calcified cysts may be seen by X-ray examination.

Prognosis.—The disease often ends fatally between the third and sixth week; the mortality varying from 2 to 30 per cent. The intensity and duration of the symptoms measure the prospect of recovery. Death may occur from (i.) diarrhœa; (ii.) asphyxia (from involvement of the respiratory muscles); (iii.) exhaustion; or (iv.) hæmoptysis or pneumonia. Health may not be restored for several months.

Etiology.—The disease is due to the ingestion of "measly pork" or other meat, and occurs in an epidemic form in families and towns. Rats act as reservoir hosts. It is much more frequent in Northern Germany, where underdone pork or ham is a popular food, than in England and France. Thorough cooking destroys the parasite, but in large joints the temperature may not destroy the parasite in the interior All meat, particularly sausages and pork, should be thoroughly cooked.

Treatment.—If the patient is seen within two or three hours after eating infected meat an emetic should be given. If the disease is discovered within twenty-four or thirty hours the gastro-intestinal tract must be thoroughly cleared out. Glycerin in large doses has been recommended in the first stage as its hygroscopic properties destroy the nematode. Filix mas, santonin, thymol, and turpentine are also recommended. Salvarsan and antimony injections have cured some cases. If, however, the second stage is reached, and the embryos are migrating, the treatment must be symptomatic, because nothing will destroy them. Fouadin is giving good results. For the pain and tenderness, opium and other anodynes may be required.

§ **594.** IV. **Myositis,** or inflammation and swelling of the voluntary muscles, is a rare condition; few cases have been recorded. Three forms are recognised: (*a*) *localised,* in which pain, tenderness, swelling, and impaired movement are localised to one muscle or group of muscles; (*b*) *acute generalised,* in which these symptoms are accompanied by œdema and redness of the skin; (*c*) *progressive generalised,* in which the disease runs a prolonged course, spreading from muscle to muscle, causing painful swellings, followed by wasting of the muscles and sometimes involving the skin (*dermatomyositis*). In *myositis ossificans* the process goes on to the formation of bone; it follows trauma, or arises in association with nerve disease. In the former, recovery may occur.

The *diagnosis* may be difficult, especially from trichinosis; see § 593. *Dermatomyositis* has been mistaken for rheumatism, and various skin diseases (lupus erythematosus, erythema, and urticaria); later, the weakness of the muscles and progressive ill-health make the diagnosis clear.

Etiology.—Syphilis, scurvy and typhoid fever have been in operation in some cases of myositis. The acute *localised* form may follow injury or extension from surrounding inflammation, as in pyæmia. *Dermatomyositis* is due to an unknown infection. It is sometimes associated with scleroderma, and due to the same pathological process.

Treatment.—Rest and heat in the form of light baths, infra-red rays and diathermy relieve. In the generalised forms all infective causes should be sought and removed. Glycin has aided some cases.

GROUP IV. BONE DISEASES

The acute diseases of bone are of especial interest to the surgeon: the chronic diseases are, in the main, of medical interest. Nevertheless, all bone diseases are frequently seen by the physician, especially in their early stages. Pain and deep-seated tenderness are often their chief and sometimes their only symptoms. Pyrexia and constitutional derangement may be present. Deep-seated swelling and deformity may appear later and, if the bone is superficial, œdema and redness of the skin.

Acute Bone Diseases.

I. Acute osteomyelitis.
II. Acute localised periostitis.
III. Acute epiphysitis.

Chronic Bone Diseases and Deformities.

I. Rickets.
II. Chronic periostitis, osteitis, caries, and necrosis.
III. Tumours of bone.
IV. Acromegaly.
V. Achondroplasia.
VI. Pulmonary osteo-arthropathy.
VII. Osteitis deformans.
VIII. Osteomalacia.
IX. Leontiasis ossea.
X. Multiple myeloma.
XI. Fragilitas ossium.
XII. Cleido-cranio-dysostosis.
XIII. Marble bones.
XIV. Perthes' disease.
XV. Osteitis fibrosa cystica.

§ **595. Acute Infective Osteomyelitis** (Syn.: Acute Necrosis) is an acute infection affecting one or more of the bones, accompanied by severe constitutional disturbances —on which account the case comes under the notice of a physician. This is the only really acute bone disease, though acute symptoms closely resembling those of osteomyelitis may arise in association with a LOCALISED PERIOSTITIS such as results (especially in children) from an injury.

The *Symptoms* are (1) pain of a very severe character coming on suddenly, and attended by extreme tenderness, starting usually at the articular end of the bone—very often the tibia—attended in the course of a day or two by swelling of the limb, at first pale, and later often red, as the inflammation makes its way towards the surface. (2) The constitutional symptoms come on suddenly, and are very marked. The temperature is high, and there may be rigors and great prostration: a positive blood culture occurs in 50 per cent. of cases. The *Diagnosis* from acute rheumatism, which it may at first resemble, because of the pain starting near a joint, is made by the fact that in rheumatism the pain and swelling are confined to the joint, by the early involvement of other joints and by signs of cardiac complications. Also in osteomyelitis the leucocyte count is usually higher. Acute epiphysitis is mentioned below. The *Prognosis* has been revolutionised by penicillin which, if given within the first 3–4 days usually prevents abscess and necrosis of the bone. The most frequent complications used to be pyæmia and the extension of the inflammation to a joint. *Etiology.*—Acute osteomyelitis is more frequent in children under the age of puberty. It is usually due to the staphylococcus aureus. *Treatment.*—As soon as it is suspected, intensive penicillin treatment should be started, and continued for at least three weeks (Table XXX). Some use also a continuous penicillin drip into the medulla of the affected bone. The limb should be completely immobilised. Surgical measures will only be necessary later if a local abscess results.

Acute Localised Periostitis may arise from traumatism, and if not infected it soon subsides. If infected either from a wound or from the blood, suppuration and necrosis take place, and the condition becomes chronic (§ 597).

Epiphysitis is inflammation beginning in the growing line, which in early life separates the epiphysis from the shaft of the long bone. The *acute* form is met with in very early infancy and is usually due to a staphylococcal infection. It may resemble acute osteomyelitis, but the profound constitutional disturbance is lacking. It is distinguished from acute rheumatism by the age of the patient, and by the excellent response of most cases to adequate doses of penicillin. Other varieties are due to infantile scurvy (§ 545) and congenital syphilis (§ 552). In the *chronic* form the process is much slower, and is of interest chiefly in relation to the diagnosis of rickets, from which it differs in being localised to one joint. As regards causation the acute form is generally due to an injury and sepsis, the chronic form to syphilis or tubercle.

§ 598. I. Rickets (Synonym: Rachitis) is a constitutional disorder of childhood attended with epiphyseal enlargements and other deformities of the skeleton. It was described by Glisson in 1675.

The *Symptoms* for which we are consulted, coming on between the sixth and twelfth month, are delayed dentition and walking, or the child cannot sit up; gastro-intestinal disorders; bronchitis; sweating about the head; or a generalised tenderness and restlessness. In the limbs the disease is typically shown by the *enlarged epiphyses*, affecting most, if not all, of the long bones. The rib-ends are the first to show the enlargements at their junctions with the costal cartilages and thus produce an appearance of "beading"—the "rickety rosary." The long bones often curve most commonly the tibiæ and fibulæ: the convexity is outwards: and greenstick fractures may be produced by slight injuries. The spine has a general backward curvature when the child sits up, due to muscular weakness; scoliosis may ensue later. The head is square-shaped, both the frontal and parietal eminences are prominent: the anterior fontanelle may remain open after the second year (normally it closes at the fifteenth to the sixteenth month): there may be craniotabes (thinning of the skull bones) especially of the occipital region. The body may be emaciated or plump and flabby. The chest is deformed, due to sinking in at the costo-chondral junctions, so that the sternum and cartilages stand out prominently in front, and are united to the ribs along a deep lateral groove. Another groove (Harrison's sulcus) runs transversely across the chest, just above the lower costal margin. The liver and spleen are both enlarged in advanced cases; the costal margin is everted, and the belly is prominent. The muscles are hypotonic, permitting hypermobility. There is recurrent gastro-enteritis, and bronchitis is frequent. Instability of the nervous system is evidenced by convulsions, tetany, or laryngismus stridulus.

Severe forms of rickets occur in association with cœliac disease and chronic interstitial nephritis in children—*Cœliac* and *Renal Rickets*. The bone changes closely resemble those of severe cases of ordinary rickets, and improve with treatment of the causative disease. Cœliac rickets arises from deficient absorption of calcium and vitamin D in cœliac disease (§ 307. IV). Renal rickets usually occurs about the age of seven, but may date from birth, or occur at 16–18 years. The chief symptoms are dwarfism, polyuria, polydipsia, and often extreme bony deformities. The urine is excessive in amount, has low specific gravity and usually a little albumen (p. 488). The blood shows retention of nitrogenous bodies. The cause of the bony deformities is the inability of the kidney to excrete phosphate, leading to a relative insufficiency of calcium (Parsons).

Diagnosis.—The disease may have to be diagnosed from *hereditary syphilis,* in which there may be enlargement of the epiphyses, but this occurs usually only in one bone, and is accompanied by other undoubted signs of syphilis. *Infantile paralysis* soon exhibits muscular wasting. *Achondroplasia* (§ 598. V) is a rare condition. In *infantile scurvy* (§ 545) the swellings affect the shaft rather than the epiphysis, and are painful. The diagnosis of rickets, *hereditary syphilis,* and *hydrocephalus* is given in the form of a table (Table XLII). It is chiefly in regard to the form of the head that the diagnosis between hydrocephalus and rickets presents any difficulty. The X-ray picture in rickets is characteristic, showing irregular and defective calcification in the epiphyses.

Prognosis.—The disease when taken in hand before osseous changes are marked is readily amenable to treatment. If untreated it leads to deformity. If death occurs it is due to some of the common complications, notably pneumonia, bronchitis, gastro-intestinal disorder, wasting or convulsions. Spinal, pelvic, and other deformities may result, and the growth is stunted. Genu valgum (knock-knee), genu varum (bow-leg), and flat-foot often occur. The prognosis of renal rickets is uniformly bad; almost all cases die in the second decade.

TABLE XLII.—DIFFERENTIAL DIAGNOSIS.

	Rickets.	*Hereditary Syphilis.*	*Hydrocephalus.*
I. History.	Gastro-intestinal irritation, sweating about head. Improper feeding.	Snuffles and rash. Stillbirths in mother.	Congenital, or acquired after meningeal inflammation, or due to tumour pressing on veins.
II. Age of patient.	Begins to show itself after six months and before the second year.	Symptoms first appear third week to the third month.	Congenital or acquired.
III. Shape of head.	Often compressed antero-posteriorly. Frontal eminences marked.	Irregular prominence on frontal and parietal bones. Skull termed natiform. Depressed bridge of nose.	Bulges in all directions. General tendency to assume a globular form.
IV. Fontanelles.	Close late.	Appear to be depressed in the hollow between the four prominences.	Bulging, separation of the bones at the sutures.
V. Other peculiarities.	Epiphyseal enlargements, delayed dentition, etc.	Pegged and notched permanent teeth. Scars about mouth, palate, etc.	Stunted growth, sometimes mental deficiency.

Etiology.—Rickets rarely appears earlier than six months or later than the second year. Both sexes are equally affected. The disease is more frequent in cities, and during the dark months of the year. Sunlight is as important as diet for the prevention and cure of rickets. Rickets is a deficiency disease, due particularly to deficiency of vitamin D. There is defective absorption and retention of calcium and phosphorus, for which vitamin D is essential. The blood is usually deficient in inorganic phosphorus; in other cases there is calcium deficiency, leading to deficient

deposition of calcium phosphate in the bones. In some cases gastro-intestinal conditions hinder the absorption of calcium, phosphorus or vitamin D. (See cœliac disease, § 307. IV.) Vitamin D_3 is formed by the action of ultra-violet radiation and of natural sunlight on a precursor in the skin and on foods containing it (§ 928). Vitamin D_2 (calciferol) is synthetic. Sunlight and various forms of lamps which are rich in ultra-violet rays can prevent and also cure rickets.

Treatment.—Correct the dietetic deficiency. Reduce carbohydrates and add raw meat juice, fresh milk, butter and cream, well-chewed fresh fruit and green vegetables. Cod-liver oil contains the essential vitamin, and has for a generation been a recognised cure for rickets. Halibut liver oil is a potent source of vitamins A and D. Calciferol or irradiated ergosterol can replace cod-liver oil; in large doses they are dangerous. Exposure of the body to ultra-violet rays, such as are found in natural sunlight or artificial lamps, is curative in ordinary and in cœliac rickets, but aggravates renal rickets (Parsons). Only in severe cases is the child not allowed to walk, lest the bones yield and permanent curvatures arise: then splints may be necessary. Treatment of *renal rickets* is that of uræmia.

§ 597. II. Under **Chronic Osteitis** and **Periostitis** are included a number of tuberculous, syphilitic, and other conditions leading to caries, necrosis, and other anatomical changes in the bone. Osteitis and periostitis are dealt with together, for although the disease may start in the bone or the periosteum it soon spreads to the other.

The *Symptoms* of osteitis and periostitis may begin with acute pain, redness, and swelling; but more frequently they start insidiously with hardening, thickening, or enlargement of the bone. These symptoms may be followed by softening (caries) or death of a portion of the bone (necrosis) with signs of abscess formation.

Causes and their differentiation: (1) *Traumatism* alone, without sepsis or toxæmia of some kind, is a rare cause of chronic periostitis or osteitis. Traumatism is recognised by its history, and by the fact that only one bone is affected. (2) The favourite seat of *tubercle* is the epiphysis, where it induces a chronic epiphysitis, especially in the neighbourhood of the hip or knee. Sometimes it gives rise to osteitis, and when this occurs in the fingers it results in a characteristic thickening of the phalanges known as tuberculous dactylitis. In any position it may go on to caries or necrosis. Tuberculous affection of the bones is recognised by (i.) the youthful age of the patient; (ii.) a tuberculous history; (iii.) the characteristic intermitting pyrexia; (iv.) signs of tubercle in the lungs and elsewhere; (v.) the chronicity of the process; and (vi.) the frequent limitation to one bone. (3) *Syphilitic* affections of the bones are very common both in the acquired and the hereditary disease. (*a*) *Acquired syphilis* may take the form of a chronic diffuse or localised periostitis (nodes), or, on the other hand, a diffuse or a gummatous (localised) osteitis. It is recognised by (i.) the characteristic flying pains in the limbs; (ii.) the nocturnal pains in the bones, which are such a frequent manifestation of syphilis; and (iii.) other evidence of syphilis. (*b*) *Hereditary syphilis* may give rise in childhood and early life to the same lesions as the acquired disease. In infancy (in addition to the foregoing) chronic suppurative osteochondritis (chronic epiphysitis) may be mistaken for rickets. One or several bones may be affected, but it never presents the same symmetry as rickets. The deformities resulting from hereditary lesions (§ 552) and the physiognomy are characteristic—the bosses on the frontal and parietal bones (Parrot's nodes), the depressed bridge of the nose, scars about the angle of the mouth, Hutchinson's teeth,

and perhaps keratitis (Tables XXXIX and XL). (4) *Rheumatism* and *gout* may give rise to chronic periosteal thickening, or periosteal nodes.

For the adequate *Treatment* of most of these different conditions, rest and surgical aid are necessary. The treatment of tuberculosis, syphilis, rheumatism, and gout have already been dealt with.

III. **Tumours of Bones** may commence with pain, enderness, and swelling like chronic periostitis. The chief *innocent* tumours are EXOSTOSES, which may occur on almost any bone, and ENCHONDROMATA, which are commonest on the metacarpals and phalanges. Both are usually multiple. The *malignant* tumours are either SARCOMA (especially myeloid sarcoma) or secondary deposits of CARCINOMA. In both, the swelling of the bone is more rapid and reaches a greater degree than in any of the other causes of swelling above mentioned, and as a rule they are limited, at any rate at first, to a single bone. Spontaneous fractures may occur.

§ **598.** Certain rare forms of chronic bone disease must be mentioned.

IV. **Acromegaly** is a rare disease with overgrowth in the skeletal tissues. The patients generally apply for treatment for some other malady: headache is often a marked feature and sometimes they complain of obscure pains in the limbs. The mentality is unaffected, but the aspect is very characteristic. The bones and other tissues of the hands and feet become markedly elongated and hypertrophied, though the growth is so gradual as to escape the patient's notice. In the head the cranium is enlarged but the bones of the face much more so—it has been called "egg-shaped," the lower jaw representing the large end of the egg. The lower jaw especially is enlarged, and may project beyond the upper jaw (see Fig. 6). The nasal bones are also enlarged, whilst the thickening of the soft parts causes hypertrophy of the ears, eyelids, nostrils, and tongue. Later in the disease there may be a similar enlargement of the bones of the limbs and the thorax, and kyphosis of the spine. When due to a tumour, the headache is associated, sooner or later, with bitemporal hemianopia, and /or with gradual optic atrophy (§ 837). The glandular defect leads to other changes such as amenorrhœa, sexual impotence, glycosuria in the early stage and high sugar tolerance later on.

Diagnosis.—Myxœdema resembles acromegaly, but it is known by the dry skin, the sluggish mentality, and the absence of all bony enlargement. X-ray may show an enlarged sella turcica. Sugar tolerance tests and the B.M.R. aid diagnosis. For pulmonary osteo-arthropathy see VI below.

Etiology.—Acromegaly occurs rather more frequently in women, generally beginning about the twenty-fifth year. Changes in the eosinophil cells of the anterior pituitary body, either hypertrophy or tumour, have been found in all the fatal cases. Gigantism and acromegaly (§ 17) are both due to disordered function of the pituitary gland; the sella turcica is often enlarged. In some cases there have also been changes in the thyroid gland, and signs of an enlarged thymus.

Treatment.—Acromegaly runs a very prolonged course of many years. Treat symptoms. Extract of thyroid is useful in the early stage; later, operation or X-rays applied to the pituitary gland may be necessary to relieve headache. Injections of the anterior lobe of the pituitary aid some.

V. **Achondroplasia** (Synonyms: Fœtal Rickets, Chondrodystrophia Fœtalis) is a rare condition in infancy, leading to dwarfism and generalised deformity, which used to be confused with cretinism, and with the deformity resulting from rickets. Fig. 140 represents a case. There is a generalised symmetrical shortening of the diaphyses (producing characteristic shortening of the limbs) with considerable thickening of the epiphyses (producing enlargement of the articulations), due to hyperplasia of the cartilaginous ends of the bones. Consequently the stature is stunted, the fingers and toes taper and are abducted from one another, the cranium is large, the face small, and the bridge of the nose depressed. There is a characteristic waddling gait. The disease is congenital, and dates from birth. The mental deficiency, facial aspect, and the changes in the hair and skin characteristic of cretinism are absent, and cases

do not exhibit in the skull the constitutional symptoms or characteristic changes of rickets. It is ascribed to a premature union of the diaphysis and epiphysis, so that lengthening of the long bones is arrested.

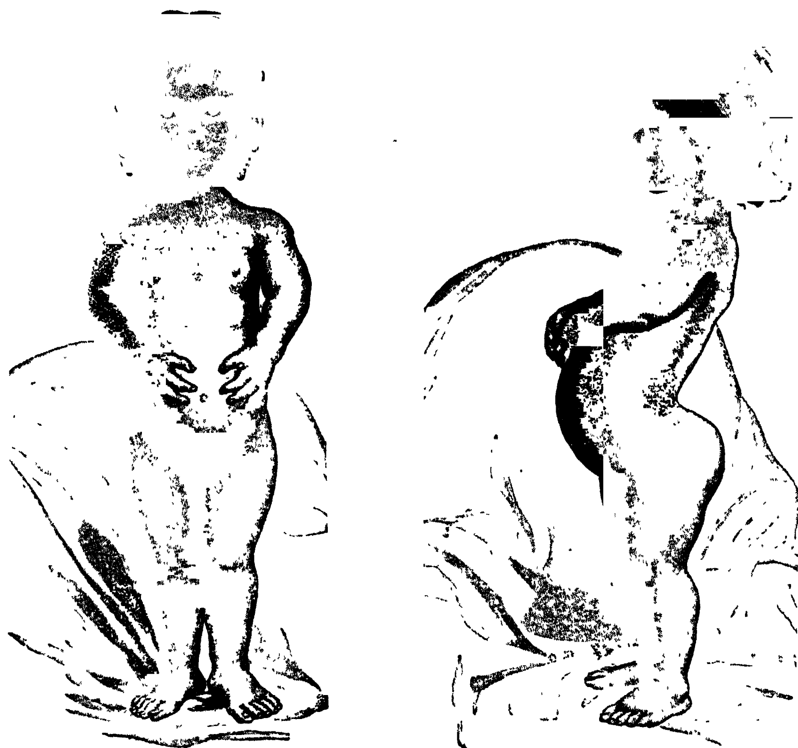


FIG. 140.—A case of ACHONDROPLASIA.

VI. **Pulmonary Osteo-arthropathy** is a chronic hyperplasia sometimes associated with chronic pulmonary disorders. There is enlargement of the hands and feet, and of the lower ends of the long bones of the legs and forearms, but the face and head are not enlarged. The nails are curved over the enlarged terminal phalanges, "filbert nails."

VII. **Osteitis Deformans** (Synonym : Paget's Disease) is a somewhat rare disease coming on after middle life, mostly in males, and consisting of a very chronic enlargement of the bones, both in diameter and in length. The histological change is a rarefying osteitis with enlargement of the Haversian spaces, producing a characteristic X-ray picture. It may affect only one bone (such as the tibia), or may be more generalised, and then tends to involve especially the cranium (not the face), spine, bones of the pelvis and legs, and clavicle. It may become manifest to the patient by the fact that he frequently has to change the size of his hat. Compression of the auditory nerve causes tinnitus and progressive deafness. Sometimes "rheumatic" pains in the bones occur. The head is projected forwards, associated with kyphosis in the dorso-cervical region, so that the attitude is characteristic. The base of the chest is expanded, the abdomen diamond-shaped, and crossed by a deep transverse sulcus,

the hips are widened, and the legs are bowed outward and forward. A high blood phosphatase is present at an early stage. There is a great tendency for osteosarcoma to develop, especially in the bones of the pelvis. Dr. Laughton Scott, having observed success by modifying the calcium phosphorus ratio in certain osteoporotic diseases in animals, reports favourably on the effects of a diet rich in calcium and poor in phosphorus. Deep X-ray relieves the pain.

VIII. **Osteomalacia** (Synonym : Mollities Ossium) is a progressive disorder of the bony system, due to gradual decalcification of the bones, which results in considerable deformities and contortions, owing mainly to muscular action. The X-ray appearances are characteristic. It is a disease of the poor, and is common in women in India between twenty-five and thirty-five years of age, mostly after pregnancy. The early symptoms consist of wandering pains in the limbs and trunk, worse at night, with weakness of the limbs. In the course of a few months there is *bending* of the bones ; spontaneous *fractures* and distortions are rare. The pelvic deformities cause difficult labour. The stature is diminished from the involvement of the spine. Death usually occurs from respiratory complications owing to the fracture of the ribs. Calciferol, calcium salts, and sunlight are curative.

IX. **Leontiasis Ossea** is the term given to a rare condition in which there are symmetrical hyperostoses of the facial bones and skull, which encroach upon the cranial cavity, and so may lead to death.

X. **Multiple Myeloma** (Syn., Kahler's disease, Myelopathic Albumosuria) is a disease in which " myeloma cells " in the bone marrow undergo a malignant type of growth and invade the bones, sometimes producing tumours : displacement of the bone marrow causes anæmia. It is more common in men than women, and is rare before thirty-five. *Symptoms.*—In the early stage the chief symptom is pain, especially in the dorso-lumbar spine, ribs and sternum : it is aggravated by sneezing, coughing and by exertion, and unlike malignant metastases, usually goes during sleep. The affected bones are sometimes extremely tender and may fracture without apparent cause : compression paraplegia may occur. The urine contains Bence-Jones' protein (§ 386) in half the cases. General weakness is common. A blood count shows anæmia, with a normal or low leucocyte count, although a leukæmoid reaction is occasionally met (§ 543) : the peripheral blood may show myeloma cells. The blood sedimentation rate is often very high, due to an absolute and relative increase in globulin : the serum calcium is normal or raised, but the alkaline phosphatase usually within normal limits. The *diagnosis* is confirmed by sternal puncture or preferably by sternal biopsy. *Prognosis :* the disease is usually fatal within 2 years, often from uræmia. *Treatment :* deep X-ray therapy seldom eases pain. Recently intravenous stilbamidine (150 mgm. per dose) 3–4 times a week to a total of 1·0–5·5 G., with a low protein diet, has relieved pain and produced symptomatic benefit.

XI. **Fragilitas ossium and osteogenesis imperfecta** are terms now used almost synonymously for a condition of abnormal bone fragility due to a defect in the bone osteoblasts. It is usually hereditary. There are two varieties—(*a*) ante-natal, in which multiple fractures are found at birth ; (*b*) in the early years of life fractures occur with slight injury or even from a sudden muscular pull. The fractures heal well and the general health may not be interfered with, but gross deformities and diminution of stature occur. Blue sclerotics are more common in this second variety.

XII. **Cleido-cranio-dysostosis** is a congenital defect. There is absence of bone at the outer third of the clavicle, with persistence of the acromio-clavicular ligament, and deficient ossification of the cranial bones.

XIII. **Marble bones** (Syn., infantile osteosclerosis, Albers-Schönberg's disease) occur in children and young adults, showing (i.) undue fragility of the bones associated with dense osteosclerosis throughout the body, which obliterates the marrow cavities ; (ii.) severe anæmia (often with nucleated red cells, myeloblasts, and myelocytes in a blood film : see § 544) ; and (iii.) enlargement of the spleen and liver, proptosis, and optic atrophy may appear.

XIV. **Perthes' disease** (pseudo-coxalgia) is a rare disease which causes fragmentation of the epiphysis of the head of the femur. It occurs in children under twelve, and is evidenced first by a limp and limited abduction; later there is shortening and muscular wasting. X-ray decides the diagnosis. There is usually spontaneous recovery in a few years.

XV. **Osteitis fibrosa cystica** (Syn.: Hyperparathyroidism, von Recklinghausen's disease of bone) is a rare disease of the parathyroid glands, associated with adenoma. The effects resemble those of prolonged administration of an active extract of parathyroid, such as parathormone (Collip). The bones become decalcified, with loss of calcium and phosphate, which are excreted in the urine and may form renal calculi. The bones become progressively rarefied, with cyst formation, which may form large tender tumours. At the same time spontaneous fractures and bending of the bones cause progressive loss of stature. Gastro-intestinal disturbances with attacks of headache and vomiting are common. The muscles become hypotonic. The *diagnosis* is confirmed by the blood findings, as the calcium is raised to perhaps 15–16 mgms. per cent., the phosphorus is below normal and the alkaline phosphatase high. After successful removal of the parathyroid tumour, and large doses of calcium and calciferol, the bones slowly recalcify, and the headaches and gastro-intestinal symptoms are lost, with a return of the blood to normal (and see § 408).

CHAPTER XVIII

THE SKIN

THE skin is not merely a covering, possessing glands and appendages, which protects the body and develops disease when irritated by internal and external causes. It plays a part in immunity, and can fix and form antibodies. It is a vital organ, with an internal as well as an external secretion. Sunshine, changes of temperature and movement of air have a profound influence upon the metabolism and hence upon the general health. The skin reacts to these natural agents through their effect upon the sympathetic nervous system, blood-vessels and endocrine glands; this is especially useful in connection with the regulation of heat. It has a respiratory function and can be penetrated by certain drugs, fats and hormones. Under the influence of ultra-violet light the endocrine function is stimulated, the ergosterol in the skin is activated and vitamin D is formed. Light clothing, adequate exposure to wind and sunlight, permit that ready response of the skin to changes of climate, humidity and temperature which is essential for the health of the circulation and the organs.

Simple observation of the skin gives information concerning the health. Many diseases which attack the organs produce changes on the skin. These reactions may record the condition of the stomach, liver and intestinal canal, sometimes of the kidneys and the endocrine glands; and of the arterial, venous and capillary circulation. Several external and internal irritants arouse different cutaneous reactions in different individuals, and even in the same persons at different times of life. Certain eruptions inform us of the existence of a deep-seated infection; of such are the trichophytides, the tuberculides and the streptococcides (Barber); the last show several forms of cutaneous reaction to streptococcal infection. Skin diseases used to be treated chiefly by external applications; to-day, we realise that their cause can often be traced only after a complete medical investigation.

PART A. SYMPTOMATOLOGY

The cardinal symptom of skin affections consists of an ERUPTION with or without SUBJECTIVE SYMPTOMS. The subjective symptoms are relatively less important, because the morbid process itself is visible. One subjective symptom attends many skin diseases—namely, PRURITUS.

§ **605. Pruritus** is the Latin word for itching. Itching may be mild and intermittent, coming on with changes of temperature, a bath, or after food or drink, or it may be continuous, with severe exacerbations, rendering sleep impossible and the patient suicidal. There are three groups of causes:

(*a*) Pruritus may be *secondary* to some visible skin disease, when the itching is localised to the neighbourhood of the eruption. Some eruptions are invariably attended by itching, such as urticaria, eczema and most acute conditions which progress rapidly. Other diseases are generally unattended by itching, such as syphilis, psoriasis, and most chronic conditions which evolve their course slowly.

(*b*) Various *local conditions* may produce more or less localised itching: (1) *Discharges* or secretions from nasal, aural, buccal, vaginal or anal orifices. Many of these cases develop later a localised dermatitis. (2) A *rough garment*, such as a flannel and certain dyed articles, may produce intolerable itching in delicate skins. (3) *Parasites*, such as *scabies* and *phtheiriasis*. The *flea*, the *harvest-bug*, *pediculus of head* or *body* and other parasites cause intense itching. *Thread-worms*, leaking *mucus* or liquid *paraffin*, and *hæmorrhoids* often cause perianal itching.

(*c*) With *idiopathic* or *internal* causes the itching is generalised. Among the causes may be mentioned certain articles of food (*e.g.*, shell-fish, eggs, milk, cheese), jaundice, digestive disorders, diabetes, leukæmia, Hodgkin's disease, a septic focus, kidney and liver disease, constipation, pregnancy, the menopause, and old age. In PRURITUS SENILIS the skin of the aged looks dry and atrophic, slightly scaly or glossy. Pruritus, with congestion of the nasal mucous membrane, also occurs with the allergic state; see § 609. In some cases itching is due to neurosis; itching may be complained of long after the cause has been removed. For *Treatment*, see § 620.

PART B. PHYSICAL EXAMINATION

The APPARATUS required for the local investigation of skin diseases is simple—a good lens, a microscope with accessories, and the means of histological examination. A pair of fine forceps is useful for removing scales, hair, or parasites. Stretch the skin firmly or use a flat glass slide to find if the lesions disappear on pressure (diascopy).

HISTOLOGICAL EXAMINATION (biopsy) frequently aids diagnosis; a small piece is removed without appreciable pain under local anæsthesia.

Sometimes one must carry out BLOOD tests (especially Wassermann), URINE examination, sugar tolerance and other BIOCHEMICAL and CULTURE tests. Scrapings soaked in 10 per cent. potassium hydroxide are required for the microscopic examination of FUNGI. PATCH TESTS are made for various organisms, drugs and external irritants. The suspected substance is strapped on the skin and left for 24 hours. Use a control dressing, lest the subject be sensitive to fixing plaster. If the patient is sensitive, on removal of the dressing, the skin is seen to be erythematous, swollen, or vesicular. A modified test for contact dermatitis due to external irritants is made with the allergen dissolved in collodion. Irritants can also be tested for by INTRADERMAL INJECTION or by SCRATCHING the skin with the suspected cause.

§ 606. The points to investigate in any given case of skin eruption are: I. The size and appearance of the prevailing elements; II. What it feels like, and whether it disappears under pressure; III. The distribution

and symmetry of the eruption; IV. Subjective symptoms; V. The duration and evolution of the eruption; and VI. Its etiology.

I. The **Character and Size of the Prevailing Elements.**—The spots are *never all quite alike*, being modified by the age of each spot, the locality affected, and the conditions to which it has been subjected (*e.g.*, scratching, pressure, or local applications). It is therefore most important to *examine every part* of the eruption. Patients may object to undress and the physician may grudge the time, but these considerations should never be allowed to weigh.

§ **607.** The principal **elementary lesions** which appear on the skin consist of three varieties of PRIMARY lesions, and three which arise secondarily to these.

1. A *macule* (or macula) is a small area of congestion not elevated above the surface of the skin; *roseola* is a generalised eruption of macules; *erythema* is a larger area of congestion with fading edges. A *wheal* is an area of congestion accompanied by slight exudation beneath the skin; it is due to transient local œdema. A generalised eruption of wheals is called *urticaria* or "nettle-rash," because it resembles nettle stings. When large, the wheal is white in the centre and red around.

2. A *papule* (or pimple) is a small solid elevation of skin, conical, round-topped, or flat and easily palpable. A *lenticular* papule is a large flat-topped papule. A *nodule* is larger than a papule, but not as large as a *tumour.*

3. A *vesicle* is a collection of serous fluid in or beneath the skin. A *bulla* is a large vesicle. A *pustule* is a collection of purulent fluid beneath the cuticle.

The SECONDARY lesions are:

1. A *scale* or squame is the exfoliation of cuticle which occurs after congestion or inflammation of the skin, or it may be the product of pathological processes special to the skin, such as cornification, or *hyperkeratosis,* in which the horny layer is thickened. In a sense a scale may be a primary lesion.

2. A *crust* or scab is dried serum, blood or pus.

3. *Fissures,* ulcers, cracks, excoriations are breaches of the surface. They may extend to and involve part of the dermis. *Cicatrices* or scars may follow when a sufficient extent or depth of skin is involved.

PIGMENTARY ALTERATIONS: *Melanoderma* indicates excessive pigmentation, due to a deposit of melanin, which is brought about by many causes. It may be diffuse or localised. *Chloasma* is a broad patch of excessive pigment; *leucoderma* is an area of skin devoid of normal pigment. *Ephelis* is a freckle. *Nævus* is a mole or birthmark, either pigmented, hairy, or vascular. A dilatation of the superficial vessels of the skin is known as *telangiectasis*. *Petechiæ* are small spots of hæmorrhage into the skin; they do not fade on pressure. *Ecchymoses* are larger patches of extravasated blood which go through the changes of colour characteristic of a bruise. Both forms of hæmorrhage occur with

purpura (§ 653). A *comedo* or " blackhead " is a little plug of sebum and horny cells in a pilo-sebaceous follicle.

The fundamental **histological changes** of the skin are congestion (hyperæmia) with or without exudation, inflammation, and infiltration. If the lesion consists of *congestion*, such as roseola, or urticaria, or simple *inflammation* without infiltration, such as eczema, it disappears on pressure. If, on the other hand, there be definite *infiltration* or neoplastic deposit, as in lupus and syphilis, or if there be *hæmorrhage* into the skin, the colour does not disappear when the skin is pressed by the finger or a glass slide, or stretched. This is a point of much significance in the diagnosis of skin diseases. There are three secondary consequences of inflammation in the skin. If resolution does not occur, there may be (1) *suppuration*, leading to pustules, ulcers, etc.; (2) *necrosis*, as in the centre of boils and carbuncles; or (3) *organisation*, as in the case of the various scars, hypertrophies, or scleroderma. In addition to the primary lesions—congestion, inflammation, and infiltration and their consequences —which occur in the skin as elsewhere, there are at least three processes special to the skin. 1. *Hyperkeratosis* is an increased deposit of kerato-hyaline material leading to an increased cornification of the surface cells of the epidermis. 2. *Parakeratosis* is the irregular or deficient cornification best exemplified by psoriasis. The prickle cells, instead of going through the **regular** process of cornification by the deposit of kerato-hyaline granules in their **interior**, and gradual conversion into dry, horny, non-nuclear cells, remain moist (though dry on their exterior), and retain their nuclei. They adhere to one another and are shed in masses of crusts and scales instead of being shed singly and imperceptibly. 3. *Acanthosis* is a term applied to the increased proliferation of the prickle cells by increased mitosis (karyokinesis), resulting in thickening of the stratum germinativum. 2 and 3 are found in all kinds of eczema; 2 chiefly in dry eczema and 3 in moist eczema.

II. What does the **eruption feel like,** and does it **disappear on pressure ?** Infiltrating lesions feel hard, and do not entirely disappear on pressure, as is evident from the histological characters (*vide supra*). A faint purpuric eruption may thus be diagnosed from an erythema.

III. The **distribution, position** and **symmetry of the eruption** is important for purposes of diagnosis, and it is therefore essential to examine the whole of the eruption. Many diseases may be recognised by the positions where the lesions are most numerous. Figs. 141 and 142 aid the student to remember the parts most frequently affected by certain eruptions. Some diseases are always more or less generalised—*e.g.*, urticaria and the exanthemata; this distribution usually indicates a toxæmic cause. Others, while sometimes affecting the whole body, have a preference for certain parts—*e.g.*, psoriasis for the knees and elbows, acne for the face and shoulders. Various terms are used to describe the distribution: thus, *punctate* when the eruption is dotted about, *discrete* when the elements are separate, *confluent* when they run together, *serpiginous*, *gyrate* or crescentic when arranged in wavy lines or segments of circles, *circinate* or annular when in circles, *corymbose* when grouped in clusters.

Any **symmetry** of arrangement on the two sides of the body should be carefully observed, though its significance must not be overrated. It may indicate the presence of some circulating toxin, as in the earlier lesions of syphilis and certain erythematous eruptions, or it may be due

to the fact that both sides are exposed to the same external conditions, as in occupational or contact dermatitis.

IV. **Subjective Symptoms** must be inquired into, such as itching, burning, smarting, etc. Syphilitic eruptions do not usually itch, a feature which helps to distinguish them. The majority of skin diseases are *unattended by obvious constitutional symptoms.*

V. The **Duration of the Eruption** and the history of its **Evolution.** The rate at which a disease has developed is a most important aid to diagnosis. For instance, lupus vulgaris will not produce so extensive a lesion in the course of years as a facial syphilide, which resembles it, will produce in the course of weeks or months. During its progress a skin disease may alter its appearance considerably; a lesion which starts as a papule may become a vesicle and then a pustule, as in small-pox.

VI. **Etiology.**—PREDISPOSING CAUSES: 1. The *age.* Lupus vulgaris usually starts in early life, lupus erythematosus after twenty-five. 2. *Sex* does not aid much in diagnosis. 3. *Heredity:* psoriasis, alopecia areata, allergic and other eruptions often run in families. 4. The *occupation* may provide an important clue as to external irritants, and 5, the *habits* of life, as to diet, drink or drugs. 6. See remarks above on symmetrical distribution.

EXCITING CAUSES.—1. A dermatitis indistinguishable from that due to internal causes can be produced by external or traumatic causes, such as soap, dyes, chemicals and plants. Friction, as from clothing, and scratching greatly modify any eruption. 2. *Parasites* produce characteristic eruptions. 3. *Micro-organisms* (*e.g.*, staphylococci and streptococci, B. coli, diphtheria, tubercle, etc.). In the eruptive fevers the organisms reach the skin *via* the circulation. 4. *Fungi, yeasts* and *monilia.* 5. Certain *drugs* cause characteristic eruptions (§ 612). 6. *Sensitisation* to certain organisms and fungi, to foods and chemical substances, induces different types of eruption, *e.g.*, urticaria, erythema, eczema or papules. 7. *Diseases of the internal organs*, especially digestive disturbances (urticaria), disease of the peripheral nerves and their ganglia (herpes, glossy skin, and other trophic changes), acute and chronic renal disease, diseases of the liver, and other abdominal diseases. 8. *Vascular changes.*

§ **608. Dermatitis Artefacta** is a self-inflicted lesion of the skin. It may be single or multiple, and may vary from erythema to deep ulceration, as it is caused by the application of strong acids or alkalies, blistering fluids or heat. The differentiating features are (1) its usually linear form and angular outline, and (2) its appearance on areas easily reached by the hand. Sir Norman Walker pointed out that if the physician remarked in the presence of the patient that a fresh lesion would probably develop in a certain region, this would appear within a day or two. It occurs in hysterical or otherwise neurotic cases and malingerers, and is dealt with by occlusive dressings and psychotherapy. (See § 888.)

PART C. DIAGNOSIS, PROGNOSIS, AND TREATMENT OF SKIN DISEASES

Routine Procedure and Classification.—The LEADING SYMPTOM is generally before our eyes. The HISTORY, DURATION, and MODE OF EVOLUTION can be inquired into while the patient undresses. Then we proceed to the PHYSICAL EXAMINATION as described in Part B.

If the eruption is QUITE DRY, and consists of (*a*) *wheals*, turn to	§ 609
(*b*) *macules* or *erythema*, turn to	§ 610
(*c*) *papules*, turn to	§ 619
(*d*) *scales*, turn to	§ 626
If the eruption is MOIST, or consists of serous exudation, vesicles, bullæ or crusts, turn first to	§ 634
If the eruption consists of *pustules*, turn first to	§ 640

FIGS. 141 and 142.—DIAGRAMS showing the parts most frequently affected by certain eruptions.

If it is *multiform* .. .. § 645
If it is *nodular* .. .. § 646
If there is *ulceration*.. .. .. § 649
If there are *warts* or *excrescences* .. .. § 650
If there are *scars* or *atrophies* .. .. § 651
If there are *vascular* or *pigmentary* alterations .. .. § 652
If there is disorder of the *sweat* .. .. § 654
If the *hair* or *scalp* is affected .. .. § 655

GROUP I. ERUPTIONS USUALLY DRY

(*a*) *Wheals*

§ **609. Urticaria** (" nettle-rash ") is a generalised eruption which consists of firm round or oval, pink swellings, white in the centre when scratched. These typical wheals are of more or less evanescent character, rarely lasting more than a few hours. The rapid onset and disappearance of the individual lesions is characteristic. Patients come complaining of the *history* of such an eruption preceded and accompanied by intolerable itching. Sometimes, although there are no wheals visible, these can readily be produced by drawing a point across the skin (dermatographia or urticaria factitia).

Varieties.—(1) There is an acute and chronic form; the first-named lasts a few hours or days; in the chronic or *recurrent* form there are constantly recurring attacks. (2) Lichen urticatus or papular urticaria affects infants and young children. Red transient blotches appear with a central papule, very itchy, which lasts several days. Occasionally it has a vesicle or a bulla on the top (bullous urticaria). (3) Angioneurotic œdema or giant urticaria (Quincke's disease) has very large swellings involving the loose subcutaneous tissue. Each lesion may last a few hours or days. There may be danger to life, especially when the mucous membrane of the tongue or larynx is involved. It may occur in association with purpura. (4) *Urticaria pigmentosa*; see § 653. (5) " *Serum Disease.*" This form may appear after serum injections, and is associated with a group of symptoms which are dealt with in § 521.

Etiology.—A wheal is the neuro-vascular cutaneous reaction to injury; the skin reacts to irritants of external and internal origin. Histamine is freed from the cells by trauma, infection or a toxin, or from a chemical, thermal or electrical irritant; the skin reacts with Lewis' " triple response " : (i.) local and direct dilatation of the minute skin vessels; (ii.) dilatation of the larger vessels, by nervous reflex, with flushing of the adjacent skin; (iii.) local increased permeability of the vessel walls, leading to exudation of serum. Common causes are: (1) Insect bites: bugs, mosquitoes, the stings of nettle or jelly-fish. (2) Nervous or emotional causes: some persons develop urticaria on meeting a stranger or before addressing a public gathering. (3) The state of allergy, *i.e.*, an inborn sensitiveness which in certain people and families is associated with hay fever, asthma and eczema or prurigo. Common foods to cause urticaria

in sensitive subjects are eggs, shell-fish, pork, acid fruits or wines. Rarer causes are intestinal worms and hydatid cysts; suspect these in the tropics; eosinophilia is usually present. (4) Gastro-intestinal toxins: bad fish, tinned foods, etc. (5) Intestinal or liver dysfunction and after enemata. (6) Bacterial causes: urticaria has disappeared on removal of septic teeth, appendix or a discharge. (7) Susceptibility to certain drugs (see § 612). (8) In rare cases urticaria follows exposure to a blow, heat, light, cold, or a bath. (9) Excess of nicotinic acid.

The subject of ALLERGY has received attention of recent years. The "allergic state" includes hay fever, asthma, many forms of urticaria, pruritus and eczema, migraine, paroxysmal hydrarthrosis and epilepsy. When a patient is sensitised to a certain substance his cells react violently towards it; to him it is poison. There is some similarity between anaphylaxis and allergy. Anaphylaxis is an acquired sensitiveness; allergy in some cases is inborn and runs in families. Anaphylaxis appears after serum injections given to one who is sensitised to serum. In anaphylaxis the date of entry of the serum is known; in allergy the time and portal of entry of the offending substance are unknown. Other factors complicate the problem; the digestion in allergic subjects is often defective, the liver fails to deal with the imperfect products of digestion and these enter the circulation. The manifestations of allergy can be produced by histamine. It is difficult to track down the allergen in operation in the individual case; Langdon Brown suggested an inborn deficiency of histaminase, which normally destroys histamine.

Cutaneous allergy. The individual becomes sensitised by repeated external or internal contact with various antigens. Even normal people can be rendered sensitive to certain substances when a large enough dose is administered. The sensitised skin reacts in several ways: erythema (dilated vessels); urticaria and œdema; vesicles and bullæ (serous exudation). When the cells of the epidermis are sensitised, there results eczema and dermatitis; when the cells of the dermis, urticaria and œdema.

Prognosis and Treatment.—Locally, use 2 per cent. phenol in calamine lotion, 1 to 3 per cent. liq. carb. deterg. or 1 per cent. menthol or camphor in lotions or powders; avoid ointments, hot baths and rough garments. Many cases subside in a few days, with a saline purge and milk diet. Glucose is valuable because it aids the antitoxic function of the liver. In recurrent types the cause may be difficult to trace; the patch test may assist. First consider the diet: give milk alone for several days; add various articles and note which are followed by urticaria. Elimination diet tables are used for obstinate cases (Rowe). Desensitisation may be effected by giving a minute quantity of the offending article of food an hour before the meal in which it figures; for non-specific desensitisation give peptone (0·5 G.) three-quarters of an hour before meals, or inject it intramuscularly twice a week. Autohæmotherapy may be required (§ 656). Calcium salts in large doses can be given by mouth, or by daily intramuscular or intravenous injection. If the gastric digestion is at fault, give hydrochloric acid and pepsin; if the colon is unhealthy, kaolin, charcoal, lavage and B. acidophilus therapy, or antiseptics such as salol and ichthyol. With lichen urticatus, Hallam found that the child recovers if it sleeps in hospital, which suggests an underlying emotional cause; bromides, calcium lactophosph. and hyd. c̄. cret. aid most cases. Urticaria can be aborted by 5 to 10 drops of adrenalin in one ounce of water, or

ephedrine gr. $\frac{1}{3}$ to $\frac{1}{2}$ twice daily. Serious forms of angioneurotic œdema require adrenalin injections ($\frac{1}{2}$ c.c. of 1 in 1000 solution). Certain antihistamine agents are phenolic ethers, effective during the period of administration. This does not exempt one from search for and removal of the allergen, or desensitisation of the patient. Of these drugs may be mentioned benadryl (50 mgm., t.d.s., followed by a course of injections of lertigon), antistin, anthisan and phenergan. After a time these drugs become less effective. When side reactions follow (drowsiness, lassitude, nausea, dizziness), give small doses of ephedrine, caffeine or benzedrine.

(*b*) *Eruptions which usually consist of Macules or Erythema*

Generalised.	*Localised.*
I. Exanthemata.	I. Rosacea.
II. Roseola (simplex and syphilitica).	II. Lupus erythematosus.
III. Erythema scarlatiniforme.	III. Erythema nodosum.
IV. Drug eruptions.	IV. Erythematous eczema, X-ray dermatitis, Bedsores, E. faciei, E. traumaticum, E. caloricum, E. pernio, and other varieties of Erythema, Livedo ; Macular Leprosy, and Pellagra.
V. Erythema multiforme.	

The early stages of eczema and of other eruptions may appear as erythema.

I. The **Exanthemata** or eruptive fevers are fully described in Chapter XV, where they form Group I of the acute specific fevers.

§ **610.** II. **Roseola** is a term employed to designate a generalised eruption consisting of patches of congestion, more or less marginated, varying in size from a pin's head to a lentil. Two main varieties are described.

Roseola Simplex may resemble measles; its chief importance is in connection with the diagnosis from this disease : it gives rise to itching, with slight constitutional disturbance. It may occur in childhood under the same conditions as urticaria, due to gastro-intestinal toxins. Its occurrence when small-pox is prevalent should make one suspect the initial eruptions of that disease. It is one of the commonest rashes associated with vaccination. Drugs may cause it (see § 612).

Roseola Syphilitica is the earliest of the syphilitic eruptions, occurring three to six weeks after infection upon the trunk, chiefly its anterior aspect, the chest, the flexures of the limbs and the palms and soles, as rosy macules becoming dusky red in a few days, disappearing on pressure, rounded, oval or irregular in shape with fading edges, varying in size from a pea to a shilling. It may last a few days to a few weeks, leaving behind it some pigmentation. Sometimes the eruption is so faint that it is overlooked ; prominence of the hair follicles may be noted first, and the rash becomes better marked after a bath or when the skin is exposed to cold. It is *diagnosed* by the history, other signs of syphilis and absence of itching. *Non-syphilitic roseola* undergoes rapid changes in size and shape ; *pityriasis*

versicolor can be scraped off and is fawn-coloured; *pityriasis rosea*, *seborrhœic dermatitis*, *eczema* and *parapsoriasis* have scales or crusts.

§ 611. III. **Erythema Scarlatiniforme**, as its name implies, consists of a widespread rash, resembling scarlet fever, preceded and accompanied by fever and constitutional disturbance, and followed by desquamation. So-called "surgical scarlatina" is probably identical with this condition. The chief causes are intestinal disorders, enemata, focal infection, streptococcal tonsillitis, sensitiveness to a foreign protein, food poisoning, certain drugs (see below), rheumatism and gonorrhœa. The *Diagnosis* from scarlet fever is difficult only in severe cases. In erythema there is less constitutional disturbance, no strawberry tongue, and there is a tendency to relapse.

§ 612. IV. **Drug Eruptions.**—An idiosyncrasy with regard to certain drugs, whether taken by mouth or applied externally, is shown in some individuals by a rash, which disappears on the withdrawal of the drug. Iodides and bromides often produce eruptions; they may even cause a *frambœsial* eruption resembling gumma. The chief eruptions produced by the internal administration of drugs are:

Morbilliform: antipyrin, copaiba, the sulphonamide group, penicillin, the barbiturates (sometimes with itching and fever).

Papulo-Pustules: Bromide and iodide of potassium (chiefly on the face), occasionally sulphide of calcium, antimony, arsenic, and mercury.

Papules: Bromide and iodides (chiefly on the face); penicillin; arsenic, gold salts and other metallic injections (lichenoid papules especially).

Erythema: Antipyrin (in round, raised patches recurring on the same site), antitoxins, cinchophen (atophan), atropine, belladonna (sometimes with fever), boracic acid, chloral hydrate, copaiba, cubebs, ephedrine, gold and other metallic injections, phenobarbitone, mercury, morphia, phenolphthalein (oval patches becoming deep red or purple, then pigmented), quinine, santal, the sulphonamide group, salicylic acid and sodium salicylate. Turpentine, iodoform and phenol by absorption from wound dressings.

Urticaria: Arsenic, atophan, chloral hydrate, copaiba, nicotinic acid, phenobarbitone, mercury, phenacetin, phenolphthalein, quinine, santonin, salicylates, turpentine, penicillin.

Erysipelatoid: (erythema with infiltration or œdema of the skin). Antipyrin, atophan, boracic acid and phenol, bromides and iodides, iodoform, mercury, phenolphthalein, quinine, the sulphonamide group. Aconite, oil of cade, chrysarobin, and phenol applied externally.

Herpes: Arsenic and other metallic injections.

Bullæ: Antipyrin, arsenic, the barbiturates, bromide, chloral hydrate, gold, iodides, opium, quinine.

Purpura: Iodide of potassium, chlorate of potash, chloral hydrate, chloroform, urea compounds, copaiba, T.N.T., sulphadiazine, adalin.

Pigmentation: Silver nitrate, arsenic, antipyrin, phenolphthalein.

Epidermic Thickening: Borax, boracic acid, injections of arsenic, the arsenobenzol group and gold.

Exfoliative dermatitis: Boracic acid, metallic injections, chiefly arsenic, gold and bismuth.

§ 613. V. **Erythema Multiforme** is an erythematous rash chiefly localised to the backs of the hands, forearms, feet or legs, sometimes the face, neck and trunk, and associated with lassitude or ill-health. The lesions vary in size from a lentil to the palm of the hand, are slightly raised, with fading edges. The centre is highest, usually livid, even hæmorrhagic. Varieties: *E. Iris*, with a central vesicle (Fig. 143); *E. Bullosum*, with central bulla; *E. Gyrata*, when adjoining patches coalesce and form wavy outlines; *E. Annulare*, with ring-like edges; *E. Centrifugum annulare*, which lasts long. A virus may be responsible when the mouth and throat are affected, as in a rare serous syndrome. *E. nodosum* is described in § 616. Erythema is known

from urticaria by its deep red coloration, more localised distribution, the larger size and more permanent character of the lesions, less itching, and more marked constitutional symptoms. Young people and males are chiefly affected. It is commoner in the spring and autumn. The *course* of the disease varies from eight to ten days, and lesions continue to appear for two to six weeks. Each may leave temporary brown pigmentation, and desquamation may occur as they fade. Complications are rare.

Etiology.—A history of food poisoning is often found. In many obstinate cases there is a septic focus in the naso-pharynx, mouth or sinuses. *E. Annulare centrifugum* is usually due to sensitisation of the vessels to streptococci or to other obscure toxic or allergic forming substances.

Treatment.—Locally, cooling lotions and powders alone are required. Rest is indicated lest any generalised infection attack the organs. The digestive system must



FIG 113.—ERYTHEMA IRIS on the hand of a single woman twenty-three years of age.

be regulated. Internally, give quinine gr. 5 t.i.d. and calcium lactate gr. 10 or gluconate gr. 25 after meals. Salicylate of soda with sodium bicarbonate aids other cases. The cause should be sought for and removed between the attacks. Vaccine and sulphonamide or penicillin therapy is indicated when due to streptococcal sensitisation.

Erythema of more or less LOCALISED *distribution.*

§ **614.** I. **Rosacea** (Synonym: Acne Rosacea) presents three stages: (1) Simple congestion or erythema of the nose and adjacent parts of the cheeks, often worse after meals or exposure to heat. (2) Dilated vessels (telangiectases). Persistent erythematous patches may develop, and in certain cases, especially when there is dandruff on the scalp, papules, small pustules and enlarged sebaceous glands. (3) In severe cases hypertrophy, with nodules of great size (rhinophyma). Rosacea runs a prolonged course; the first stage alone may extend over many years.

The *Diagnosis* is not difficult, except in its early stage, when the erythema may be mistaken for lupus erythematosus and other kinds of erythema of the face (§ 617). The former, however, is recognised when

a lens reveals the presence of a fine "tissue-paper" scarring. The absence of comedones distinguishes it from acne vulgaris.

Etiology.—Rosacea affects both sexes, but is more common in women. It also affects cabmen, coachmen, mariners, and others exposed to the weather. Alcoholism is a frequent cause, but total abstainers may have rosacea. Dyspepsia is a common cause; test meals have often revealed deficient hydrochloric acid. Severe cases usually have liver or intestinal trouble. In other subjects it is associated with catarrhal conditions of the nose and throat, sinuses, or infection from obscure foci, *e.g.*, teeth, cervix or gall-bladder.

Treatment.—In the first stage apply soothing remedies such as calamine lotion; later, ichthammol. Add weak sulphur when the scalp has dandruff. Alkalies with gentian before meals aid many; where the acid secretion is at fault give hydrochloric acid after meals. Farinaceous and sweet foods should be restricted and fluid taken between meals. Many benefit with calcium; others require endocrine therapy. Treat dilated veins with the galvanic current. Some use small doses of X-ray. For rhinophyma, diathermy fulguration or cautery and cosmetic surgery are required.

§ 615. II. **Lupus Erythematosus** is the most chronic of the erythemata. The eruption has a spreading erythematous border, which as it spreads leaves a very thin permanent scar in the centre. In the first stage the disease begins with one or more small, red, slightly raised spots. By spreading at the margin and increasing in number the little patches form, in the course of many months, an irregular bluish-red area, with thin cicatricial centre and erythematous margin covered with scales, and sometimes with crusts. In another variety there is a marginated erythema with numerous black specks, or large gaping openings of the sebaceous glands; the central part of the skin appearing depressed, and covered with adherent dry scales, interspersed with venules. The favourite sites of the eruption are the cheeks and bridge of the nose (butterfly distribution); then other parts of the face and forehead, the lips, ears, scalp, the extensor surfaces of the hands, fingers and toes, and more rarely on other parts of the body. The patches are generally symmetrical. In rare cases the erythematous patches become rapidly widespread over the body, and severe constitutional symptoms are present.

Etiology.—The disease is more frequent in women than men, and rarely occurs under twenty, an important fact in the diagnosis from *lupus vulgaris*. Direct sunlight and reflection from snow and water can excite new lesions. The disease can be caused both by tubercle and by streptococci; the acute form is probably streptococcal.

The *Diagnosis* from *lupus vulgaris* is given in tabular form (§ 646). Before cicatrices appear it may be hard to distinguish from *Rosacea*. The acute disseminated type may be confused with *Erythema Multiforme.*

Prognosis.—The chronic or discoid type of L. erythematosus extends over ten or twenty years, always terminates in cicatricial changes in the skin, and permanent baldness of a hairy part. Beyond the disfigurement the chronic form of the disease is not serious. The acute disseminate variety usually terminates fatally.

Treatment.—In the early stage employ soothing remedies (*vide* acute eczema). Painting with pure carbolic or carbol-camphor, linear scarification, diathermy and carbon dioxide snow have all given satisfactory results. Internally quinine, salicylic acid and intestinal disinfectants have cured mild cases. Cure has sometimes followed an autogenous vaccine after removal of a septic focus. Bismuth and gold therapy are the mainstay in treatment. Once a week bismuth is given intramuscularly.

Gold is used in afebrile and tuberculous types of the disease; myocrisin (0·05 G.) in aqueous solution can be injected weekly, for ten weeks or longer. Sulphonamide drugs should be used cautiously, lest latent foci become active. Penicillin, in acute disseminated cases, has been disappointing; relapse follows initial improvement.

§ 616. III. **Erythema Nodosum** is an eruption with an acute onset, consisting of erythematous lumps about the size of a pigeon's egg, occurring most frequently over both shins or just above the knees, round or oval, raised, non-marginated, painful and tender. The centre is deeply coloured, whence the purplish tint gradually fades to the margin. There is usually some malaise and pyrexia; sometimes joint pains and other rheumatic symptoms. Each nodule lasts one to two weeks, and successive crops may continue for a month or two. They never ulcerate. Patients are usually young women with a rheumatic or tuberculous tendency. The condition is *diagnosed* by the position of the lesions and the acute pain and tenderness. In periostitis the lesion is usually single. The disease, in streptococcal cases, usually recovers in a month or two, but may recur. *Treatment.*—Give salicylates, saline aperients, and after the acute symptoms have subsided, iron and quinine. Lead and opium lotion allays the pain. As certain cases appear to be due to latent tuberculous infection one must make careful search for the cause; when of streptococcal origin seek to eradicate any septic focus.

§ 617. IV. Other forms of erythema to be borne in mind are:

Erythematous Eczema may run its course without presenting any vesicles. The skin is red, dry, and rough, with slight scaling. It frequently attacks the face, when the eyes may be almost closed, and is attended by burning and itching. See Eczema (§ 634).

Dermatitis due to **dyes,** hair and fur dyes, especially those containing paraphenyldiamin, or work with **munition** products such as trinitrotoluol, may cause a blotchy erythema which may pass on to acute vesiculation. Certain **plants,** *e.g.*, primula obconica and rhus toxicodendron, bulbs and citrus fruit skins, may be the cause of recurrent dermatitis in susceptible persons (dermatitis venenata). The causes of contact and traumatic dermatitis are legion and demand patient investigation (§ 634).

X-ray Dermatitis may be acute, consequent on a single large dose, or chronic, after repeated small doses of X-rays. In the acute form there is erythema, swelling, sometimes bullæ, and sensations of burning or intense pain, according to the severity of the reaction. In mild chronic cases there is temporary loss of hair and pigmentation. Years later, telangiectases develop; atrophy, fissures, warts, ulcers and malignant disease may follow. Treatment is prevention by ensuring greater protection to X-ray workers. Sedative lotions and pastes hasten the recovery of acute dermatitis; antiseptics should not be employed. For chronic forms forbid further X-ray work.

Bedsores are due to pressure over bony parts, such as the sacrum, trochanters, heels, or ankles of the bedridden, or to the pressure of a badly adjusted splint or plaster. A local patch of erythema appears, then ulceration and slough form. Bedsores have three causes: pressure, perspiration and excretions in cases of incontinence, the lowered vitality of the sick and aged. In certain nerve diseases, especially myelitis, the sloughs form rapidly. Necrosis may lead to emaciation and septicæmia.

Treatment.—Good nursing prevents postural bedsores, by cleanliness, dryness, and relief of pressure. (i.) The parts should be cleansed night and morning and the draw-sheet pulled through immediately when soiled. (ii.) After washing, the skin should be dried, gently massaged, rubbed over with surgical spirit and powder. (iii.) Relieve pressure by a water-bed, ring pads, and by changing the patient's position every one or two hours. If an ulcer or slough forms, when not near bone and the infection is not virulent, cover with adhesive plaster overlapping an inch on the healthy skin. Excise necrosed tissue; give sulphathiazole or penicillin as indicated.

Erythema Faciei is a flushing of the face which occurs chiefly in association with dyspepsia. It may follow exposure to bright sunlight; too long exposure

may cause dermatitis. Treat with soothing creams. Prevent by applying calamine paste or lotion containing 2 per cent. quinine, 10 per cent. tannic acid in vaseline or in 25 per cent. spirit or *p*-aminobenzioc acid in vanishing cream or in spirit. *E. traumaticum* develops on any part subject to long-continued pressure—*e.g.*, the garters and tight waist-bands. *E. læve* is found on the legs of dropsical persons. *E. caloricum* appears on the shins, due to sitting close to a fire. *E. intertrigo* is found in opposite parts, such as the thighs and armpits, in infants and corpulent people. It may become infected with monilia or streptococci. *E. pernio* (Synonyms : dermatitis congelationis, frostbite, chilblain) is a painful inflammatory condition of the skin of the fingers, toes, heels, or other portions of the feet or hands, caused by exposure to cold and damp, and attended with itching and tenderness, sometimes by vesication, ulceration, or gangrene. Children and old people frequently suffer from this complaint during successive winters. *Livedo annularis* is the purplish mottling seen on the extremities of chilly people when it has reached the stage of resembling a permanent network. *Treatment:* Use stimulating liniments, iodine and camphor, the sinusoidal current and paraffin wax applications. Itching is relieved by bathing in very hot water. Give calcium (especially with high potency calciferol), thyroid, iron and arsenic, and ultra violet to the whole body.

Other forms of chronic Erythema due to a toxæmia appear to be closely allied; sensitivity to a streptococcus is suspected. *E. Annulare* : circinate and gyrate lesions appear on the neck, trunk and limbs. They last some days ; fresh crops may appear for months. Especially is this type seen in children with acute rheumatism, when it may be overlooked. *E. Annulare centrifugum* begins as papules and extends to form circinate and gyrate patches with raised hard margins. It may last for months, and recurrence is usual. *E. perstans* **has large** lesions, very persistent. *E. elevatum diutinum* has symmetrical elevated flat dark plaques over pressure points. It is thought to be due to rheumatism.

§ **618. Pellagra** is a deficiency disease occurring commonly, but by no means only, in maize eaters.

Symptoms.—The classical clinical syndrome of dermatitis, diarrhœa and dementia is encountered only in advanced cases ; many patients first consult their doctor on account of *prodromal symptoms :* anorexia, asthenia, loss of weight, dyspepsia, insomnia, nervousness, palpitation, mental depression, forgetfulness and mental confusion. According to Wood, the seasonal incidence is important, prodromal symptoms occurring late in the winter, alimentary features in the early spring, and pellagrous dermatitis in the early summer. (i.) The *skin* lesions appear on areas of the body exposed to mechanical irritation or the sun, such as the dorsum of the hands, wrists, elbows, face, neck, knees, feet, perineal region, and under the breasts. The rash begins as an erythema, resembling severe sunburn, accompanied by burning, itching and sometimes vesiculation. Gradually the acute phase subsides, the skin becomes thickened, reddish-brown and itchy. Desquamation follows. The rash is symmetrical, with a sharply demarcated, often pigmented, border. Sometimes skin lesions never appear : "pellagra sine pellagra." (ii.) Involvement of the *gastro-intestinal tract* shows in the later stages by glossitis and stomatitis. At first the tongue is red and swollen only at the tip and margin ; later it is all involved ("beet" tongue). Burning pain in the tongue, pharynx, œsophagus and stomach aggravated by condiments and hot and acid foods may cause bitter complaint. Other alimentary features include nausea, vomiting, ptyalism, abdominal pain, discomfort and distension after food, and severe persistent diarrhœa. Some 60 per cent. of cases show achylia gastrica ; anæmia, in some cases of the megalocytic type, is not uncommon. Other mucous membrane lesions are vaginitis, proctitis and urethritis. (iii.) *Nervous* symptoms : early depression, apprehension, increased irritability, insomnia, headache and burning sensations in the extremities. Later, tremor, jerky movements, altered reflexes and a spastic or ataxic gait may develop. Insanity often supervenes ; many patients die in mental hospitals.

Prognosis.—The disease runs a chronic course, is subject to remissions and seasonal fluctuation, and, if untreated, leads to death in from 3 to 15 years.

Etiology.—Pellagra may occur at any age, in both sexes, in any race; it is endemic in lower Egypt, Turkey, Spain, Italy and parts of the Southern United States. It is related in part to deficiency of nicotinic acid in the diet, and is often associated with increased porphyrinuria. According to Spies, it is found in three groups of people: (1) The indigent and those with erroneous dietetic habits and idiosyncrasy. As a rule there is a history of a long period of diet high in carbohydrates and fats and relatively low in protein, minerals and vitamins. Lean meat, eggs, milk, fish, fresh fruits and vegetables, which are of value in preventing and treating pellagra, are generally lacking. (2) Chronic alcoholics who take an inadequate diet, substituting alcohol for food. (3) People with organic disease involving the gastro-intestinal tract, cirrhosis of the liver and the like, which interfere with the normal utilisation of food.

Treatment.—Pellagra is prevented by foods rich in the pellagra-preventing factor, such as yeast, marmite and wheat germ. The minimal preventive dosage is 8 to 10 mgm. nicotinic acid daily. Once the disease has developed the patient should go to bed on a high calorie diet, rich in protein and low in carbohydrate. Fresh milk, lean meat, liver, marmite, tomato juice and vegetables are of special value. Nicotinic acid in adequate dosage heals the alimentary and mucous membrane lesions, induces blanching of the erythema, reduces the porphyrinuria to normal, and is followed by improvement of the mental symptoms. Give 500 mgm., one tablet (50 mgm.) every 2 hours for 10 doses for 10 days, then gradually reduce. With children and infants hog's stomach has proved more effective than liver. Co-existing diseases must be treated; for associated polyneuritis give vitamin B_1 parenterally, as nicotinic acid does not cure this complication.

Macular Leprosy appears as brownish or mahogany-red patches of erythema of various sizes (§ 647).

LEUKÆMIA and MYCOSIS FUNGOIDES show large areas of infiltrated dusky erythema with intense itching (§ 647).

With DERMATOMYOSITIS there may occur a widespread erythema, often accompanied by telangiectases and recurrent œdema, especially of the face (§ 594). It is usually associated with some degree of scleroderma.

(c) *Eruptions which usually consist of Papular Elements*

Common.

I. Acne vulgaris.
II. Prurigo.
III. Scabies.
IV. Skin diseases sometimes papular at *one stage*: (i.) Papular eczema.
  (ii.) Psoriasis and other scaly eruptions.
  (iii.) Exanthemata.
  (iv.) Pustular and vesicular eruptions.
  (v.) Nodular eruptions.
V. Milium.
VI. Keratosis pilaris.
VII. Lichen planus.
VIII. Papular syphilide.

Rare.

IX. Lichen scrofulosorum.
X. Adenoma sebaceum.
XI. Granuloma annulare.
XII. Trichophytides.
XIII. Keratosis follicularis.
XIV. Fox-Fordyce disease.

§ 619. I. **Acne vulgaris** is a common disease, with comedones and papules which are very persistent, and often pass on to pustules, with discharge and resulting scars. The skin of the acne patient is usually oily, coarse and dusky. The eruption affects chiefly the face, and often the back, shoulders and chest, parts where the sebaceous glands are active.

The comedo is characteristic, with its black point at the mouth of the follicles. This so-called "blackhead" is due to a chemical change in the hyperkeratosis which blocks the gland and leads to retention of sebum and subsequent inflammation around.

Etiology.—Acne usually starts at puberty, when the pilo-sebaceous glands become active; both sexes are affected. There is excessive oily secretion of the skin, often also of the scalp; the enlarged follicles contain overgrowth of the horny cells and masses of Sabouraud's micro-bacillus. The work of Darier, Barber and others suggests that there is a definite "seborrhœic state," with altered secretion and composition of the cutaneous fat, which permits the growth of certain micro-organisms more readily than in normal skins. There is usually excessive acidity of the urine, often constipation and dyspepsia. The underlying cause is endocrine imbalance, especially excess of the male hormone, combined with dietetic errors, such as excess of carbohydrate and fat in the diet, defective oxidation processes and too little exercise. The blood sugar may be high. Fresh crops of papules often follow over-eating, especially of chocolates and fatty foods, and also of sweets and wines.

Varieties.—(1) *Acne punctata, A. indurata,* and *A. pustulosa* are stages, not true varieties. (2) *Bromide* and *iodide acne* are indistinguishable; the individual spots closely resemble acne vulgaris, but comedones are absent; they affect chiefly the chest and back, and the face is always first affected. (3) Working with *tar* and *mineral oils* can produce acne with close-set comedones, then pustules on the areas exposed to these agents. Friction with camphorated oil has produced groups of comedones on the chest. (4) *Acne excoriée des jeunes filles* shows much disfigurement because the patient continually picks at the lesions. (5) *Acne varioliformis* has indurated papules, chiefly affecting the brow and adjacent scalp, but also sometimes the face and chest, rarely other parts. Itching is usual. Slowly a vesico-pustule forms and leaves a pitted scar.

The *Diagnosis* of acne is simple on account of its characteristic position and the presence of comedones. Papular, pustular, and tubercular *syphilides* affecting the face are usually copper-coloured, and grouped in a serpiginous manner. *Acne Rosacea* may accompany acne vulgaris: there is hyperæmia after meals, between the papules. *Lupus vulgaris* has characteristic apple-jelly nodules, and no comedones.

Treatment.—The local treatment of acne consists in employing soothing calamine lotions when there is much inflammation, and following up with sulphur ointment or lotion. Comedones must be gently pressed out daily with a comedo extractor. When comedones and hard, small papules predominate, the skin must be frequently washed with warm water and soap, followed by hard friction with a rough towel. When acne is not complicated by internal causes, it responds to sulphur externally. A sulphur ointment (20 to 40 grains to the ounce) should be rubbed on night and morning, or a sulphur lotion (*e.g.*, pot. sulphuret, zinc sulphat, āā gr. xx.; industrial spirit 120♏, Aq. ad. fl. oz. 1) or resorcin in strength varying

according to the individual skin. In some cases strong exfoliating remedies succeed; they are applied at night and the patient remains indoors during the peeling stage. For pustular acne give mercury (in ointment or lotion) as in all suppurating affections; in some cases try penicillin or sulphonamide therapy. Boracic compresses are used when the pustules are tender; lance later. Diet is important; in obstinate cases investigate the urine and the stools. Chocolates and alcohol should be forbidden; fats and carbohydrates cut down. In some cases bacon fat, in others the fats of butter, milk or cheese, are harmful. Treat constipation and dyspepsia, and remove any septic focus present. Fresh air, exercise and sunlight often work wonders. Large doses of alkalies are useful for many; for others use endocrine therapy, especially thyroid. Recently œstrin therapy has had success with men, and with women when the menstrual period is associated with exacerbation of the acne. Vaccines in small doses often aid pustular acne. Ultra-violet light, high frequency and massage all have their advocates. When the acne bacillus predominates, small repeated doses of X-ray benefit; care must be taken to avoid after-effects.

§ **620.** II. **Prurigo** is a disease in which the leading and sometimes the only symptom is generalised itching (pruritus), but it is frequently accompanied by an eruption of papules, urticarial patches, and scratch-marks. The papules are hard, shotty, acuminate, pale red, frequently better felt than seen (giving the sensation of a nutmeg-grater), appearing in crops

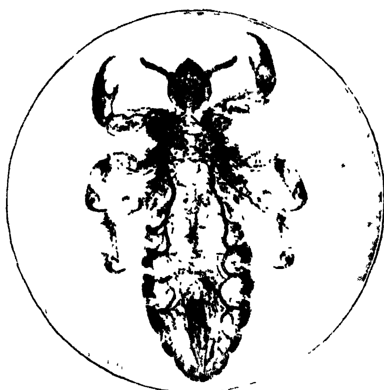


FIG. 144.—PEDICULUS CORPORIS, magnified about ten times.

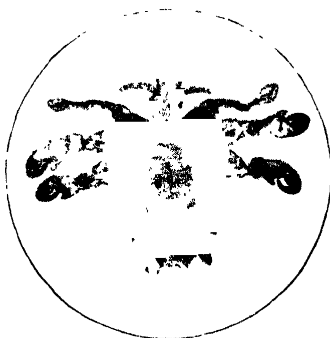


FIG. 145.—PEDICULUS PUBIS, magnified about ten times.

on the extensor surfaces of the thighs and arms, the trunk, especially the back and buttocks, and only occasionally the face. Each crop lasts a week or two, and is sometimes accompanied by urticarial blotches; dermatographia can generally be elicited. The intense itching leads to scratch-marks. In time prurigo is followed by a dry, rough, thickened, pigmented skin (lichenification).

Varieties.—Lichen Urticatus (Synonym: Urticaria Papulosa; see § 609). The papules are small, chiefly on the back, and the urticarial element moderate. It starts about the fourth month of life, and recurs until about the fourth year. In very young children lichen urticatus sometimes shows vesicles or bullæ, chiefly where the horny layer is thick (palms and soles). In *P. senilis* (Synonym: Pruritus Senilis) the eruption may be insignificant or absent, and the irritation intractable, with a tendency to induration and purpuric complications. *Besnier's prurigo* is usually an allergic manifestation; the patient or his relatives have had infantile eczema or asthma (§ 609); the eruption affects the flexures of the knees and elbows, parts of the face, dorsum of hands, wrists and ankles; it may become eczematised. In *P. ferox*, the prurigo of Hebra, all the lesions are on a larger scale, the inguinal glands involved, and the general health deteriorated. *P. hiemalis* occurs in cold countries or in the winter only.

The *Diagnosis* is simple in well-marked cases because of the intense itching and the thickened skin, with exaggeration of the natural grooves, like a mozaic (lichenification). The prolonged course of the disease is distinctive. In scabies the papules are almost confined to the *flexures of the joints* instead of the extensor surfaces; in pediculosis, to the *back* and *shoulders*, where are seen typical long scratch marks and pigmentation. The diagnosis from *papular eczema* is not always easy; especially as the areas in time, with scratching, often become eczematised, even secondarily infected. Lichen urticatus, when bullous, has been mistaken for *varicella*; in the latter the vesicles are superficial and the lesions occur in crops (often on the head and face, sometimes on the palate), so that several stages (papules, vesicles and crusts) are visible at once.

Etiology.—Prurigo tends to occur at the two extremes of life. The causes are the same as those of pruritus (§ 605). Some hereditary defect in digestion or the liver, leading to an allergic reaction to certain proteins, is probably the cause of the severe chronic type. Sources of toxæmia and sepsis may also cause it. See also urticaria and the allergic state, §§ 521, 609.

The *prognosis* depends upon the variety and the cause. Papular urticaria in childhood may respond quickly to treatment with diet and rest from nervous strain. Some types decline at puberty or about twenty-five. Besnier's prurigo tends to relapse throughout life. The severe forms of prurigo, which depend upon metabolic errors of assimilation, may last a lifetime.

Treatment.—The first indication is to discover the cause, for without carefully sifting this question no treatment can be successful. For Lichen urticatus, see § 609. Seek carefully for pediculosis, scabies or other parasites. For pediculi Ung. hyd. ammon. chlor. has been replaced by 10 per cent. D.D.T. powder. The seams of the clothing must be cleansed from the ova. For scabies, see § 621. Then seek for other sources of local irritation, as in the garments, or a discharge; and finally turn to the internal causes. The correct treatment may depend upon the findings

after investigation of the blood and excreta. Glycosuria, jaundice, leukæmia and renal insufficiency give clear indications. Potassium iodide has helped senile pruritus. Treatment is difficult when there is an obscure intestinal infection or septic focus. Raising the resistance with rest, good food and change of air is often successful. Arsenic and cod-liver oil do good, especially in children. In Besnier's prurigo, success has been gained with an open-air life, ultra-violet light, intestinal antiseptics, a lacto-vegetarian diet and antihistamine agents (§ 609). Calcium salts in large doses by mouth, or by intramuscular or intravenous injection are useful; calcium lactophosphate with bromide is a favourite remedy. In obstinate cases autohæmotherapy, autoserotherapy, and other methods of desensitisation (see §§ 521, 609) may be required. X-rays, locally and over the spinal roots, lumbar puncture and intravenous injections of bromides, all have their advocates. Drugs acting upon the sympathetic and parasympathetic play their part—pilocarpine and belladonna. For itching, use phenol 1 in 80, menthol ½ per cent., alkaline lotions, vinegar, chloral and camphor (60 gr. of each liquefied and added to 1 oz. starch powder). White's coal tar ointment is a good local dressing. Cocaine preparations must be used only for short periods.

§ **621.** III. **Scabies** is the eruption produced by the acarus scabiei. It consists of papules and vesicles of varying sizes; the latter often become infected and pustular. In addition to its multiform character, it is diagnosed in its typical form by (1) short white or black burrows due to the insect tunnelling in the stratum corneum; (2) scratch-marks; (3) severe itching, always worse at night when the patient is warm in bed; (4) its distribution—Scabies may extend over the trunk and limbs; it commences and predominates where the skin is thinnest—*i.e.*, between the fingers or toes, the flexures of the wrists and elbows and on the point of the elbows, the breasts and lower abdomen, the anterior borders of the axillæ, the penis, the inner side of the feet, ankles, thighs, and below the buttocks; in young children and infants the lesions can occur on the face, soles of the feet, and back of the neck; (5) the discovery of the insect or its eggs (Fig. 146); (6) other members of the family are often infected.



FIG. 146.—ACARUS SCABIEI (female), magnified about twenty times. The female burrows in the horny layer to lay her eggs, the burrows thus formed being typical and most frequent on the wrist and finger webs. The male roams over the body and clothes, and is rarely captured.

Etiology.—Scabies is usually acquired by sleeping with an infected person. It is disputed whether it can be contracted from clothing, towels and bedding, except when clothing is worn continuously and washing is rare. When caught from an *animal*, burrows are absent; the eruption may

be limited to the area with which the animal comes in contact; it tends to spontaneous cure. In *Norwegian scabies* there is excessive crusting, with pus and burrows; the face, neck, hands and nails may be infected.

The *diagnosis* is clear when a burrow is found; it forms a sinuous line, often black; at the end is a white speck, the acarus, which may be lifted out with a needle point. When no burrow is seen, the history of itching on going to bed, others of the household similarly affected, and the characteristic sites aid diagnosis. Sensitisation papules may be so extensive over the body, that scabies has been mistaken for gouty eczema by experienced physicians.

Treatment must be thorough, or reinfection persists. The remedy must be rubbed over the whole body. Ung. Sulph. B.P., two-thirds strength, and Ung. Sulph. polysulphidi (B.P.C.) are effective. Stronger applications may cause itching dermatitis which is often mistaken for fresh infection. Soak in a hot bath, rub with coarse flannel and soap to open up the burrows. The ointment is rubbed over the entire body, from the neck down, on three successive days. Two ounces and twenty minutes are required for each application. Then another hot bath and clean underclothing and bedding should complete the cure. The 1942 Advisory Committee on scabies recommended benzyl benzoate emulsion (25 per cent. in lanette wax SX 50 per cent. and water to 100), when it can be applied by a trained person. The patient cannot carry out this procedure alone. After a hot bath and drying, the emulsion is painted on with a flat brush over the whole body from the neck down. Allow to dry; then have clean clothing. Give two such applications on two successive days or within eight days. D.D.T. is also effective. It is now believed that a hot iron provides sufficient disinfection for clothes and bedding.

IV. **Skin Diseases sometimes Papular.**—In PAPULAR ECZEMA the papules rapidly pass on to vesiculation, or are associated with definite patches of eczema. Papules frequently form a stage, generally an early stage, in PSORIASIS, SEBORRHŒIC DERMATITIS, PITYRIASIS RUBRA PILARIS, the EXANTHEMATA and ERYTHEMATA, SYCOSIS, and in XANTHOMA and URTICARIA PIGMENTOSA. Papules of a lichenoid type may follow injections of gold, arsenic and bismuth salts.

§ 622. V. **Milium** is an eruption of small whitish or yellowish pearly pin-point to pinhead sized granules, which affect chiefly the delicate skin under the eyes, the eyelids, cheeks, temples, scrotum, and labia, due to a horny cyst formation in the sebaceous follicles. *Treatment:* make a small incision and squeeze out the contents. Or destroy by electrolysis or diathermy.

VI. **Keratosis Pilaris** (Synonym: Pityriasis Pilaris) affects generally young adults. The orifices of the hair follicles of the thicker portions of the skin—*i.e.*, on the extensor and outer surfaces of the limbs—are occluded with corneous plugs. Hard friction with 2 per cent. salicylic ointment at night, and a rough towel in the morning, generally cure in a few weeks. It is sometimes associated with vitamin A or with thyroid

deficiency. The plugs may be surrounded by a red papule—*lichen pilaris*. In *lichen spinulosus* the plugs stand out like spines; in a child one may see the buttocks covered with fine spines. Sometimes both conditions are associated with lichen planus—*lichen planopilaris*—or other skin disease.

§ 623. VII. **Lichen Planus** (Synonym: Lichen Ruber Planus) is an eruption consisting of flattened, angular, shiny, dull red papules, often presenting a central depression, and a greyish striation on the surface. These tend to coalesce and form irregular patches of a peculiar purplish hue. Occasionally rings are formed (*lichen annularis*). There is no exudation. When the papules disappear much pigmentation may be left and occasionally atrophy (*lichen atrophicus*). The eruption is frequently symmetrical; the characteristic sites are the flexor aspect of the wrists and forearms, and the inner side of the knees. Sometimes the distribution becomes rapidly widespread. Lesions also develop along scratch marks. The mucous membrane of the mouth is often affected and may be so for long before any lesion appears on the skin. Itching may be slight or severe. By the fusion of several papules large plaques may be formed, and when these take on a raised growth, as about the ankles, the condition, which is very intractable, is known as *l. hypertrophicus*, and when warty, *l. verrucosus*. *Lichen planopilaris* shows follicular spines together with ordinary lichen planus lesions. In *lichen spinulosus* horny spines protrude from the follicles. In *lichen nitidus* the papules are very small and pale, and do not itch. Lichen occurs in youth and middle-age, and chiefly in women. The cause is unknown; a virus is suspected. It is aggravated by nervous states, septic foci and intestinal toxæmia. There should be no difficulty in *diagnosing* lichen planus from a *papular syphilide* or *eczema*, on account of its typical position, angular shape, purple colour, and flat waxy surface. The *Prognosis* is good under treatment, although this may have to be extended over many months and even years in some cases. Fresh crops may appear from time to time.

Treatment.—Locally, give soothing lotions, powders and pastes, such as F.36 and 75; and see also § 656. For chronic hypertrophic areas use salicylic acid (gr. 30 to 60 to the ounce of paraff. moll.) or in a paint of equal parts of ether and alcohol, and when dry, cover with plaster. Treat the general health. Give full doses of liq. hyd. perchlor. for two months. Arsenic and mercury can be injected intramuscularly twice a week for eight to twelve weeks. Useful also is X-ray, locally as for psoriasis, and also applied in small doses over the nerve roots at the exits of the brachial and lumbar plexuses.

§ 624. VIII. **Papular Syphilide.**—Syphilitic eruptions are often multiform, but papules generally form the most prominent feature of all syphilitic rashes, especially in the secondary stage. The papule, indeed, forms the prototype of all syphilitic eruptions. These papules are firm, glistening, and project above the surface of the skin with a hard, infiltrated margin, and *vary in size* from a pin's head to a bean. They are of a brownish-red colour (like copper or raw ham) which does not entirely disappear on

pressure. The wide variability in the size of the papules is a feature distinguishing this from other papular diseases. As they increase in size the centres often becomes depressed, or cupped. The distribution is more or less generalised, often symmetrical, but the favourite sites are the forehead, around the mouth, the *flexor aspects* of the arms, and the trunk. When near the corners of the mouth or the anus their surface may be moist, and the *exudation is highly infectious.* Itching is rare—a point of great importance in diagnosis. Other constitutional signs of syphilis may be present. Shotty glands in the groin and neck and elsewhere give valuable aid in the diagnosis of all syphilitic eruptions ; they are present even when no other signs are found, and may last throughout the patient's life. Two *varieties* of papular syphilide are described : *papular syphilide* if the spots are small and numerous, *lenticular syphilide* if large and few. The former is met with more in the early, the latter in the later stages of the disease. Large, moist, flat papules, usually seen near the anus, are called *condylomata.* Rarer forms are the *corymbose syphilide,* in which there are clusters of very small papules surrounding a central larger papule ; and the *follicular syphilide,* which shows small papules resembling lichen pilaris (see also §§ 552 and 645).

Rare Papular Diseases

§ **625.** IX. **Lichen Scrofulosorum** consists of minute yellowish red papules, flat, or conical when around a follicle. Itching is rare. It is seen chiefly on the trunk, sometimes on the limbs, occasionally on the face ; usually before the age of twenty. The disease may last for years, hardly noticed by the patient, and disappear when some tuberculous lesion in the body becomes cured. It is one of the **tuberculides,** a term which denotes certain eruptions which occur in persons suffering from some manifest or latent tuberculous focus and denote sensitisation. Tuberculides are more or less symmetrical, usually livid, firm, involving skin and subcutaneous tissue ; they tend to slow but spontaneous cure. **Acne agminata** is a rare tuberculide, appearing chiefly on the face, as indolent dark red papules which involute with or without pustulation and leave a scar. It is akin to **Folliclis,** which affects the body and arms, and to other papulo-necrotic scarring tuberculides.

X. **Adenoma Sebaceum** consists of numerous small hemispherical elevations, discrete, grouped usually about the middle of the face. In size they vary from a pinhead to a split pea. Their surface is crimson or pinkish yellow, and associated frequently with telangiectases. They have no visible orifice. Some disappear spontaneously, leaving a small scar. The disease is almost always congenital, though it may not be observed till puberty, when it takes on fresh activity. Occasionally it shows nævi similar to those met with in Von Recklinghausen's disease. It has been associated with intellectual inferiority in some cases, when lesions affect the brain (Tuberose Sclerosis, §§ 829, 907*c*). The knife, cautery or electrolysis destroys the skin growths.

XI. **Granuloma Annulare** is occasionally seen in young people and children. especially on the hands. Flattened papules, white or pink, appear in ring-shaped patches, a shilling to half a crown in size, and with a depressed centre. They appear to be associated with latent tuberculous or streptococcal infection. The lesions last long, but may disappear spontaneously or with electrolysis or salicylic acid ointment.

XII. **Trichophytides, Epidermophytides.** With suppurating forms of ringworm and with epidermophytosis certain patients develop an eruption over the trunk and limbs, not unlike Lichen Scrofulosorum, showing red or brown papules, often acuminate,

follicular, with a scale or tiny pustule. The rash is due to sensitisation of the skin to the antigen of the fungi, and to the entry into the circulation of the fungal elements. It vanishes when the original focus is cured. A special vesicular reaction on the hands is described in § 635. Erythematous and other forms of reaction are rare.

XIII. **Keratosis Follicularis** (Synonym: Darier's Disease) is a very rare disease, due to overgrowth and degeneration of cells in the mouths of the pilo-sebaceous follicles. The papules are at first pin-head size, resembling keratosis pilaris. They contain in the centre a horny plug, which is difficult to remove. Some become enlarged and hyperæmic; others become confluent, presenting a papillomatous surface covered by hard yellowish crusts. These may ulcerate, and the area may be covered with a mucopurulent discharge. The disease affects first the face and head, and after the gradual development of years, appears over the sternum, spine, loins, hypogastric and inguinal regions and the extremities, with symmetrical distribution. The *Diagnosis* is difficult, in the early stages, from keratosis pilaris and ichthyosis, and later from acanthosis nigricans. *Treatment* consists in the use of salicylic acid, sulphur, or other keratolytic applications, but the disease is resistant. Cure has followed X-ray, Grenz ray and large doses of Vitamin A.

XIV. **Fox-Fordyce disease** affects a few women, usually after middle age. Close set, tiny papules, red, brownish, or skin coloured, occur in groups in the axillæ, pubis, areolæ of the nipples, umbilicus, less often the sternal region. Itching is severe, X-ray does good, but the disease is obstinate.

(*d*) *Eruptions usually Scaly or Scurfy*

Common.	*Rare.*
I. Psoriasis.	VI. Exfoliative dermatitis.
II. Seborrhœic dermatitis.	VII. Pityriasis rosea.
III. Tinea circinata.	VIII. Pityriasis rubra pilaris.
IV. Squamous syphilide.	IX. Ichthyosis.
V. Skin diseases sometimes scaly at one stage—*e.g.*, eczema, lichen, erythematous diseases.	X. Erythrasma.
	XI. Parapsoriasis.

§ **626.** I. **Psoriasis** is a common disease, occurring as irregular patches slightly raised, covered with copious silvery scales, unattended by any exudation, and situated chiefly on the elbows and knees. The lesion starts as a tiny papule (P. punctata) which from the first has on the top a scale, which, however, may not be visible till scratched. The papule gradually enlarges (P. guttata). In a short time it reaches the size of a coin (P. nummularis). The disease generally then remains stationary for some weeks or months, and may tend to undergo spontaneous involution. The healing process usually starts at or near the centre, without scar or atrophy, and gives to the eruption a circular or serpiginous appearance (P. circinata, P. gyrata). The lesions are usually few and develop slowly; sometimes, especially in young people, many lesions appear rapidly over the body. The rash is scaly and elevated from the first, and always dry, and when the top scales are scratched off, bleeding hyperæmic papillæ are exposed. The distribution is characteristic, affecting the knees and elbows, frequently the scalp, trunk, and other parts of the limbs, especially the extensor aspects, and only rarely the face, palms, or soles. There is little or no itching, unless complicated by streptococcal invasion. Psoriasis of the nails causes pitting, ridging, and elevation of the free border.

Etiology.—The disease is most frequent in early life, though rare under seven, and affects both sexes. There is a hereditary predisposition in some families. The seasonal influence varies; many say that it recurs each winter or spring. It often attacks rheumatic and arthritic subjects, and may alternate with arthritic and other types of rheumatism and gout. In some cases septic foci play a part, perhaps contributory; examine the teeth, tonsils, sinuses and cervix. Psoriasis in children and young adults is often associated with septic tonsils. Acute widespread attacks are often preceded by tonsillitis. *B. coli* infection appears to influence older patients.

Diagnosis.—It is important to distinguish psoriasis from *scaly syphilide.* The syphilide has more infiltration; rarely affects the elbows and knees, and generally prefers the flexor aspects, and palms and soles; the centre of the patches are usually depressed, stained, and healing; the scales are scantier, less silvery, and on being scraped off, do not leave bleeding points. There may be difficulty in diagnosing acute and extensive psoriasis from *seborrhœic dermatitis,* in which the patches are less crimson, the scales scantier, greasier, more orange coloured; they occur chiefly along the middle of the chest, back and front, and if on the limbs usually the flexor aspects. The patches are often surrounded by satellites. The scalp may be affected in both diseases.

Prognosis.—Psoriasis disappears and recurs spontaneously for many years. In severe cases, after over-vigorous treatment, and when complicated by streptococcal infection, it may spread over the whole body and cause exfoliative dermatitis. A streptococcal focus in the body may be the cause of sterile pustules amongst the scaly lesions on the palms and soles (pustular psoriasis).

Treatment.—For acute and widespread cases, order rest in bed, baths and soothing applications, with salicylate of soda internally. For the usual chronic, localised cases, frequent bathing, followed by removal of the scales, is necessary. Chrysarobin gr. 15–60 in one ounce is the time-honoured remedy, but it stains the linen purple and may set up dermatitis; hence it cannot be applied to the face or scalp. Chrysarobin—20 per cent. in chloroform—may be painted on once a week, and covered with collodion. Tar, oil of cade, and salicylic acid, are also efficacious. Scales on the scalp must be removed, then use hyd. ammon, chlor. gr. 20 in one ounce of vaseline. Reports on the results of special diets are conflicting. Treatment directed to the colon sometimes cures; hence probably the success in certain cases of the Danysz entero-vaccines. The removal of septic foci benefits when followed by an autogenous vaccine. For extensive types Goeckerman's method is advised: apply 2 to 4 per cent. crude coal tar, 2 per cent. zinc oxide, 60 per cent. corn starch in paraff. moll. Leave on all night; next day remove with a mineral oil, leaving on a film during exposure to increasing doses of ultra-violet light. Every second day give autohæmotherapy 10 c.c. Treat rheumatism and gout when present. Shock and desensitisation therapy rarely help. Thyroid and pituitary aid

when indicated. Arsenic is valuable in some chronic cases. X-ray may be used for chronic patches, never for extensive areas. It does not prevent recurrence; the after-effects must be remembered.

§ 627. II. **Seborrhœic Dermatitis** (Synonym: Pityriasis Circinata) shows several types of eruption. In young people it usually appears as small, brick-red, soft papules round the pilo-sebaceous follicles. As inflammation extends, these coalesce and form circular, ovoid or gyrate patches with fawn centres, sloping margins and greasy yellow scales. At this stage there is slight serous exudation beneath the scales. This type of seborrhœic dermatitis affects chiefly the scalp, brow (*corona seborrhœica*), and the median line of the face, chest and back, where the sebaceous glands are most active. Sometimes it becomes widespread, with scales so profuse that the condition can be mistaken for acute psoriasis.

In older and less healthy individuals the patches often become eczematised, with itching, serous exudation and crusts. This is usually associated with secondary streptococcal and staphylococcal infection; it has been described as streptococcal dermatitis. The chief sites are the scalp, behind the ears, the axillæ, under the breasts and the groins; this eruption may become widespread.

Etiology.—The name seborrhœic dermatitis is a mistaken one; it used to be thought that the malady affected chiefly those with oily skin. Sabouraud found that the disease originated with infection by the pityrosporon of Molassez, see § 655. III. Staphylococci and streptococci are found with the extensive reaction associated with constitutional factors.

Diagnosis.—The scales of psoriasis are more silvery, and on removing them hyperæmic bleeding papillæ are seen; in extensive seborrhœic dermatitis minute points of oozing serum are seen on removing the scales.

The *Treatment* must be directed to the scalp as well as the body; or the disease will recur. Frequent washing is necessary. A pomade of hyd. ox. rub. (gr. 4 to the ounce) should be rubbed in twice a week, or a lotion of hyd. perchlor. (gr. 1 to the ounce of spirit and water, equal parts). On the smooth skin ichthyol or sulphur are better. A good prescription is sulph. præcip. gr. 4., acid carbol. ♏2, to the ounce of vaseline. When the malady tends to recur, treat the colon, give much fruit and vegetable in the diet, and restrict fats and carbohydrates. Alkalies aid many of these cases.

Dry Impetigo occurs chiefly in children with fine skins; it affects the face, less often the neck, "*dartres volantes.*" White or slightly pink patches with delicate scales appear in circular or ovoid shapes. They may become eczematised. Sabouraud classed it as a dry impetigo due to streptococci; it is often associated with streptococcal fissures of the nose or mouth. A rare eczematoid tuberculide resembles it, but more often attacks the trunk and limbs. Resorcin 2 per cent. is usually curative.

III. **Tinea Circinata** may appear as small red patches, of an oval or ringed shape, slightly scaly. When the head is affected with the small-spored ringworm, these patches may often be seen on brow, neck, and shoulders. The large-spored fungus usually causes a ringed scaly eruption;

see § 636. Epidermophyton infections have become very common in recent years. *Tinea cruris* (dhobie itch), due to the *Epidermophyton inguinale*, grows in the horny layer of the skin, forming very irritable red patches which join and may extend over a wide area, with a polycyclical margin, scaly or even vesicular, on the genitals, perineum, natal cleft, sometimes the thighs, axillæ, and under the breasts. The epidermophyton also causes several varieties of infection of the feet and hands: (1) the so-called interdigital eczema between the toes, especially the fourth and fifth, is often seen. Below the sodden white skin fissures may develop, leading to recurrent attacks of streptococcal lymphangitis spreading up the leg; (2) deep-set vesicles on the soles and palms, resembling cheiro-pompholyx; (3) hyperkeratosis of the soles and palms, especially of the heels; and (4) scaly patches on the feet and hands.

Treatment.—The fungus is attacked by liq. iod. fort. 1 in 10 of alcohol, by carbol fuchsin paint (10 per cent.), and by Whitfield's ointment, salicylic and benzoic acid āā 3 to 6 per cent. in vaseline. Other fungicides are on trial, *e.g.*, undecylenic acid or phenylmercuric nitrate in ointment and powder. The marginal vesicle tops and scales should be rubbed off with 3 per cent. argent. nit. The sodden skin between the toes should be softened with salicylic acid ointment, then removed with forceps and destroyed; the parts must be kept dry, with wool or muslin and a powder of talc, with salicylic acid of strength varying according to the thickness of skin to be detached. Recurrence is common: boil the infected underlinen or stockings, or soak for an hour in 1 per cent. thymol in spirit. Boots and shoes are disinfected with a 2 per cent. formalin swab or are placed at night in a closed tin containing a receptacle with two ounces of pure formalin.

Tinea Imbricata occurs in the tropics. The fungus causes a characteristic scaly eruption with a watered-silk appearance.

§ **628.** IV. **Squamous Syphilide.**—The squamous syphilide occurs as a later stage of the papular or the tubercular syphilitic eruptions (*q.v.*). The scales are thin, scanty, and greyish, lying upon patches of stained and infiltrated skin (*i.e.*, the syphilitic papules) which are deep brown or copper coloured, usually round, or in *segments of circles*, having raised serpiginous scaly borders. It may occur on any part of the body, but the flexor aspects and the palms or soles are particularly characteristic situations, the converse of *psoriasis*. A scaly syphilide of the palms is diagnosed from dry *eczema* by its raised serpiginous border, with sometimes an area of normal, atrophied, or pigmented skin in its centre.

V. **Skin Diseases Scaly at one Stage.**—A *scaly* or scurfy condition of the skin, especially of the face, is produced by hard water and exposure, in certain states of ill-health, after scarlet fever, measles, and other *eruptive fevers*. In *eczema*, scales and crusts form, but the presence of exudation is its differentiating feature. Most *erythematous* lesions develop some degree of scaling. In several varieties of *lichen*, a thin silvery scale is found, but lichen is chiefly a papular eruption. *Lupus erythematosus* has adherent scales and crusts.

§ 629. VI. **Exfoliative Dermatitis.**—The term Exfoliative Dermatitis implies any chronic or sub-acute generalised inflammatory disease of the skin, whether primary or supervening upon other cutaneous disturbance of long standing, which is characterised by vivid hyperæmia of the entire surface, and *abundant and repeated exfoliation,* accompanied usually by shedding of the hair and nails. There is usually associated constitutional disturbance, and the itching may be severe.

Etiology.—Occasionally, as a *secondary* affection, it may follow psoriasis, leukæmia, lichen planus, mycosis fungoides, pityriasis rubra pilaris, pemphigus foliaceus, and arsenic, gold or bismuth medication. As a *primary* condition the disease is serious. It starts in several ways; a rapidly spreading hyperæmia of the skin is common to all. Varieties which have been described as separate diseases only differ in their mode of onset and etiology.

An epidemic exfoliative dermatitis (Synonym: Savill's disease) was observed in 1891. One hundred and sixty-three cases occurred among the patients in the Paddington Infirmary, with a case mortality of 12·5 per cent. It was traced to milk treated with a formalin preservative.

Treatment.—Rest in bed with local soothing applications or inert powders is essential. Care for the general health, and eradicate septic foci. Quinine, ox-gall and hexamine have cured some cases; autohæmotherapy and colonic lavage have cured others. Of soothing creams, zinc in olive or castor oil, glycerin amyli, subacetate of lead in glycerin, are all of value. Cod-liver oil in paraff. liq. and vaseline has advocates. For the exfoliative dermatitis of infants (see Ritter's disease, below) attack the organism with 5 per cent. sulphathiazole ointment, or pigment. tinctorium (N.F.). Some infants have responded to vitamin B complex together with riboflavin 2 mg. t.d.s.; some to sulphonamide therapy.

Ritter's disease is probably the exfoliative stage of epidemic pemphigus neonatorum, such as occurs in Institutions and used always to be fatal.

§ 630. VII. **Pityriasis Rosea** consists of numerous pink patches, slightly raised and pea-sized, and oval-shaped rings, with slight scaling on the pink margins, and a fawn-coloured centre. A "herald patch" usually appears on the trunk some days or weeks before the generalised eruption, which comes out in successive crops, starting usually on the sides of the trunk, spreading to the neck, upper arms and thighs, rarely to the face. Itching may be absent or severe. Pityriasis rosea runs a course of a few weeks to a few months, and disappears spontaneously. The disease occurs in both sexes, and at any age, but is most frequent in young adults. Many cases occur in early spring. Its importance lies in the fact that it may be mistaken for *syphilitic roseola,* which has a darker colour (§ 610). *Seborrhœic dermatitis* has greasy scales and appears on different sites. *Tinea circinata* is rarely so widespread, and the fungus can be found. *Psoriasis* has more infiltration and silvery scales.

Treatment.—Baths with weak Condy's fluid do good. One per cent. cignolin in vaseline is curative. Use soothing lotions when itching is marked.

§ 631. VIII. **Pityriasis Rubra Pilaris** (Devergie), Lichen Acuminatus or Lichen Ruber (Hebra). The eruption commences as tiny hard papules of hyperkeratosis involving the hair follicles; gradually these become fused together into one reddened patch which exfoliates. The distribution is symmetrical and starts where the lanugo hairs are found—on the backs of the hands and forearms; thus it often presents a glove-like distribution on the upper and lower extremities. The scalp has thick scales; the hair does not fall until the secondary stage of erythrodermia sets in. The disease may spread over the whole body. The progressive margin is always marked by the same tiny scale-capped papules. General erythrodermia with pyrexia may appear at intervals, then disappear, except from such regions as the face, palms and soles, which usually remain red, tense and scaly. Ectropion is common, due to the stretched condition of the cheeks.

Diagnosis.—The disease is differentiated from *psoriasis* by its distribution and by the characteristic marginal papules with a central hair, but is indistinguishable from *dermatitis exfoliativa* over the whole body. In the earlier stages dermatitis

exfoliativa does not present the small acuminate papules which constitute the elementary lesion of P. rubra pilaris.

The *Causes* are obscure; the disease usually occurs before the age of twenty-one. Recent evidence points to deficiency of Vitamin A. The disease lasts, with remissions, for months or years, but recovery is usual.

Treatment is symptomatic. Give abundant food. Thyroid benefits mild cases; in chronic cases try arsenic. Cures have been reported with gold injections, shock therapy and vitamin A in large doses. Locally, apply vaseline and lanolin, equal parts, with salicylic acid varied in strength according to the degree of thickening.

§ **632.** IX. **Ichthyosis** (Synonym: Xeroderma) is a congenital condition of the skin, characterised by dryness and scaliness of the epidermis, and in some cases by wart-like outgrowths. It is sometimes not diagnosed till the child is some years old.

There are three *clinical types* or degrees of the affection. In the first or mild type (*Xeroderma*) there is simply an undue harshness or roughness of the skin, and consequently throughout life a tendency to the supervention of "chaps," eczema, and other skin affections. It is more marked on the extensor aspects. In a second type (*I. vera*) the superficial layers of the epidermis are thickened, and appear stretched; the hardened cuticle presents fissures and cracks which, bounding polygonal areas, give the patient the appearance of having a fish or crocodile skin. The everted eyelids and nostrils, the atrophied hair and nails, and the hardened, scale-like condition of the skin are characteristic. The third variety is described in § 650. IV.

The *Diagnosis* is not difficult, owing to the congenital nature of the malady.

Prognosis.—Apart from the inconvenience and the liability to eczema, the first type is not serious. In the second type the disease progresses to the age of puberty, and then remains stationary.

Treatment.—No remedy influences the severe forms of this disease. Resorcin 2 per cent. in glycerin. amyli, vasolanolin (vaseline and lanolin āā), baths with superfatted soap followed by inunction with paraff. moll. alb. soften the skin. Thyroid often controls mild cases and sun and ultra-violet light are beneficial.

§ **633.** X. **Erythrasma** consists of defined scaly discs with a serpiginous border, pale red, yellow, or dark brown in colour. The scales can be scraped off, and contain a fungus, the *Microsporon Minutissimum*. The patches are extremely chronic, and are found on the opposed surfaces of the scrotum, groins, and adjacent surface of the thighs, axillæ, and mammæ. They itch when perspiration is excessive. Treat as for pityriasis versicolor (§ 652).

XI. **Parapsoriasis** consists of brownish red patches, with slight or no infiltration, and fine scales; in one form delicate papules occur. It may be localised or widespread. Itching is usually absent. The *cause* is unknown, and it is resistant to treatment. Xantho-erythrodermia Perstans is one form with little scaling.

GROUP II. VESICULAR AND BULLOUS ERUPTIONS

Eruptions in which the elements are usually vesicular and the exudation serous, are commonly classed into those with small vesicles, and those with vesicles of larger size, bullæ.

I. Eczema.
II. Cheiro-pompholyx.
III. Streptococcal skin infections.
IV. Herpes.
V. Varicella.
VI. Scabies.
VII. Tinea circinata (sometimes).
VIII. Sudamina.
IX. Hydrocystoma.

X. Dermatitis herpetiformis.
XI. Pemphigus.
XII. Epidermolysis Bullosa.

XIII. Hydroa Aestivale.
XIV. Lymphangioma circumscriptum.
XV. Anthrax.
XVI. Pustular and other diseases in which vesicles and bullæ may occur at some stage.

Note.—Syphilides are practically never vesicular.

§ **634. I. Eczema** is a catarrhal inflammation of the skin, running sometimes an acute, sometimes a chronic course. There has been much discussion as to the differentiation of eczema and dermatitis ("contact" dermatitis). Some held that eczema had its origin in causes arising from within the body, whereas dermatitis was due to external irritants. The same inflammatory reaction of the skin occurs, and the clinical problem is to track down the cause in the individual case.

Eczema is chiefly a vesicular condition, but at different stages of its course it may show most of the primary and secondary lesions of the skin. In acute *erythematous eczema* there is swelling and redness, and the palpating finger feels a roughness which is not present in simple erythemas. This stage may subside, leaving fine desquamation till the skin heals, or it may pass on to form small *papules* or *vesicles*, or these may arise in groups without preliminary erythema. At a still later stage the vesicles rupture and serum exudes, sometimes drying to form yellow crusts, sometimes flowing profusely, the so-called "weeping eczema." In the subacute stage crusts, scales and excoriations due to scratching are seen on a slightly swollen base. *Pustules* indicate secondary infections. In chronic stages there is no exudation, but the horny layer flakes off in fine or large scales, whilst in some cases there remains so much thickening of the skin that the part is described as "lichenified." Eczema occurs on any part of the body, and is accompanied by throbbing and burning, or with marked itching, in proportion to the degree of inflammation present.

The *Diagnosis* of eczema or dermatitis is simple; the diagnosis of the cause is difficult. *Seborrhœic dermatitis* is covered by greasy yellow scales. *Syphilides* never resemble acute or subacute eczema, or, indeed, any vesicular disease, a fact of considerable value in diagnosis. It is difficult sometimes to distinguish patches of dry chronic eczema from *psoriasis*, but the latter affects the extensor aspects, and is covered with silvery white scales. *Paget's disease* has a defined margin and hard infiltration.

Varieties of eczema have been named according to the position of the lesions: eczema palmaris, ani or vulvæ, etc., or according to the predominant lesion: erythematous, papular, vesicular, squamous, rubrum (raw), or rimosum (fissured). When the skin becomes sensitised to staphylococci the resulting dermatitis shows large vesicles or vesicopustules with yellow crusts; the condition is known as *dermatitis infectiosa eczematoides*. This form frequently occurs in the vicinity of an infective discharge, from the ear, nose, vagina, anus, a sinus or wound; in underfed or unhealthy subjects, with lowered resistance to the infecting organisms, it may last, with remissions, for years. When streptococci invade an eczematous region there is often an acute exacerbation; the area becomes swollen, red, oozes profusely, and fissures form in any adjacent folds. This variety (*intertrigo*) is often seen in the groins, the intergluteal fold, under the breasts, and spreading from the margin of the scalp behind the ears, at the corners of the eyes, nose and mouth (*perlèche*). Eczema of the face in *infancy* may be followed in later life by eczema of the flexures

—Besnier's prurigo or atopic eczema. An acutely spreading intertrigo may also be due to infection with *monilia*; the findings of a culture or, in the scrapings, masses of small spores with a little mycelium settle the diagnosis. *Eczema Marginatum* is really a tinea infection of the groins and genitals (§ 627).

Etiology.—Eczema is a reaction of the skin, either to an external irritant or to some internal poison which is probably conveyed by the circulation to the skin, where it meets the antibody. Yet there would appear to be a third causal factor, because not only may certain external or internal causes be present in average people, without producing eczema, but even in those who are subject to eczema, the presence of the usual excitants does not always lead to the development of the skin lesions. The third factor seems to be a hypersensitiveness due to changes in the skin itself or in some cases to a combination of several toxins in operation at the same time. The eczematous eruption apparently develops when there is sensitisation of the skin cells and also of its blood vessels. Thus, for example, the eczema noticed in certain *trades*, such as the eczema of bakers, grocers, and workers in oil, tar, teak and other woods, may not develop until some gastro-intestinal abnormality lowers the resistance. At the same time the use of strong alkalies for cleansing the skin, thus removing the natural protective oil, may play its part in precipitating the attack. Important amongst the *external causes* of eczema are chemicals, such as nickel, chromium and the metol of photographers, cement and lime used in building, the paraphenyldiamine group of dyes used by workers in furs and hair dyes, drugs used in toilet preparations for the teeth, mouth, nails and lips. Alkalies in washing soda, soaps and other cleansing materials; sulphur, lysol, formalin, and other antiseptics used by medical men and nurses; varnish and furniture polish, lotions used against perspiration, depilatories; certain plants and oils; fat solvents such as petrol, benzine, mineral oils, turpentine, mustard plaster and gas, can all give rise to eczema. Many other drugs applied externally can cause dermatitis: *e.g.*, arnica, arsenic, capsicum, carbolic acid, chrysarobin, croton oil, iodine, tar, soot, procaine, picric acid, mercurial salts, sulphur, etc. Other external causes are extremes of heat and cold, sunlight (light sensitisation), wind, friction, pressure. Eczema is apt to affect the dry skin of the aged and those with even mild forms of ichthyosis. Infection of the skin with *staphylococcus aureus* and with the *streptococcus* may cause eczema. The staphylococcus causes a dermatitis near a discharge (see Varieties of Eczema, above). The eczema due to streptococcal infection is usually an acute process; fissures are frequent in this form. The " eczema rubrum " which affects the legs, especially in old people, is often due to streptococcal infection.

Internal Causes.—More than one cause may be in operation, and in some cases the development of an internal cause may make a previously harmless external factor act as an irritant to the skin. Hence the legal difficulty with industrial dermatitis and compensation. Dyspepsia, con-

stipation, too little or too much exercise or food, all play a causal part. Eczema may occur with diabetes, gout, inefficient gastro-intestinal digestion, defective detoxicating function of the liver; it may accompany albuminuria and kidney disease; appear with every pregnancy and after lactation. A septic focus may cause eczema, just as it may precipitate diabetes or urticaria in other individuals. The dermatitis caused by exposure to sunlight is usually associated with abnormal intestinal flora, vitamin and hepatic deficiency, sometimes also infective foci. Mental states such as grief, worry or over-strain always reduce the power of resistance of the body. Sometimes eczema is an allergic manifestation, hay fever and asthma alternating with it. It then usually occurs in several members of a family—the "asthma, eczema, prurigo complex." In such cases there may be a history of infantile eczema of the face. This type is often due to sensitiveness to a protein found in oatmeal, wheat, milk, beef, pork, oranges or other foods.

The *Treatment* of eczema differs according to the stage of the disease. First of all, the cause, internal or external, must be patiently sought for and, if possible, removed. In an obscure case the *patch test* (§ 605) enables us to discover the cause. The principles of local treatment are those underlying the treatment of all skin diseases (see § 656). Washing is forbidden in the acute stages, especially the use of soap of any kind; but soaking in warm normal saline is refreshing. In chronic cases, baths (F. 1 and 2) are soothing, and thorough washing is permitted for dry, thickened patches. For acute erythematous eczema, give powders of talc and zinc oxide, and lotions of calamine, zinc and lead (F. 36 and 42). For vesicular and for weeping eczema, give lotions and pastes which absorb the exudation. Pastes are spread on gauze or butter muslin, and kept in contact. Ointments at this stage would confine the exudation beneath the oily basis. An excellent paste is Lassar's paste (F. 75). In the early stage a saline aperient with vin. antimonialis ♏10 t.i.d. is a time-honoured remedy, and may cut short an acute outbreak. In subacute eczema add a little mercury and weak tar to the soothing paste or lotion. For chronic eczema give ointments containing a higher proportion of the stimulating remedies, mercury, tar and salicylic acid, in vaseline, and rub the ointment in thoroughly. The strength can be gradually increased; it is well to go cautiously, because the eczema can be aggravated by the use of too strong remedies. For chronic, infiltrated patches I find it useful to paint with pure alcohol and seal up the part with Unna's gelatine. For lichenified patches strong salicylic and phenol (āā gr. 20 in one ounce) is useful; in such cases X-ray in small doses, at weekly or fortnightly intervals, is often the quickest method of cure.

As regards diet, the individual case must be carefully considered. In the average simple case mild aperients and restriction of strong tea, coffee, sugar and alcohol bring about rapid improvement. For acute widespread cases Duncan Bulkley's method is good: he gave for five days a strict diet, with only boiled rice, cream or butter, water, and salt and pepper

to taste. Elimination diet tables should be consulted in chronic cases. In recurring types, with defective intestinal digestion and marked indicanuria, the method which aids many sufferers is one with lacto-vegetarian menus, glucose or lactose, raw oatmeal with B. acidophilus milk, and sometimes hydrochloric acid and pancreatic extract (see § 656). This method of diet and medication is especially useful in the eczema complicating Besnier's prurigo ; it often also aids eczema due to light sensitisation. In other cases restricting fluid to two pints a day, and reducing the carbohydrate intake is effective. High colonic irrigation may be necessary in cases with a loaded cæcum, or colonic spasm and tenderness over the region of the sigmoid. In some patients vaccines made from the stool organisms may determine a cure after all else has failed. Calcium salts, Vitamin B and dilute hydrochloric acid aid eczema with much swelling and oozing. Desensitisation methods, especially autohæmotherapy, succeed with allergic cases (§§ 609 and 656).

Treatment of Varieties.—For *streptococcal* eczema Sabouraud advised painting twice a week with silver nitrate (gr. 4 in spt. aeth. nit. fl. oz. 1) or 1 per cent. iodine in 90 per cent. alcohol. Some prefer 2 per cent. aqueous gentian violet, and later a paste of ichthyol 2, zinc oxide 6, hydrous lanolin 4, and vaseline 10 parts. When there is profuse oozing a simple calamine lotion is more comforting. When eczema becomes *pustular* from infection by a discharge, etc., mercury in paste or lotion is indicated, never tar. In both these types of eczema, ultra-violet light is beneficial, in local as well as general doses. Use penicillin when the organism is penicillin sensitive. Autogenous vaccine may be required and every possible measure for building up the health.

Eczema of special regions : behind the ears, with fissures, see streptococcal eczema (above). Eczema of the vulva and anus may be obstinate. To prevent relapses the cause must be removed. The chief causes are : discharge from the cervix or vagina, *B. coli* or sugar in the urine, worms (especially in anal cases), diarrhœa, colitis, leakage of paraffin, frequent use of irritating suppositories, pessaries or douches, piles, fungus or monilia infections. When perianal eczema is an allergic manifestation treat with antihistamine drugs and remove the allergen (§ 609). Ensure sleep without barbiturates. In all cases see that scratching or friction is prevented. Enjoin cleanliness, and protection before passage of excreta. Use a soothing calamine cream or paste at night, and a dusting powder by day. For infiltrated cases give U.V. light and fractional doses of X-ray. Many of the local anæsthetics aid, but must be *used only to tide over crises*—*e.g.*, nestosyl, anethane, benzocaine and percainal. Menthol 1 per cent., phenol 1 in 60, liq. carb. deterg. 1 in 20, or cyllin m. 30 in one pint is a useful lotion.

§ 635. II. **Cheiro-pompholyx** is a deep-seated vesicular and bullous eruption affecting the hands *symmetrically*, and sometimes the feet. Vesicles appear in the clefts between the fingers and toes, like boiled sago grains, and creep on to the palmar and dorsal surfaces—an important diagnostic feature from a scaly syphilide of the palms. Some of the vesicles coalesce into bullæ; these rarely rupture, but their contents become absorbed, and exfoliation occurs. It often occurs in hot weather and was believed to be due to sweat irritation. Recurrences are usual. Use soothing lotions, pastes and powders, and attend to the general health. A similar eruption is an allergic reaction to epidermophytosis, and vanishes when that is cured. Monilial infection of the vagina has caused the same rash on the hands.

III. **Streptococcal** skin infections cause vesicular eruptions, which may be mistaken for eczema or herpes on the one hand, and pemphigus or urticaria bullosa on the other. They also may cause intertrigo and fissures (see varieties of eczema). The bullæ are situated on an inflamed base; streptococci are found in the bullæ. *Impetigo Contagiosa* has vesicles which soon crust over; and in severe cases, bullæ (§ 640). The so-called **pemphigus neonatorum** is due to an infection soon after birth. Streptococci have been found in the bullæ in many epidemics; recent work incriminates the staphylococcus aureus.

IV. Herpes Zoster consists of one or more *clusters of vesicles* on a crimson base, associated with neuralgic pain. It commences with a red patch or a group of flat papules, on which vesicles rapidly appear, larger than those of eczema, round, hemispherical, and uniform in size; and as there is no tendency to spontaneous rupture there is usually no oozing. They smart or burn, rather than itch. The vesicles contain clear serum, and after a few days, dry up and form little crusts which fall off, often leaving a scar. See § 826.

Herpes simplex (Syn.: Herpes febrilis) is entirely distinct from Herpes Zoster. It shows a small group of tiny vesicles, preceded by a burning sensation, which affects the face, chiefly the nose, lips, mouth, cheeks, and less often the genitals.

Etiology.—Herpes zoster is dealt with in § 826. It has been noted to precede or follow varicella in contact cases. There is considerable evidence that some cases of herpes zoster are caused by the same virus as varicella (§ 476); but an individual who has had varicella is not protected against herpes zoster. *Herpes simplex* may be primary, or may accompany a "cold," pneumonia or other respiratory disease, certain fevers, such as malaria and cerebrospinal meningitis, and occasionally it occurs with drugs (arsenic, bismuth) such as may produce H. zoster. It may follow an alcohol injection of the Gasserian ganglion. It is due to a filterable virus. Subjects of recurrent herpes are permanent carriers of the virus; their serum contains the specific antibody.

Diagnosis.—Herpes is distinguished from all other vesicular conditions by its distribution, its limited duration, and the occurrence of the vesicles in *clusters*, on an erythematous base.

Prognosis.—Herpes simplex tends to spontaneous recovery in the course of a week. It is apt to recur, sometimes at periodic intervals.

Treatment of *herpes simplex.*—Protect the vesicles by starch or zinc powder, or paint with collodion, or use some soothing ointment. The vesicles may be aborted with aspirin by mouth, and by frequent applications of surgical spirit or boracic powder. Remove the contributory cause in recurrent cases; sometimes this is due to a chill and a septic focus in the teeth, tonsils, nose or colon. A vaccine of the virus and intradermal small-pox vaccination have given good results.

V. Varicella is described in §§ 476 and 479. The points of distinction between varicella and small-pox are conveniently tabulated thus:—

Varicella.	*Small-pox.*
No symptoms before rash.	Three days before rash, sudden onset of illness with backache.
Soft pink papules becoming vesicular.	Shotty papules becoming vesicular or pustular.
Chest, neck, and trunk, fewer on face and limbs. Rash is centripetal.	First on face and wrists; more on limbs than on trunk. Rash centrifugal.
Successive crops, and thus find small papules beside vesicles of various sizes.	**All one stage** (papular or vesicular, or pustular) at one place.

VI. Scabies is chiefly a papular eruption (§ 621); but in children the vesicular element often predominates; it may then be mistaken for eczema or varicella. The burrows, the itching, worse at night, and the position of the lesions, aid the diagnosis.

§ **636.** VII. **Tinea Circinata,** or ringworm of the body, is vesicular; the fungus is usually of animal origin; the arrangement of the vesicles in the form of a definite ring is so characteristic as to be unmistakable (Fig. 147). When originating from the horse or cattle, there may be suppuration. When associated with scalp infection the lesions are usually dis-



Fig. 147.—Hand of a woman suffering from Tinea Circinata, in which the vesicular element is unusually prominent. A large ring of vesicles encloses a scaly area. Verified by microscopic examination.

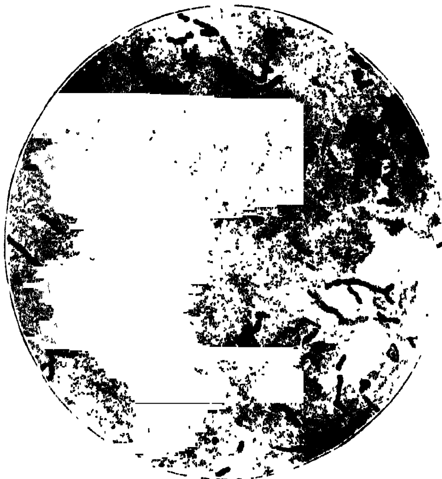


Fig. 148.—Mycelium of Tinea Circinata (ringworm of the body).—A scraping from the skin, stained by Gram's method. Mycelium of equal segments having truncated ends bifurcating in places; spores almost absent. Compare figures in § 655.

coid or annular, with a slightly raised scaly margin, and with minute papules or vesicles. The favourite localities are the face, neck, and hands. The Epidermophyton infections are described in § 627. Under the microscope,

the mycelium (Fig. 148), and perhaps a few spores, can be seen on scraping under the roof of the vesicles or the scales from the margin.

Treatment.—Rub thoroughly with ung. hyd. amm. chlor., or Whitfield's combination of salicylic and benzoic acid, iodine (1 per cent. in 90 per cent. alcohol), ung. iodi. denigrescens or the new fungicide, undecylenic acid.

§ **637.** VIII. **Sudamina** are clear, scattered, non-inflammatory vesicles, about the size of a pin's head, occurring chiefly on the trunk, in conditions of violent exercise, high temperature and fevers such as acute rheumatism, which are attended by profuse perspiration. They disappear in a few days. They are a non-inflammatory disorder of the sweat glands, whereas in *miliaria* (commonly called prickly heat) a mild inflammatory condition at the mouths of these glands causes similar papules and vesicles on a red base. *Treat* with dusting powder.

IX. **Hydrocystoma** shows deep-seated, tense, translucent vesicles on the face, pin-head to a pea in size, lasting for months. They are formed by a cystic swelling of the duct of the sweat-gland, and never become purulent. They disappear spontaneously, chiefly in cold weather. They chiefly affect middle-aged women whose lives are spent in a warm, moist atmosphere, or who perspire much, especially when warm weather sets in. *Treatment.*—Puncture the vesicles, and use dusting powder.

§ **638.** X. **Dermatitis Herpetiformis** (Synonym: Duhring's disease) may be defined as a relapsing disorder of prolonged duration, characterised by the appearance of successive crops of erythematous or papular elements, always in clusters, which usually go on to the formation of vesicles, pustules, or bullæ, are always attended by intense irritation, and sometimes by pigmentation. Different varieties are described according to the element which predominates. Sometimes this consists of circumscribed patches of bright red erythematous or semi-urticarial inflammation, which spread by raised edges, and leave a pigmented centre. In the usual *papulo-vesicular* variety vesicles predominate, varying in size from a pin's head to a split pea, and many become large bullæ. The fluid in the bullæ and also the blood contain eosinophil cells in great excess. Sometimes papules predominate, and become scratched and covered with blood-stained scabs. Intense itching accompanies all varieties. Scars and temporary pigmentation may ensue. In many cases the general health seems undisturbed, but often the appearance of each crop is attended by pyrexia, and occasionally gastro-intestinal disturbance. The distribution tends to be symmetrical, and to favour the flexor surface of the wrists, the axillæ, groins, abdomen, sacral region, and buttocks. The head, face, palms and soles may escape. The mucous membrane of the mouth and pharynx may also be involved. Each successive crop lasts from one to four weeks; between the attacks are longer or shorter intervals of comparative freedom. The disease may last months or years. *Herpes gestationis* appears to be the same malady occurring during pregnancy; the outlook is serious for both mother and child.

Diagnosis.—The disease differs from pemphigus vulgaris in the following respects: (1) The smaller size of the vesicles or bullæ, which are (2) constantly arranged in clusters; (3) the presence of erythematous patches beneath the vesicles and elsewhere, and the multiform character of the eruption; and (4) itching is usually severe. From *eczema*, *urticaria bullosa*, and *erythema multiforme* the disease is distinguished by consideration of the above features.

Etiology.—The disorder is more common in men, and between the ages of sixteen and thirty. It is supposed to be due to a virus.

Treatment.—Arsenic is of great service; it should be given in gradually increasing doses, up to the physiological limit. Andrews advises sulphapyridine G. 0·5 q.i.d. with sod. bicarb. and much fluid. Forms of "shock" treatment and autoserum injections (§ 656) have aided many cases for a time. Locally, sedative lotions, pastes, or spirit and powders may be prescribed. Duhring and H. Montgomery advise sulphur 30 grains to the ounce of paraff. moll. Lumbar puncture sometimes relieves the itching.

§ 639. XI. **Pemphigus** is one of the rarer diseases of the skin, characterised by the presence of bullæ and constitutional symptoms. *P. chronicus* or *vulgaris* is the more common and typical variety in which the bullæ develop in crops, each bulla varying in size from a pea to a hen's egg, being tense with clear albuminous serum, which becomes turbid, purulent, and occasionally hæmorrhagic. The bulla is characterised by having at first no ring of erythema round its base. The fluid is either absorbed with formation of crusts, or the blebs burst, leaving a raw surface on which new epidermis soon develops. Any part of the skin may be affected, as well as the mucous membrane of the mouth, nose, throat, vulva and conjunctivæ. Each bulla lasts a few days; fresh crops may continue to come out for months. The constitutional disturbance depends largely upon the number of bullæ and the frequency of the crops. The outlook is grave; even after long intervals of comparative freedom the end is usually fatal. In *P. foliaceus* the bullæ are very thin and flaccid, and rupture early; but the epidermis, instead of re-forming, continues to peel off until large areas of red, raw, exuding surface are exposed, with the epidermis folded at the margins —a point which distinguishes it from eczema rubrum. This process slowly extends for a year or two until the whole body may be involved, with a fatal issue. *P. vegetans* develops papillomatous vegetations. Often starting in the mouth, the disease spreads; the bullæ remain long unhealed, and in moist positions such as the groins and axillæ vegetations develop with offensive discharge. Prostration and death usually occur in a few months.

Etiology.—The cause is unknown; a virus is suspected. *Pemphigus neonatorum* has long been supposed to be due to streptococcal infection, but recent evidence incriminates *Staphylococcus aureus*.

The *diagnosis* is clear in straightforward types of the disease; other bullous diseases and drug rashes must be passed in review. In children, *bullous impetigo* affects chiefly the face and has characteristic crusts; the *bullous syphilide* is seen in infants chiefly on the palms and soles. *Treatment* is palliative. Arsenic, in various forms, has been of great service. Give nutritious diet; add salt when there is much oozing. Local remedies: sulphathiazole 5 per cent. in glycerin, or penicillin (400 units/gramme), in wax or eucerin, are helpful. Dusting powders are more comforting than ointments. Continuous baths may be required.

XII. **Epidermolysis Bullosa** is a rare congenital disease in which slight traumatism causes the formation of bullæ. It usually runs in families. The bullæ appear on parts exposed to friction or pressure, and even the nails and scalp may be affected.

XIII. **Hydroa Aestivale** is a papulo-vesicular or bullous eruption occurring on parts exposed to light, and appears chiefly in summer in successive crops, continuing for a few weeks. The lesions are preceded by a burning sensation; they last a few days, then crusts form, and scars frequently follow. In the juvenile type it is often associated with hæmatoporphyrinuria and is due to an inborn error of metabolism. The adult type is mentioned under Varieties of Eczema (light sensitisation).

XIV. **Lymphangioma circumscriptum** is a rare nævus formed of overgrowth of lymph vessels, and shows deep-seated, thick-walled yellow vesicles.

XV. ANTHRAX begins as a papule. A ring of vesicles forms, and on an inflamed base a central gangrenous scab develops. Constitutional symptoms are present (§ 490).

XVI. Other diseases occasionally showing vesicles or bullæ—erythema multiforme, urticaria, congenital syphilis, drug eruptions, acne varioliformis, lichen planus, leprosy, etc.—are described under their respective headings. Stings and bites of insects may cause small or large vesicles, with a central punctum which aids diagnosis in obscure cases. The external application of strong acids, liquor epispasticus, or overdoses of ultra-violet light may be unsuspected causes of bullæ.

GROUP III. PUSTULAR ERUPTIONS

Eruptions in which the elements are mainly pustular—

(*a*) *Superficial Pustules.*

I. Impetigo contagiosa.
II. Ecthyma.
III. Impetigo Herpetiformis.
IV. Cutaneous Diphtheria.

(*b*) *Pustules on an Indurated Base.*

V. Sycosis.
VI. Pustular syphilide.
VII. Pustular acne.
VIII. Pustular folliculitis (some cases).
IX. Bromide and other drug eruptions.
X. Variola.
XI. Acute glanders.
XII. Pustular tuberculide.
XIII. Dermatitis vegetans.

(*c*) *Furuncular eruptions with a Slough.*

XIV. Boils.
XV. Carbuncles.
XVI. Kerion.
XVII. Anthrax (later stages § 490).

Eczema and all the diseases mentioned in Group II may become pustular, owing to secondary infection by pyogenic cocci. Conversely, nearly all the pustular diseases just mentioned may start as vesicles. Most pustular conditions of the skin are due to invasion by various strains of staphylococci. Other organisms may cause suppuration in conditions favourable to their growth, such as tuberculosis, syphilis, fungi and yeasts. The staphylococcus albus is found in superficial and less severe lesions, the staphylococcus aureus in deep-seated severe infection of the follicles.

(*a*) *Superficial Pustules*

§ **640.** I. **Impetigo Contagiosa** is a disease frequently met with on the faces of children, and is so called because it is readily conveyed from one child to another. At first the lesions are vesicular, but they become pustular in a few hours. The pustules vary in size, and are discrete, but adjacent lesions run together. In the course of a few days they dry into yellow crusts, which, falling off, leave a flat congested mark covered by new cuticle. They do not leave scars unless scratched. The favourite positions are the face, especially round the mouth, scalp, and hands of children, but they may occur on any part of the body. If untreated, fresh pustules appear in other places for a week or two; or the disease may die out spontaneously in a few weeks. It is usually trivial, without constitutional disturbance, and with only slight itching. In severe types the lesions may be circular, bullous or ulcerative. The disease is conveyed by contagion to other parts of the body or to another individual, especially with bullous impetigo.

Etiology.—Impetigo will spread through a school (*e.g.*, scrum-pox in football) or family of children, attacking weak and strong alike. Adults are comparatively immune. The usual cause is a streptococcus (often hæmolytic), soon followed by secondary staphylococcal infection. A form due to staphylococci has now become more common.

Diagnosis.—Impetigo pustules are readily distinguished from acne, sycosis, pustular syphilide, and all other pustular eruptions by (i.) their superficial character, (ii.) the crusts, and (iii.) their typical position. The bullous form may have to be distinguished from other bullous diseases.

Treatment.—The crusts must be removed before using a local application. The average case is cured with Eau d'Alibour—zinc. sulph. ; cupr. sulph. 4 ; aq. dest. ad 1000. Penicillin can be applied in spray or cream, but epidermal sensitisation occurs in some cases. If not successful within a week discontinue it and use the older remedies, especially hyd. ammon. chlor. gr. 5 in pasta zinci. Sulphathiazole cream 5 per cent. was used before penicillin became available. It must not be continued for longer than five days nor when the skin is exposed to bright sunlight.

II. **Ecthyma** is a term used to describe *large isolated and crusted pustules*, or the superficial sores which may form part of impetigo, scabies and pediculosis. Ulceration occurs beneath the heavy crusts, due to low tissue resistance, in delicate children and aged persons. In severe cases pigmentation and scarring follow. The lesions have to be distinguished from scabies and from suppurating syphilides. Treat the lesions as in impetigo ; the general health requires building up.

III. **Impetigo Herpetiformis** is a rare disease described by Hebra. It is characterised by the appearance of clusters of miliary pustules, usually starting on the inner surface of the thighs, whence they spread, generally associated with the pregnant or puerperal state, and usually terminating fatally. The mucous membranes may be severely affected. There is continuous or intermittent fever, and each fresh crop of pustules is attended by rigors and increasing prostration. It is usually connected with the later months of pregnancy and tends to recur in a subsequent pregnancy.

Treatment is as for pemphigus. Pregnancy may have to be terminated. Injections of autogenous serum or serum from healthy pregnant women have often succeeded.

IV. **Diphtheria** of the skin resembles a widespread, obstinate impetigo, with large, sometimes sanious crusts, and is usually diagnosed by the discovery of the organism after the disease has resisted ordinary treatment for impetigo. Injections and local applications of diphtheria antitoxin readily cure the lesions.

(*b*) *Pustules on an Indurated Base*

§ **641.** V. **Sycosis** is a slow pustular eruption, evidence of a deep-seated infection of the sebaceous glands and hair follicles of the beard and sometimes the moustache, by the staphylococcus aureus. Clinically, three conditions present the appearance described as sycosis. (1) *True Sycosis* (Synonym : Folliculitis Barbæ) begins round the hairs with discrete red papules ; these rapidly become yellow pustules ; inflammation may extend over the intervening area and cause nodules. In the later stage the hairs can be easily drawn out, followed by a drop of pus. The condition is due to the staphylococcus aureus ; hence it is sometimes called coccogenic sycosis. It may be contracted at the barber's, or it may be due to infection from a nasal discharge ; the eyebrows, eyelids, axilla, pubis and other hair follicles may also have pustules. (2) *Tinea Sycosis*, ringworm of the beard, is due to the trichophyton tonsurans. The large-spored ringworm, and the ringworm of horses, cows, cats, and dogs may produce its two varieties : (*a*) *Superficial*, characterised by scaly red rings, in which the hairs are only slightly involved ; and (*b*) *deep-seated*, showing hard nodules and purple red lumps. In this form the hairs are easily pulled out from the onset. Unless the invading fungus be found, it is often difficult to diagnose. (3) *Eczema barbæ* which has been secondarily in-

fected. In true sycosis the pustules predominate ; the intervening inflammation is secondary. Eczema barbæ may be a streptococcal dermatitis which has with intermissions lasted from youth ; the inflammation extends to the face and follicular involvement is rare. All three conditions may last long.

The *Treatment* of true sycosis is often lengthy. Keep the hair short ; open the pustules. Penicillin sometimes gives immediate success, but relapse is usual. Few now use sulphonamides locally. For mild cases, hydrarg. perchlor. (1 in 2000 parts of rectified spirit) can be used at night ; and by day a dusting powder of calomel gr. 10 in oz. 1 of talc powder. The part may be bathed at night with a warm solution of boracic acid followed by inunction with hydrarg. ammon. chlor. gr. 10–15 to oz. 1 and a lotion and powder by day. Quinolor ointment is often effective. In some cases vaccines, especially autogenous and intradermal, are very successful. Epilation by X-rays may cure, or be followed by relapse. Increase the vitamin C and reduce the sugar content of the diet. Remove septic foci. In Tinea sycosis, epilate ; then use a mercurial lotion and ointment.

§ **642.** VI. **Pustular Syphilides.** (1) The *Small Papulo-pustular Syphilide* shows papules about the size of a pin's head, upon a hard base, which in a week or ten days scab off, leaving the characteristic indurated lesions with depressed centres. They are arranged in groups, circles, or circular lines. (2) The *Large Pustular Syphilide* (Rupia) consists of pustules varying in size from a split pea to a halfpenny, flat or hemispherical, and surrounded by a raised brick-red infiltrated margin. They may be grouped, ringed, or isolated. The pustule bursts, the pus escapes, and crusts are formed with ulceration beneath them. The ulceration tends to spread serpiginously, and leaves permanent scars, rings, and pigmentation. Both varieties may occur on any part of the body. See § 645 for characters common to all syphilitic rashes.

Diagnosis.—The smaller pustular syphilide is distinguished from *acne* by the presence of comedones, and the slower course in *acne.* Pustular syphilide on the face may be hard to distinguish from *lupus vulgaris,* but in lupus the patient is usually younger, and the lesions grow more slowly. When pustular syphilide is diffuse, it may be mistaken for *small-pox,* but in the latter there is a history of a vesicular stage, of backache, and constitutional symptoms (§ 479).

§ **643.** Various other pustular eruptions may be mentioned :

VII. **Pustular Acne** is recognised at once by the presence of comedones, papules, and pustules on the face, and sometimes the upper part of the back. **Acne varioloformis** has no comedones, but a central depression with crust and resulting scar.

VIII. **Pustular Folliculitis** is a papulo-pustular condition of the hair follicles, due to infection by the staphylococcus aureus. It affects the hairy parts, especially the legs in men. It is diagnosed by the fact that each pustule involves a single hair follicle. In **Bockhardt's impetigo** only the upper third of the pilo-sebaceous follicle is involved ; hence these pustules are not always situated on an indurated base. It affects chiefly the scalps of infants or young children, or sites where an irritant has been rubbed into the skin.

IX. **Iodides, Bromides** and other drugs are mentioned in § 612.

X. VARIOLA (Small-pox).—The concluding stage of the eruption in this infectious fever is another illustration of pustules forming upon an indurated base (§ 479).

XI. The eruption of **Acute Glanders** when it has reached a pustular stage is so much like small-pox that it may very pardonably be mistaken for it (§ 491).

XII. A pustular **Tuberculide** may appear as small or large pustules arising on a hard papular base. These may coalesce, and under the scab an ulcerating surface develops.

XIII. **Dermatitis vegetans** may develop upon other diseases, especially in the groins and axillæ. Profuse dark-red granulations occur, with pus or crusts.

(c) *Pustular Eruptions prone to become Furuncular, or Sloughing: viz., Boil, Carbuncle, and Kerion*

§ 644. XIV. A **Furuncle,** or boil, shows an acute, red, tender and very painful nodule in the skin, varying in size from a pin's head to a bean. Sometimes the inflammation slowly subsides (blind boil); usually it increases and involves the surface; in a few days pus forms, the skin breaks, and the central necrosed portion or core is discharged. The cavity fills with granulation tissue and heals with a scar. Boils are often multiple, or fresh crops may continue for months (furunculosis). A boil may be a comparatively mild malady, running a course of 1 to 3 weeks; but on the face, especially the upper lip, it may lead to septicæmia and death.

Etiology.—Most boils are due to an inflammatory process involving the lower part of the hair follicle; the *Staphylococcus pyogenes aureus* is the usual causal organism; in mild cases, *S. albus* and *S. citreus.* Boils are seen in the obese, the over-fed and diabetic; carbuncles affect especially the aged, the underfed and debilitated. Friction with contaminated clothing is apt to infect the adjacent hair follicles and cause fresh boils; hot fomentations and ointments may act similarly. Crops of boils may also occur where there is poor resistance to various septic foci, *e.g.*, in the sinuses, the teeth and residual jaw infection.

XV. A **Carbuncle** may be regarded as a cluster of several boils, constituting an inflammatory area spreading beneath the skin, with numerous openings in the skin through which the pus pours. A leathery slough forms in the deeper layers of the dermis. It occurs most often on the neck or back, but may affect any part; on the face it is serious. The pain and constitutional disturbance are often very severe.

Treatment.—Protect from friction; apply 1 per cent. iodine in absolute alcohol or 2 per cent. gentian violet over and around the boil. If fomentations have been used to relieve pain, disinfect the neighbouring skin in the same way. Never squeeze a boil or a carbuncle, nor extract the central hair. Keep the part at rest, especially in the case of carbuncles; for this purpose use a firm occlusive dressing. Do not incise unless pus is pointing; in most cases conservative methods are better than surgical. When the skin breaks, encourage the flow of pus with dressings of gauze soaked in a saturated solution of magnesium sulphate in glycerin, changed several times daily, when the discharge is profuse. Injections of edwenil,

colloidal manganese and manganese butyrate help many cases. For severe boils and carbuncles penicillin is recommended by injection, 200,000 units in sterile water thrice daily for three to eight days. The dosage is still under revision. Local preparations may be used, but with many people there ensues sensitisation. Vaccines (especially autogenous) are often effective for boils (beginning with 30 or 50 million staphylococcus aureus) especially when given intradermally. Investigate the general health. Some require more, others less food; restrict sugar; give fresh foods. Vitamin C is especially indicated. Some advise sulphadiazine therapy. Autohæmotherapy (§ 656) and X-ray are very useful; others like short-wave diathermy and ultra-violet light. X-ray: a single suberythema dose often aborts boils and carbuncles; fractional weekly doses are used for recurrent boils. It is rarely necessary to resort to excision with a cautery or diathermy knife.

XVI. **Kerion** is a condition occurring chiefly on the heads of children suffering from ringworm of animal origin, usually a large spored ectothrix trichophyton. It is a pustular folliculitis, and resembles a carbuncle, but without induration. It shows a circular, raised, inflamed, boggy area of skin with holes discharging serum and pus (§ 655). It tends to spontaneous cure.

GROUP IV. MULTIFORM ERUPTIONS

Multiform eruptions are sometimes found in the following conditions —syphilis, scabies, eczema, erythema multiforme, varicella, leprosy, dermatitis herpetiformis and pityriasis lichenoides.

§ 645. General Characters of Syphilitic Eruptions.—(1) They are of many different *kinds*, and several forms may be present at one time (polymorphism). All varieties of elementary lesions may appear on the skin, but very rarely a vesicle; eczema and other vesicular lesions are never found as a result of syphilis—a diagnostic feature of great importance. (2) The syphilitic *papule* may be regarded as a prototype of a syphilitic skin lesion. It is the starting-point of them all. (3) The *features common* to all syphilitic rashes are their reddish-brown colour, generalised or symmetrical distribution, grouping in segments of circles, preference for the forehead and flexor aspects, polymorphism and absence of itching. The later skin lesions are usually asymmetrical, and with a marked tendency to ulceration.

The clinical features which distinguish syphilides are explained by three histological facts. (1) All syphilides are due to a deposit in the dermis or epidermis of a cellular *infiltration*. Hence the colour does not disappear on pressure, and is followed by staining. (2) The cells constituting this gummatous or granulomatous infiltration are of low vitality. They do not organise into connective tissue, but tend to undergo either *involution* by absorption or *suppuration* and pustulation. Hence the depressed cup-shaped centre, the great tendency to polymorphism and the absence of vesiculation. (3) The infiltration *spreads centrifugally*. Hence the raised peripheral edge is the newest part, the shape most frequently assumed being that of a crescent, circle, or segment of a circle, leaving a stained centre where the papule began. If these three principles be appreciated all the clinical features are explained.

Scabies (§ 621) is usually a multiform eruption, consisting of burrows, papules, vesicles, sometimes pustules, and scratch-marks. By the presence of the burrows and the sites infected, the diagnosis is made.

Pityriasis lichenoides et varioloformis is a rare disease with, in addition to the rash of pityriasis rosea (§ 630), crops of soft papules and lesions with umbilication and central scab. It is usually widespread, lasts long, and is important in that it may be mistaken for secondary syphilis or chicken-pox.

GROUP V. NODULAR ERUPTIONS AND TUMOURS OF THE SKIN

A nodule may be defined as a solid deposit in the skin, which is larger than a papule. The *commoner forms* are: I. Lupus Vulgaris; II. Syphilitic Gummata; III. Various Benign Tumours (*e.g.*, sebaceous cyst, lipoma, rheumatic nodules, vascular nævi, etc.); and IV. Epithelioma; while the *rarer forms* include: Leprosy; Bazin's Disease; Molluscum Contagiosum; Molluscum Fibrosum; Von Recklinghausen's Disease; Sarcoma Cutis; Leukæmia; Mycosis Fungoides; Xanthoma; Sarcoid; Myoma; Delhi Boil; Yaws; Actinomycosis; Blastomycosis; Sporotrichosis; and Mycetoma.

Some papular eruptions may take on a nodular form - *e.g.*, urticaria pigmentosa and prurigo. Rodent ulcer (§ 649. IV) may in the early stage be mistaken for a nodule.

§ 646. I. Lupus Vulgaris is a chronic disease of the skin presenting groups of small nodules seated in the corium. These are pinhead to lentil size, reddish brown, with characteristic semi-translucent appearance, soft, due to infection with the tubercle bacillus, frequently the bovine type. A zone of inflammation occurs around them, so that the so-called "apple jelly" nodules may only be seen when the patch is firmly pressed under a watch-glass. The disease may attack any part of the body; the face and neck are frequently, the head rarely affected. After measles or other acute fevers patches may develop extensively over the body. The mucous membranes (mouth, nose and larynx) are often invaded. Lupus usually starts in early childhood, rarely after the age of twenty, and usually progresses slowly. The patch may desquamate, or may heal in the centre with cicatricial atrophy, a few typical "apple jelly" nodules in the centre or margin enabling the diagnosis to be made; or it may ulcerate, with secondary infection, pus and crusts, causing destruction and deformity when on the nose, eyelids or mouth.

The *Prognosis* depends upon early treatment and till recent years was unfavourable. For *diagnosis*, see Table XLIII.

Treatment.—*General* treatment is necessary, especially with sunlight and diet. The carbon arc to the whole body is best; large doses are borne when the skin is anointed with olive oil. The Gerson diet has had success—abundant vitamin content; fresh fruit and vegetable juices; raw vegetables and salads; restricted water intake; salt replaced by calcium and other minerals; high meat and fat content. Calciferol has

replaced previous methods. One high potency tablet is taken thrice daily (150,000 units daily). Stop the drug for a month when there are clinical symptoms of nausea and lassitude or when the serum calcium

TABLE XLIII.—TABLE OF DIAGNOSIS.

Nodular Syphilide.	*Lupus Vulgaris.*	*Lupus Erythematosus.*
Nodular or diffuse infiltration with raised edges. Nodules firm.	"Apple-jelly" soft nodules in dermis. Sebaceous follicles not specially involved.	Superficial erythema. Sebaceous follicle plugs attached to surface scales.
Destroys more in a month than lupus in a year. Stellate scarring.	Destroys slowly and usually leaves puckered scar.	Never ulcerates; usually leaves a superficial fine atrophy.
Sometimes symmetrical.	Asymmetrical.	Bat's - wing distribution on face. Generally symmetrical.
Adults.	First appears in childhood or before twenty.	First appears in middle life or after twenty.
Responds to Hg and KI.	Hg. no marked effect.	Hg and KI no good.

content reaches 12 mgm. per cent. *Local* treatment: (1) The Lomholt modification of Finsen light is the best ultra-violet lamp; (2) diathermy fulguration to each nodule; (3) caustics; Adamson's method of liquid acid nitrate of mercury rubbed over the part; acute reaction follows, with resulting fine scar.

II. **Syphilitic Gummata** are round or ovoid nodules in or beneath the skin. In the course of a few weeks they form an indolent abscess, which leaves a circumscribed punched-out ulcer, sometimes of considerable depth. They may occur anywhere, but especially on the legs, brow, nose and sterno-clavicular region. A gumma must not be mistaken for an abscess, and lanced.

III. There are several other relatively common **Benign Tumours** or nodules originating in the subcutaneous tissue, which may involve the skin—*e.g.*, sebaceous cyst, fatty tumour, rheumatic nodules, fibro-neuroma, subcutaneous nævi, and lymphangiectasis. **Sebaceous Cyst** (Synonyms: Steatoma, Wen) is a tense, painless, cystic tumour due to the occlusion of a sebaceous follicle, usually single. It should be dissected out *with the capsule*, or it will grow again. **Fatty Tumours** are known by their doughy feel, lobulation, the puckered depressions seen on trying to lift up the skin over them. **Rheumatic Nodules** occur in successive crops, as small, hard, or elastic nodules, sometimes adherent to the skin, usually freely movable beneath, sometimes tender on pressure. Their favourite situation is over the fibrous tissue of the superficial bones around the joints and along the spine. **Lymphangioma**, or dilatation of the lymphatics, shows white or pink vesicles, usually in groups; telangiectases may be associated. On puncture lymph exudes. It occurs in childhood and often affects the tongue.

IV. **Epithelioma,** the squamous cell type, affects the skin in several forms. The *papular* form occurs as hard, pale, flat papules, which grow very slowly, and become cracked, fissured, and ulcerated. The deep-seated form occurs as close-set, flat, or slightly raised, firm, somewhat translucent nodules. In the course of months or years it becomes a spherical or flat, hard tumour, with shining, waxy, or rosy surface, traversed by vessels. As the result of spontaneous reaction the centre is often drawn in. Later, ulceration occurs. *Papillomatous* or warty growths may be found in the same individual; the first is the most common and slow growing. In all forms the glands become involved and metastases may form. The favourite sites are the lower lip—at least 50 per cent.—the tongue, and external genitalia. The majority of cases occur in **men**. Malignant growths often develop in pigmented moles, senile keratoses, the late stage of xeroderma pigmentosum, scars of lupus and X-ray dermatitis, and the dermatitis following exposure to mineral oils, tar or arsenic. **Paget's disease** affects women over 40. It begins as a scaly patch resembling eczema, which spreads with a defined margin, becoming bright red and glazed. It usually affects the nipple and areola, but may occur on any part of the body. Later, a distinctive hard infiltration is felt under the skin. **Bowen's disease** is another rare pre-cancerous disease, which in the early stage is often mistaken for syphilis. Groups of lenticular nodules occur, with crusted tops beneath which ulcers extend.

§ 647. Certain **rarer forms** of nodule and neoplasm also affect the skin.

Leprosy (Synonyms: Lepra, Elephantiasis Græcorum, Leontiasis Satyriasis) is characterised by pigmentary, sensory, and nodular changes in the skin, due to *Mycobacterium lepræ*, discovered by Hansen in 1871. The organism is acid-fast and occurs in large clumps in skin lepromata, septic ulcers and nasal mucus. Leprosy used to be a widely prevalent disease, but mainly imported cases are now found in England. It is still endemic in Norway, parts of Southern Europe, Africa, Mexico, America, and in China, India, West Indies, etc. It is communicable from man to man, but resistance is high in adults. Children are much more susceptible and infection usually occurs in early life. The actual mode of its dissemination is unknown. There are two clinical forms in its earlier stages. (*a*) *Maculo-anæsthetic* leprosy consists of patches of anæsthesia, sometimes of pigmentation or leucoderma, usually associated with thickening of the nerve trunk connected with the part, and a widespread eruption of reddish spots and patches over the body. These signs may be preceded by pain, and followed by paralysis and atrophy of the muscles supplied by the affected nerves. (*b*) In *Nodular* leprosy are found small diffuse thickenings, sometimes pink, yellowish-brown, or without much alteration of colour of the skin and mucous membranes. These increase to form bosses which on the face give the patient a leonine aspect in course of time (facies leonis). The viscera and mucous membranes are similarly involved, and wherever the granulomatous material is formed the characteristic bacillus is found. *Mixed* forms of these two types are met with. The course of the disease is extremely prolonged, and generally fatal. The differential *Diagnosis* is from lupus vulgaris, skin tuberculosis, yaws and syphilis. The anæsthetic type has to be distinguished from progressive muscular atrophy, amyotrophic lateral sclerosis, cervical rib, neuritis, scleroderma and Raynaud's disease. In nodular leprosy the organisms may be found in the nasal mucus or in snippets of skin removed with curved scissors, but in pure nerve cases the bacilli only occur in the nerves themselves. The ulnar, peroneal, great auricular and other nerves may be palpably enlarged. *Varieties.—Lepromatous leprosy* indicates a poor resistance of the individual, with many bacilli in the lesions and a negative lepromin reaction. *Tuberculoid leprosy* demonstrates a high resistance, with few bacilli present and a positive lepromin reaction.

Treatment.—Good food, fresh air, exercise and the elimination of intercurrent disease are essential points. The sulphone compounds offer the best chance of success. Those commonly used are promin (1–5 G. daily intravenously), promizole (0·5–1·0 G. t.i.d. by mouth), diasone (0·3–0·9 G. daily by mouth) or sulphetrone (0·5–2·0 G. t.i.d. by mouth): anæmia, leucopenia and dermatitis may result from these drugs. Iron

and Vitamin B complex should be given concurrently, with regular blood counts. The drugs are best given in repeated courses, separated by short intervals, and treatment is prolonged for a year or more. Some success has been claimed for streptomycin, but treatment must be prolonged and loss of further response and toxic effects supervene.

Bazin's Disease (Synonym: Erythema Induratum) shows two forms. One is a tuberculide, affecting chiefly young women, characterised by chronic subcutaneous bluish-red nodules in the calves of the legs, which may ulcerate. They yield to rest and tonic treatment, also to a few injections of neoarsphenamine. They are sometimes difficult to distinguish from syphilitic gummata, but these make more rapid progress and yield to iodides. This type is especially liable to affect girls whose legs show a cyanotic condition with vascular stasis. (See §§ 576 and 617.) The second type is seen chiefly in women between thirty-five and fifty who suffer from rheumatism; it may also occur in men. The nodules are smaller but more painful. The swellings may develop rather rapidly but do not break. They subside in a few days when the patient stays in bed. A focus of infection should be sought for and dealt with.

Molluscum Contagiosum consists of rounded, pearl-like elevations, varying in size from a pin's head to a pea, with semi-translucent appearance. They are due to the reaction of the prickle cells to a minute infecting organism, which is apparently conveyed by towels and other washing materials. A tiny depression is found in the centre through which the contents can be squeezed. If left alone inflammation and suppuration may occur, with spontaneous cure, or large nodules may form. The treatment consists either in snipping them off, or in squeezing out the contents, and touching the inner surface with silver nitrate, pure phenol or iodine.

In **Molluscum Fibrosum** there is a formation of fibrous tissue in or just beneath the corium, slowly developing into tumours of varying size (up to 32 pounds), which may be sessile or pedunculated. Their favourite situation is the back. They should be removed by knife or ligature. In **Von Recklinghausen's disease** there are pedunculated growths containing nerve as well as fibrous tissue (neurofibromata); they are widely distributed and pigmentation occurs in patches. Although probably congenital, they may not develop till middle or old age. It has (rarely) been associated with adenoma sebaceum; and see § 803.

Sarcoma Cutis may be primary or secondary. It is met with in the form of purplish tumours of varying size of hard or spongy consistence. A small deposit with satellites around it is very characteristic. Sarcoma may develop on pigmented moles, a melanotic sarcoma being then reproduced elsewhere.

In both types of **Leukæmia** (§ 543), but especially in the lymphatic type, and in **lymphadenoma,** nodules in the skin may occur of the same character as those in the spleen, liver, etc. The nodules vary in colour from that of the surrounding skin to a deep red or even to a distinct grey, and are often the site of hæmorrhage. The greenish hue of an old bruise may suggest that they are chloromatous, but this is rare. They appear in any position, and are variable in size and persistence, sometimes disappearing for months at a time. If a blood examination is not made, such cases are often regarded as mycosis fungoides. Itching may be severe or absent. In some cases there is extensive infiltrated erythrodermia with severe itching.

Mycosis Fungoides is characterised by the formation, after a long preliminary period, of reddish fungoid tumours. In the preliminary stages, which may last for months or years, there is an erythema or a scaly eczema attended by itching. Later, brownish-red papules develop, leaving pigmented depressions. These are followed by smooth purple tumours, sessile or pedunculated, which ulcerate, with a typical granulomatous base. The eruption usually appears on the trunk, and leads to emaciation and death. X-rays have proved useful in retarding the progress of the disease.

Xanthoma occurs with liver disease and diabetes in the form of raised yellow patches (X. planum) or nodules (X. tuberosum). It is described in § 653. IX.

Sarcoid is a term used for several rare eruptions associated with a tuberculous

focus. (1) Darier and Roussy described deep-seated, dark-red or purple elastic nodules below the skin, which usually resolve but may have a central softening. (2) Boeck described four types of sarcoid: (i.) crops of superficial dark-red or brown, firm papules affecting symmetrically the face, scalp, shoulders, limbs or trunk, usually involuting and leaving a fine scar. The above are **tuberculides,** *i.e.*, cutaneous inflammatory reactions to tubercle bacilli; the von Pirquet is positive. (ii.) A rare superficial form, affecting face, scalp and neck, which extends with a serpiginous border; (iii.) deeper nodules, pea to bean-sized, purple or reddish-brown; and (iv.) a diffuse infiltrated type, affecting the nose, cheeks, ears, backs of hands and fingers—**lupus pernio.** Schaumann's researches proved that the last two varieties belonged to the class of chronic benign lymphogranulomata. They may be widespread, occurring in the skin, tonsils, lymphatic glands, lungs, spleen, liver, and the medulla of the bones of the extremities. X-ray reveals changes in the lungs and circular areas of rarefaction in the bones of the phalanges, metacarpals, metatarsals, sometimes in the carpus, tarsus and the ends of long bones. The von Pirquet is negative. In cases where pulmonary tuberculosis develops, the von Pirquet becomes positive and the skin lesions disappear.

The *prognosis* is usually good.

Treatment.—Cures have been obtained by intramuscular injections of gold and of sodium morrhuate, local and general ultra-violet light and X-ray. Sutton finds arsenic specific for Boeck's sarcoid.

Myoma is composed of non-striated muscle, and very rare. The nodules are small, smooth, red, often painful, usually diagnosed after biopsy. Treat by excision.

Delhi Boil (Bagdad button, Oriental sore, cutaneous Leishmaniasis) occurs in the East from the bite of a sandfly infected with *Leishmania tropica.* It begins on the exposed parts of face and hands as a hard papule, which enlarges, softens, becomes dark purple in hue, and in three or four months usually ulcerates, with a foul yellow secretion. It tends to heal, leaving a scar in about a year. The diagnosis is made by finding the Leishmania in the papule or ulcer margins. Locally, diathermy coagulation, pastille doses of X-ray, or the use, after scraping, of a 2 to 4 per cent. tartrate of antimony ointment, are all good. Cure may be obtained by intravenous injections of tartar emetic, given thrice weekly. Begin with ½ grain and work up to a maximum dose of 2 grains; 15 to 30 grains may cure. **Espundia** is a similar condition found in S. America. It is accompanied by oropharyngeal ulceration, and is amenable to tartar emetic injections.

Frambesia or **yaws** is a tropical disease of widespread distribution affecting dark-skinned races especially in childhood. The causative agent, *Treponema pertenue*, is indistinguishable from the spirochæte of syphilis, *T. pallidum*, and is spread by direct contact. The primary lesion is extragenital and frequently not seen: the secondary eruption, 2–4 months after infection, may be accompanied by fever, headache, bone pains and arthralgia. It is symmetrical and may appear in successive crops on face, neck, arms, buttocks and genitals, occasionally the mucous membranes. Small papules form, which desquamate, produce a raw raspberry-like surface exuding yellowish serum which dries, with crusts resembling rupia. The tertiary lesions include osteitis, periostitis, arthritis, sabre tibia and chronic gummatous-like ulceration of the subcutaneous tissues. Gangosa, goundou and juxta-articular nodes are now regarded as sequelæ of yaws.

Etiology.—Yaws has been regarded as syphilis of primitive races, but it is of non-venereal origin, never congenital, and does not involve the viscera or central nervous system.

Treatment.—Neoarsphenamine, penicillin or bismuth as in syphilis. The secondary lesions rapidly clear, but the amount of treatment necessary is not yet known.

§ 648. The following diseases are rare in Britain; their lesions have to be diagnosed from each other. Actinomycosis, Blastomycosis and **Sporotrichosis are** due to a fungus entering through abrasions or pricks. All show nodules which ulcerate; some become verrucose or papillomatous. Before the various fungi are detected, the lesions may

be mistaken for tuberculosis, syphilis or Bazin's disease. The prognosis varies: some skin lesions yield to iodides, penicillin and X-ray; systemic and visceral involvement with fever, neutrophil leucocytosis and anæmia is very serious, often fatal.

Actinomycosis usually starts in the mouth, tonsils, tongue or jaw, and spreads to the skin of the face and neck, due to infection by the Actinomyces fungus. A hard, slow-growing tumour ulcerates, with a thin sero-purulent discharge, containing yellow granules in which the fungus is found (§ 921). Or the digestive tract, especially the cæcum and appendix, is attacked, with secondary involvement of the liver and lungs and with severe constitutional symptoms. Penicillin is effective for the anærobic, sulphadiazine for the ærobic fungus. Large doses of penicillin in repeated courses are required for a long period (Table XXX).

Blastomycosis affects chiefly the face and hands. It may attack the viscera primarily or secondarily. It responds to large doses of sod. iodide, X-ray and excision.

Sporotrichosis is due to infection with sporotrichia. Inflammatory nodules occur on the skin and mucous membranes, in subcutaneous tissue and bones; they may grow slowly or come out in rapid crops. Some ulcerate and discharge a sticky pus containing the fungus. Potassium iodide cures.

Mycetoma (Madura foot: elephant foot) is a chronic granuloma affecting especially the foot. Multiple nodular swellings suppurate and break down, forming sinuses and fistulæ. The pus contains fungoid granules. There is generally a history of trauma with thorns or sharp-grained cereals in barefooted natives, the fungi thus gaining access. Systemic manifestations are absent unless secondary infection supervenes. *Prognosis:* Complete surgical removal of the diseased tissue, or amputation is necessary when sulphonamide therapy fails.

GROUP VI. ULCERATIONS

§ **649.** An **Ulcer** is a loss of substance of the whole skin; a granulating surface is exposed, with sero-purulent exudation. Certain forms of ulcer involve the deeper tissues, muscles, tendons, and periosteum. Ulcers must not be confused with ruptured vesicles or bullæ, when only the cuticle is absent. From the clinical standpoint, ulcers may be classified under six headings.

(*a*) *Indolent* inflammatory ulcers appear on the legs, especially when there is lack of vasomotor tone, with œdema and no support from boots. As this type is so often associated with varicose veins, it is usually called "varicose ulcer." It also occurs independently of varicose veins, in those whose occupation entails much standing; with defective circulation; with obstruction of the lymphatic or venous return, as in pregnancy or pelvic tumour; with disease of the veins, as after typhoid and pneumonia; or after local injury. The ulcer is usually preceded by œdema, cyanosis, even dermatitis. The ulcers associated with varicose veins vary in size from a pinpoint to the entire circumference of the leg.

Treatment.—Vitamin E and calciferol are on trial. Copper sulphate reduces redundant granulations; zinc ionisation and ultra-violet light stimulate healthy healing. Dickson Wright uses 6 metres of 3-inch adhesive plaster bandage of an elastic type, extending from the toes to the knee, binding it firmly and evenly over all, even if ulcer and dermatitis are present. The greater the œdema, the tighter must be the bandage.

In some cases with marked eczema and dermatitis, begin with a zinc gelatine bandage (varicosan, viscopaste). Varicose veins are injected with 5 per cent. sodium morrhuate, the upper parts being first dealt with (§ 569). If the ulcer is very large, skin grafts are laid upon it and the bandage is bound over all. The ulcer receives no dressing; powdered aspirin is blown over it if painful. Penicillin aids when penicillin-sensitive secondary infection is present. The bandage remains on from three to thirty days; the time of its removal depends on the amount of discharge and shrinking as the œdema diminishes. A moderate amount of exercise is encouraged.

(*b*) *Contagious Ulcers* are seen with glanders, hard and soft chancre, acute vulvar ulcers (rare and of unknown causation).

(*c*) *Ulcers due to nerve lesions : e.g.*, tabes dorsalis, trophic ulcers.

(*d*) *Neoplastic ulcers* and ulcers due to the breaking down of granulomata or infiltrations of the skin and subcutaneous tissue, such as syphilitic lesions (rupia and gumma), tuberculous lesions such as lupus vulgaris, verruca necrogenica, the skin over tuberculous glands; malignant disease, such as epithelioma and rodent ulcer; Bazin's disease, leprosy, yaws, veld sores, sporotrichosis, actinomycosis, blastomycosis, Leishmaniasis, Espundia, and other nodular and pustular conditions.

(*e*) Breaking down of cutaneous *scars*, such as occur with radiodermatitis, scleroderma, xeroderma pigmentosum.

(*f*) Two forms of ulcer frequently met with in the tropics deserve mention here: (1) The so-called *Tropical Ulcer* occurs in warm, damp climates. It begins as a bulla which ulcerates slowly through muscles and vessels to the periosteum. It is believed to be a deficiency disease, due to lack of protein and salt. It has been cured with daily injections of calcium chloride (gr. 15 in 10 c.c. aq. dest.), also by excision and skin grafting. (2) *Granuloma inguinale* occurs in negroes in the tropics and southern regions of America. It is described in § 571. VI.

An **infiltrating, ulcerating,** *and* **scarring** *eruption in a person of young or middle age is usually* due to lupus, tubercle, *or* syphilis. *If it occurs over forty or forty-five,* rodent ulcer *and* epithelioma *enter the category.*

I. LUPUS VULGARIS may ulcerate when near a mucous orifice, or subjected to injury and secondary infection. The nodules around are sufficiently characteristic (§ 646).

II. **Tuberculous Ulceration** occasionally originates from tuberculous deposits in the skin, but more often spreads from a caseating gland or bone disease. It is usually chronic. The edges of the ulcer are dark purple, and undermined, never rounded and clean-cut as in syphilis. Generally other evidences of tuberculosis, or scars from past disease are seen. The patients are usually children, occasionally an old person; and see Bazin's disease (§ 647). *Treatment :* Promin is proving useful when applied locally.

III. **Syphilitic Ulceration**—other than the primary chancre—is of two kinds: (1) The large papular or lenticular syphilide *in* the skin gives rise to shallow irregular ulceration which may be covered with a scab which resembles the layers of an oyster shell (**rupia** of older authors). (2) The gumma nodule which has started *beneath* the skin produces a deep punched-out ulcer. The three characteristic signs about all syphilitic ulcerations are—(1) the peripheral ring of infiltration, (2) the punched-out edge, and (3) the comparatively rapid march.

IV. **Rodent Ulcer** is a basal-cell epithelioma affecting especially men, and chiefly the upper part of the face, usually after forty years of age.

It has several forms: the most common begins as a small nodule with a hard, pale, "rolled edge." Slowly, an ulcer develops with a scanty, viscid crust. The glands are not involved, and there are no metastases. In another variety multiple lesions are seen, resembling at first senile keratoses; they spread at the margins, with tiny, pearly nodules. They may affect any part of the body and occur chiefly in women. In another type there is rapid ulceration extending even to the bone.

TABLE XLIV.—TABLE OF DIAGNOSIS.

Syphilitic, Rupial, or Gummatous Ulceration.	*Ulcerating Lupus.*	*Rodent Ulcer.*
Anywhere.	Chiefly face.	Chiefly face.
Adult life.	Begins between ten and twenty years.	Over forty.
Progress rapid, destroying in weeks what others do in months.	Very slow.	Slow.
Sharp, clear-cut, punched-out, deep.	Edge rounded, sloping, and surrounded by apple-jelly nodules; superficial.	Edge rolled and *hard*; deep in later stages.
Discharge copious; offensive.	Scanty, inoffensive.	Scanty, and in later stages sanguineous.
KI and Hg efficacious.	Variable result.	No good.

V. **Epithelioma** in course of time undergoes ulceration. The diagnosis rests upon the character of the initial growth, which is warty or infiltrated, and hard. The ulcer has a hard margin. If a small piece of the hard growth can be examined, the microscope reveals the typical "cell-nest" growths of epithelioma which are absent in rodent ulcer.

The *treatment* of neoplastic ulcers: (1) Appropriate constitutional treatment; (2) various operative procedures—removal, diathermy, or cauterisation; and (3) expert use of X-ray or radium. Carbon dioxide snow and diathermy coagulation give beautiful results in some cases of rodent ulcer.

GROUP VII. WARTS AND EXCRESCENCES

§ **650.** This group consists of Verruca (wart); Corns; Condyloma; Papilloma lineare; Keratodermia; Rupia; Acanthosis nigricans; Porokeratosis; and Angiokeratoma.

I. **Verruca,** or wart, consists of thickened epidermis above hypertrophied, elongated papillæ. Warts may occur singly, or they may be multiple. They are frequently met with on the hands; less often on the soles of the feet, the head, face, neck, or genital organs. Warts are due to a filter-passing virus. They are auto-inoculable and sometimes contagious.

Varieties.—V. Vulgaris, the common wart, occurs chiefly on the hands, about the size of a small pea; on the soles of the feet it is often mistaken for a corn. Warts may be flat, raised, acuminate or pedunculated. The so-called *seborrhœic wart* is a flat, dark-brown elevation found on the face

or body, usually of old people; it gradually spreads. It must not be mistaken for the scaly patch, *keratosis senilis,* which may become malignant. *Juvenile flat warts* affect chiefly children; they come in groups, and are pale, smooth, flat, like lichen planus except in colour. The venereal wart (*condyloma*) affects the genital region. When moist, large foul-smelling vegetations may form. *Acuminate warts* occur where there is discharge, at the ano-genital region, armpits, folds, rarely the scalp. *V. necrogenica* is a tuberculous infection of the hand; the post-mortem wart contracted by doctors, butchers, post-mortem porters, etc., begins as a red, indurated papule, which spreads, becomes pustular, and leaves behind a stellate cicatrix.

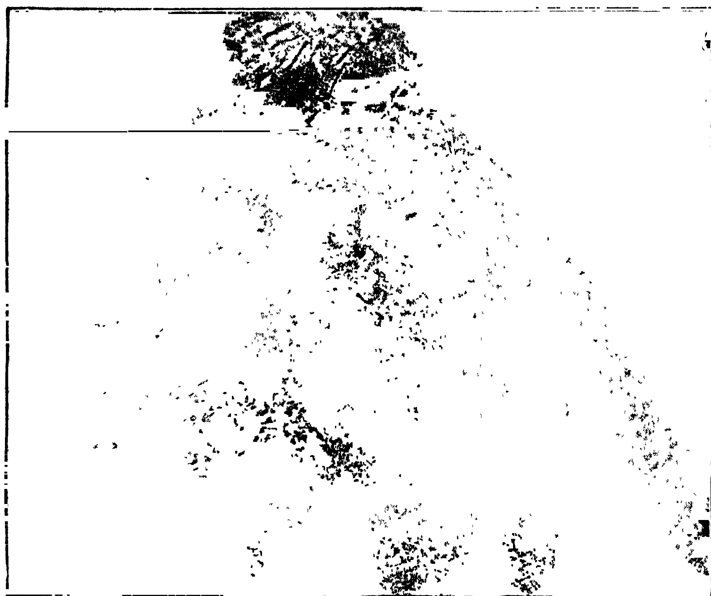


FIG. 149.—VERRUCA NECROGENICA on the hand of a gamekeeper, aged thirty-five.

Treatment.—Warts can be treated by many methods. After injecting a local anæsthetic, they can be curetted out, or destroyed by diathermy fulguration. Carbonic acid snow in a stick or a slush with acetone is preferred by some. Weekly painting with strong acids (glacial acetic, nitric) or daily application of a moistened silver nitrate stick also is effective. For patches with many flat juvenile warts, paint with 1 in 1,000 hyd. biniod. in spirit, and ionise with zinc sulphate after pricking each with a needle. The base of large warts can be transfixed in two directions with a zinc needle at the positive pole; a current of 2 ma. is passed for 2 or 3 minutes. Acuminate warts are destroyed by diathermy or podophyllin resin in oil when correctly applied. Internally, mag. sulph. gr. viii t.d. has aided some cases.

II. **Corns** are localised thickenings of the epidermis consequent on intermittent pressure. The side of the toe is a common position. They may be cured by painting with salicylic acid (20 per cent.) in collodion every night and paring off the softened horny layer. X-ray is used for obstinate corns. *Soft corns* arise between the toes, due to hard corns becoming soddened. *Treatment* consists in keeping them dry with dusting powder and the toes separate with small pads of cotton-wool. In both forms it is essential to relieve pressure.

III. **Syphilitic Condyloma** is really a papular syphilide occurring (1) on the mucous membranes; (2) near the junction of mucous membrane and skin; or (3) where opposed skin surfaces are in contact. They are common at the angles of the mouth, and between the buttocks or labia. They are slightly raised discs of various sizes, covered with greyish epithelial or soddened epidermal flakes, and exuding a highly contagious fluid.

IV. **Papilloma Lineare** shows a thickset aggregation of little horny, wart-like processes which entangle the dirt, and thus look brownish-black. These are arranged in streaks, and often described as nævus verrucosus.

V. **Keratodermia** may occur in (1) syphilis. In the tertiary stages it appears as a thickened brownish hyperkeratosis of the sole of the foot, usually associated with a thickening of the whole leg. It may also occur in the secondary stage, when it is bilateral and not usually so marked in degree. In debilitated subjects a secondary syphilitic lesion may ulcerate, with a dried blood-stained crust, like a limpet shell, and is known as **Rupia.** (2) Keratoses of the palms and soles associated with gout and with epidermophytosis have a surrounding redness and often fissures. Psoriasis of the palms or soles may simulate keratosis. (3) Gonorrhœal keratoses occur chiefly on the soles, but may affect the palms and other regions. (4) Keratosis palmaris and plantaris (tylosis) is a family and hereditary hyperkeratosis which may have marked horny excrescences on palms and soles. (5) Arsenical keratosis affects chiefly the palms and soles.

VI. **Acanthosis Nigricans** is a rare condition characterised by progressive pigmentation of the skin, with symmetrical papillary growths, often terminating fatally in a few months. The colour varies from a sallow hue to bronze and dirty brown. It is generalised, but more pronounced in the flexures. The disease may occur at any age. In most of the older patients it has been associated with abdominal (and especially gastric) cancer. In young cases the disease may last for years, no definite cause being found.

VII. **Porokeratosis** is very rare. Occurring chiefly on the backs of the hands and on the feet, it has patches of atrophic skin, surrounded by a thin horny ridge or " wall " immediately inside which are tiny grey papules, which can be picked out. It starts in childhood and progresses slowly.

VIII. **Angiokeratoma** consists of telangiectases, which develop into warty growths, occurring usually after chilblains, on the backs of the fingers, toes, hands, and feet. *Treatment :* warmth, electrolysis, diathermy coagulation or CO_2 snow.

GROUP VIII. ATROPHIES AND SCARS

§ 651. Scars, scleroderma, and atrophy of the skin may be considered together, because they not only resemble each other clinically, but fibrosis of some of the cutaneous tissues and atrophy of others occur in varying degrees in all three conditions. The disorders met with in this group are:

I. Scars. II. Atrophoderma. III. Scleroderma. IV. Sclerema neonatorum. V. Scleredema adultorum. VI. Keloid, Acne keloid and other rare conditions: Rhinoscleroma, Kraurosis and Leukoplakia Vulvæ.

I. **Scars** and atrophy may follow several skin diseases, such as lupus erythematosus, syphilis, leprosy, radio-dermatitis, lichen planus, hydroa æstivale and xeroderma pigmentosum. Scars may result from burns, wounds, or infiltrating or suppurating eruption in which there has been an ulcer or a loss of substance. If deformity or loss of mobility results, plastic operations are called for; but much can be done in young patients by means of persevering massage with oily substances.

II. **Atrophoderma** (Atrophy of the Skin) occurs as: (*a*) Atrophy of the entire cutaneous covering is common in old age. (*b*) Primary diffuse atrophy begins on the limbs and may extend to the body. The skin is shiny, wrinkled, lax; the subcutaneous tissues atrophy. The cause is unknown. (*c*) Lineæ albicantes is a term applied to the atrophic streaks found on the abdomen and breasts after pregnancy, over the hips and other parts when a stout patient has been reduced, and in obesity due to pituitary deficiency. Striæ atrophicæ may also occur after fevers, especially pneumonia and typhoid. (*d*) In macular atrophy small spots appear, with crinkled, loose skin; they may follow syphilitic or tuberculide papules. A form without known cause shows bladder-like patches, rose or white, into which the finger can be pushed, owing to the lack of elastic tissue (anetodermia). (*e*) *Unilateral atrophy* is met with in Hemiatrophy Facialis (§ 857), which is of nerve origin. (*f*) Atrophy may occur with scleroderma. (*g*) *Insulin Fat Atrophy* involves the subcutaneous fat: it is liable to occur in local areas at the site of repeated insulin injections.

III. **Scleroderma** occurs in two clinical forms: (*a*) Localised (or morphœa); (*b*) diffuse.

(*a*) LOCALISED SCLERODERMA (Synonym: Morphœa) is a disease consisting of one or more localised ivory patches or bands of sclerosed skin with, in the earlier stages, a congested lilac border or telangiectases. The guttate variety may be atrophic with a rose or lilac border (*White Spot disease*). There are few or no subjective reactions, but the tactile sensation is diminished. Some cases undergo spontaneous resolution in the course of years. The favourite situations are the face, neck, and beneath the breast. There is a tendency to symmetry. The position of some patches appears to correspond with the cutaneous distribution of a posterior nerve root. Women are more often affected, and especially in the first half of life. Beyond the disfigurement and contraction the patient suffers but little inconvenience.

(*b*) DIFFUSE SCLERODERMA starts insidiously, often symmetrically, usually affecting first the upper part of the body. The skin becomes smooth, glossy, thick, like parchment or wax in its substance. This progressively increases until the parts become completely hidebound and immobile. The face in such cases wears a smooth, expressionless aspect. Pigmentation may be present and extensive. Fissures and ulcers form and rigidity of the parts involved leads to deformities. Death from some intercurrent malady usually follows. Many degrees of severity are met with. The disease may be stationary for years, and in some the condition only produces liability to cold and to various superadded skin lesions. In the form known as **sclerodactylia** the disease begins with pallor and shrinking of the extremities, as in Raynaud's disease; atrophy and stiffening follow and spread upwards.

Treatment.—Hot air baths and local massage with oily applications are very helpful. Mild generalised scleroderma often improves under thyroid and œstrin. The constant current, electrolysis, radiant heat baths, diathermy and fibrolysin aid the localised form. Vitamin D holds promise.

IV. **Sclerema neonatorum** appears at or soon after birth, as hard, shiny, sometimes livid swellings, usually symmetrical, on legs, thighs, buttocks, back, arms and cheeks. They are due to solidification of the fat, owing to unknown causes; there are also crystal deposits in the tissues. Similar swellings appear in marasmic infants some

weeks or months old. Both conditions have been confused with scleroderma. In some cases the swellings disappear spontaneously; in others there is a fatal issue.

V. **Scleredema adultorum** (Buschke) affects chiefly the skin of the face and neck, with swelling and induration, due to swollen collagenous bundles. It follows a febrile state (throat, influenza) and tends to spontaneous recovery. The disease has been mistaken for scleroderma, but the history of a preceding febrile malady is distinctive.

VI. **Keloid** consists of a fibromatous deposit in the skin occurring occasionally in unaffected skin, but chiefly in old cicatrices, especially after burns. The lesion appears as a small firm nodule, of a crimson or pinkish colour, which slowly enlarges by means of tentacle-like processes. At first it is raised above the skin level. A **hypertrophic scar** does not spread beyond the region of the original scar. If excised, keloids immediately recur. The negative pole of a mild constant current, iodine ionisation, fibrolysin injections, radium, thorium X and X-rays have succeeded in early cases. Old, hard keloids are more resistant.

Acne Keloid (Syn.: Dermatitis Papillaris Capillitii) affects the follicles of the nape of the neck, a slow pustular affection, resulting in keloid formation. Epilation, ung. hyd. ammon. and X-rays in later cases give the best result.

Rhinoscleroma is a chronic inflammatory affection characterised by the development of hard, circumscribed plaques in the skin and mucous membrane, most commonly of the nose and naso-pharynx, due to a specific bacillus. The bones and cartilage may be involved. X-ray treatment is indicated.

Kraurosis Vulvæ affects the mucous membrane of the external genitals in elderly and in castrated younger women. Gradually the parts atrophy and shrink; cracks and fissures may follow. Telangiectases form near the urethra and on the vulva; sometimes these recur, with at the same time sore patches in the mouth. Urethral infection and vaginal discharge may be partly causal. The atrophy and contraction are aided by pelvic diathermy and œstrin.

Leukoplakia vulva shows milky white raised patches. Four conditions—lichenification, chronic eczema, kraurosis and lichen planus—are often wrongly labelled leukoplakia. As leukoplakia is a precursor of malignant disease early excision or diathermy coagulation should be performed.

GROUP IX. PIGMENTARY AND VASCULAR ALTERATIONS

§ **652.** Alterations of colour depend mainly upon the condition of the vessels and the amount of pigment in the skin. In healthy persons, exposure to sunlight, direct or artificial, leads to pigmentation of the skin. Pigmentation of the buccal mucous membranes is seen chiefly in Addison's disease.

A *diminution* of pigment occurs in two conditions: (1) **Albinism,** a congenital condition in which there is deficiency or absence of pigment in the skin and its appendages, and in the iris and choroid; and (2) **Vitiligo.**

Vitiligo (Syn. Leukoderma) shows white depigmented areas surrounded by increased pigmentation. The patches are oval or round, usually of symmetrical distribution, affecting any part, but especially the face, arms and trunk. As the white regions grow and adjacent patches join, the surrounding hyperpigmented zone becomes conspicuous.

The *diagnosis* is usually simple because of the concave and polycyclical margin of the pigmented zone. *Syphilitic leukoderma* which affects the neck has a more reticular appearance. The sensory changes in *leprosy* differentiate it from vitiligo.

Etiology.—Vitiligo affects both sexes, at any age, rarely under ten, and usually begins before thirty. It has been associated with alopecia, scleroderma, syphilis, Graves' disease and various organic nervous diseases such as subacute combined degeneration, and with vertebral disease involving the nerve roots. Septic foci have sometimes appeared to be causal.

Prognosis.—The disease extends slowly for years, but often recovers eventually.

Treatment usually consists in making the whiteness less conspicuous by applying a dark colour over the patches. Good results follow painting with 10 per cent. oil of bergamot in alcohol and exposing to ultra-violet light. Mercuric chloride lotions may be tried, as for freckles (see below). Gold salts have cured some cases. The general health should be treated—remove septic foci, and give abundant nutrition, also vitamin therapy with the whole B. complex. B. Sieve had success with *p*-aminobenzoic acid.

a. Large areas or a *generalised* increase of pigment occurs in (1) arsenical and silver pigmentation ; (2) Addison's and Graves' disease ; (3) abdominal tuberculosis, cancer and other growths ; (4) cardio-vascular disease ; (5) bronzed diabetes ; (6) constipation ; (7) melanotic sarcoma ; and (8) acanthosis nigricans ; (9) pernicious anæmia. In these the pigmentation is subordinate to other symptoms.

b. A *localised* increase of pigment or alteration in colour occurs in :

I. Chloasma.
II. Lentigo (freckle).
III. Pityriasis versicolor.
IV. Telangiectases and Nævi.
V. Purpura.
VI. Urticaria pigmentosa.
VII. Carotinæmia.
VIII. Xeroderma pigmentosum.
IX. Xanthoma.
X. Morphœa alba and nigra.
XI. Ochronosis.
XII. Leprosy.
XIII. Von Recklinghausen's disease.
XIV. Mongolian spot.

I. **Chloasma** occurs in single or multiple patches of diffuse discoloration on various parts of the body, varying in shade from a light yellow to a deep brown. 1. It is met with most frequently in pregnancy or uterine disease, and its most usual position is on the face and round the nipples. 2. It occurs in association with malaria, cancer, senile atrophy, rheumatoid arthritis, hepatic and pancreatic cirrhosis, lymphadenoma, abdominal tubercle or cancer, and exophthalmic goitre. Pigmentary syphilide may take this form, usually seen on the neck. 3. *Chloasma traumatica* is the pigmentation beneath the garters, or around the waist with tight corsets, after prolonged scratching, with pediculosis and lichenification, and after sinapisms, blisters, etc. In this category may be included the pigmentation which follows chronic eczema, friction, syphilis, lichen planus, psoriasis, X-ray or other cause of long-continued dilatation of the skin capillaries. 4. *Chloasma caloricum* is the reticulated pigmentation due to sun and wind, or to heat, as on the shins of women who sit over the fire.

II. **Lentigo** (Synonyms : Freckles, Ephelides).—Freckles are multiple, circumscribed, small flat pigment spots on the portions of the body exposed to light. Hydrarg. perchlor. ½ per cent. in alcohol may be tried cautiously ; paint on twice daily till scaling ensues. Or touch with pure phenol, wiping it off with spirit when the part whitens. In old people pigmented patches of all sizes may appear, the so-called "liver spots." In pituitary deficiency, dark spots are often seen.

III. **Pityriasis Versicolor** (Synonym : Tinea Versicolor) is a fungus infection of the horny layer, due to the *Microsporon furfur* (Fig. 150). It appears upon the trunk, especially the chest and sternum, as variously sized, irregularly shaped, dry, scaly, yellow-brown coloured patches, easily scraped off.

Treatment.—Lentigo and chloasma are treated with strong mercurial lotions (see above), but are obstinate. The general health requires attention in chloasma For pityriasis versicolor cleanse vigorously with a hard brush and soap and apply ung. sulph. or a lotion of sodium hyposulphite (gr. 60 to the ounce). The underclothing must be disinfected.

IV. **Telangiectasis** is a localised dilatation of a skin vessel. It is seen with some skin diseases, such as lupus erythematosus, rosacea, and dermatomyositis. It also occurs with certain diseases of the liver, kidney and heart. **Multiple hereditary telangiectases** occur as a familial and hereditary malady transmitted by male or female to both sexes. There are multiple small nævi which may bleed profusely from mucous membranes. **Schamberg's Disease** shows a fine network of vessels or pin-point purpuric patches, chiefly on feet and legs. The cause is unknown. Later, these are replaced by pigmentation.



FIG. 150.—MICROSPORON FURFUR, the fungus of PITYRIASIS VERSICOLOR, × 50.—Note branching irregular mycelium and constellations of spores. Gram's Stain.

Nævi or Birthmarks are congenital abnormalities in the skin and underlying tissue, in which one element is over or under developed—vessels, pigment, sebaceous glands or horny layer. They are usually seen at birth, sometimes not till childhood or puberty. (*a*) Vascular nævi (angiomata)—(i.) Capillary vessels extending in spidery superficial telangiectases from a central point (*N. Araneus*); (ii.) flat red purplish patches of varied size, due to capillary dilatation, the *Portwine* mark (*N. flammeus*); (iii.) small, slightly raised patches, the so-called " strawberry marks " which may be associated with (iv.) the *cavernous nævus*, a spongy swelling involving both the skin and the subcutaneous tissues.

(*b*) The non-vascular nævi, **Moles**, may be pigmented or non-pigmented. *N. spilus* is a soft raised swelling, usually pigmented. *N. pilosus* is covered with hairs; it may be small or extend over a large area. *N. verrucosus* is often pale, of any size, warty or papillomatous in appearance; sometimes occurring in long streaks down a limb: *N. unius lateralis.* Nævi may also be due to dilatation of the *lymphatics*

Prognosis.—Most of the vascular nævi respond to treatment; they are not premalignant. Moles, especially when bluish black, may become malignant when irritated by friction or by inadequate methods of destruction. One type, the nævi-carcinoma, is particularly dangerous; metastases rapidly spread, with fatal issue.

Treatment.—*N. araneus* and *telangiectases* are readily destroyed by electrolysis. The *portwine mark* has had many disappointing methods; recently Thorium X gives good promise—1200 to 2000 units to the c.c. administered in varnish once a month over a long period of time. The *strawberry mark* responds to CO_2 snow; but of recent years it is advocated that no treatment be given, as they usually go spontaneously. *Mixed* and *cavernous* types may be excised, X-rayed or injected with sclerosing agents. Ordinary moles are removed by electrolysis; large verrucose moles are best dealt with by diathermy fulguration or surgery.

§ 653. V. **Purpura** consists of dark, abrupt-edged purple spots due to extravasations of blood into the skin. The patch does not fade on pressure.

There is extravasation of blood through the walls of the capillaries; it is a disease of the capillaries rather than of the blood. Normally the blood platelets (thrombocytes) block any intervals in the endothelial lining of the capillaries, and so a diminished platelet count is often associated with purpura. *Causes:* Purpura used to be classified under four main groups: (*a*) *P. Simplex,* a mild and frequently recurrent type; (*b*) *P. Hæmorrhagica,* severe and often associated with a low blood platelet count (thrombocytopenia); (*c*) *P. Rheumatica,* a variety with pains in the joints, due to small hæmorrhages into them; it has no relation to acute rheumatism; (*d*) *Henoch's purpura,* with bleeding into the intestinal wall, causing swelling, melæna, and abdominal colic (§ 584). A causal grouping is (*a*) *Infective:* Associated with (i) acute specific fevers, hæmorrhagic forms of measles, scarlet fever, etc., in severe types, often fatal. (ii.) Typhus, small-pox, cerebro-spinal fever ("spotted fever") and spirochætosis ictero-hæmorrhagica. (iii.) Septicæmia, especially hæmolytic streptococcal septicæmia; other organisms can act similarly, *e g.,* the malarial parasite and the tubercle bacillus in miliary tuberculosis. (iv.) Malignant endocarditis, partly due to infection but chiefly to minute emboli. (*b*) *Toxic:* (i.) Bacterial toxins, as with a septic focus, especially if a hæmolytic organism is present. (ii.) Endogenous toxins, such as those associated with jaundice, diabetes mellitus, chronic nephritis and gout. Any condition with defective liver function is liable to produce purpura. (iii.) Exogenous poisons, such as snake venom and antitoxic sera. (iv.) Drugs in susceptible people, especially salicylates, antipyrin, phenacetin, cubebs, quinine, iodides, phosphorus, mercury, alcohol, the arsenic, benzol and sulphonamide compounds. (*c*) *Blood diseases,* such as leukæmia, aplastic anæmia, lymphadenoma, pernicious anæmia, and primary thrombocytopenia. In these the platelets in the blood are diminished, but the tendency to purpura is not always greatest when these are lowest. The hæmorrhages may also occur from internal surfaces, and so bleeding from the kidneys, trachea, nose, alimentary tract, as well as retinal hæmorrhage, is common. In the acute forms of leukæmia purpura is marked. In aplastic anæmia the thrombocytes fail to be manufactured by the bone marrow in proportion to its failure to form red and white cells. In lymphadenoma and pernicious anæmia purpura is not a prominent symptom: as the blood regenerates with liver therapy in pernicious anæmia, the purpura ceases as the thrombocytes rise. *Primary thrombocytopenia* has a severe hæmorrhagic tendency; the hæmorrhages may be large and widespread, or may only occur spontaneously from one mucous surface at a time; patients can become exsanguinated in a few hours. A secondary anæmia occurs, proportional to the degree of bleeding, and the thrombocytes are low (*e.g.,* 10,000 as compared with normal 200,000 to 250,000). The tendency to bleeding, sometimes familial, is shown by the *capillary resistance test:* when a ligature is tied round the arm a profuse purpuric rash occurs within five or six minutes below the ligature. The bleeding time is prolonged, but the clotting time is normal. The spleen is rarely enlarged, but splenectomy causes an immediate cessation of hæmorrhage (even during the operation), with a rise in thrombocytes. In familial cases splenectomy usually fails. (*d*) *Cachectic conditions, e.g.,* marasmus, carcinomatosis, old age and advanced tuberculosis. Advanced disease of the liver comes under this category. (*e*) *Senile purpura,* after the age of 60, follows minor traumata: recurrent attacks affect the outer sides and extensor surfaces of the forearms, and sometimes the face. (*f*) Purpura is said also to occur in certain *nervous diseases,* syringomyelia, tabes.

The *Diagnosis* of purpura is easy, but difficulty lies in ascertaining its cause.

The *Prognosis* is extremely grave when associated with the specific fevers, or with a high temperature. P. simplex usually results in recovery in a few weeks. P. rheumatica is rarely fatal, though it may last for months or years, and may recur; its complications may be serious.

The *Treatment* is that of the cause. Rest in bed is indicated whilst the rash is still appearing. Liver extract is of value only in cases with pernicious anæmia.

Horse serum or human serum in 10-c.c. doses decreases the hæmorrhagic tendency in most cases, and blood transfusion may be of great value. Splenectomy is indicated in primary thrombocytopenia, if there is no family tendency. With liver disease, Vitamin K should be tried; ascorbic acid (40 to 150 mgms. daily) may help.

VI. **Urticaria Pigmentosa** is a chronic condition with spots of brown pigmentation; when rubbed, these develop wheals. The disease starts in early childhood; it may cease spontaneously about puberty, but it more frequently continues for many years.

VII. **Carotinæmia.**—A yellow pigment (carotin) may occur on the palms and naso-labial folds, even the whole body in marked cases, but not on the sclera (which distinguishes it from jaundice). It is caused by excess of carrots and other lipochromic foods.

VIII. **Xeroderma Pigmentosum** is a rare disease of a chronic progressive character starting on the face in early childhood, often in members of the same family, and marked by small dark freckles, with subsequent atrophy and contraction of the skin, and telangiectases. There is a tendency to malignant new growth, both in the skin and the internal organs. The distribution is universal; the contraction gives rise to eversion of the eyelids and other orifices. It usually terminates in death before the age of twenty-two. The only treatment is protection from light.

IX. **Xanthoma** (Synonym: Xanthelasma) is of gradual onset, with yellowish flattened patches which usually occur on the eyelids; or as nodular deposits widespread on limbs or trunk, varying in size from a millet-seed to a bean, or larger (cp. § 647). It is associated with disordered fat metabolism and glycosuria, hence often with cholesterol excess. *Xanthoma diabeticorum* shows a rapid onset of red papules with yellow tops, occurring chiefly on the buttocks, knees and elbows. Diathermy fulguration and electrolysis remove the lesions; or they may be excised. In diabetic cases insulin and suitable diet succeed.

X. **Morphœa Nigra** and **Morphœa Alba** are names which denote patches of localised scleroderma (§ 651), attended by excess or deficiency of pigment. With scleroderma pigmentation may be widespread.

XI. **Ochronosis** is characterised by blackening of the cartilages (visible in the ears), ligaments and fibrous tissue, and by pigmentation. The sclerotics and extensive areas of the skin may show black pigmented patches. There may be arthritis, and alkaptonuria (§ 386); in the acquired type there is carboluria, due to prolonged use of phenol.

XII. Leprosy (§ 647).—Patches of pigment and white spots may occur in the early stage of anæsthetic leprosy, and dark spots occur, especially on the face, in the early stage of nodular leprosy.

XIII. Von Recklinghausen's disease is dealt with in §§ 647 and 803.

XIV. The **Mongolian spot** is a blue nævus, a congenital condition found on the lower sacral region in dark races. It usually disappears before the age of five. **Blue Nævi** are multiple and persist to adult life.

GROUP X. DISORDERS OF THE SWEAT

§ **654. Anidrosis,** or deficient perspiration, occurs with hypothyroidism, the senile skin and scaly skin diseases. **Hyperidrosis** is an excessive secretion of the perspiration. *General* hyperidrosis occurs with excessive warmth, deficient ventilation, low vascular tone, the crisis in fevers, malaria, tuberculosis, acute rheumatism. After hot or stimulating foods it is usually more localised, on the face or brow. *Localised* hyperidrosis affects chiefly the feet, palms, axillæ and the ano-genital area; when mixed with the fatty acids of the sebaceous secretion it gives off a pungent odour. *Unilateral* hyperidrosis occurs as a reflex through cholinergic nerve fibres, and hence may be seen with pneumonia, aneurysm and such nervous conditions, as peripheral facial paralysis.

Bromidrosis is offensive sweat, due to keratin decomposing with staphylococcal growth in alkaline perspiration. When affecting the feet or axillæ the odour renders the person disagreeable to his companions.

Chromidrosis is coloured perspiration. This can be caused by an external dye; by lepothrix (Fig. 156) of the pubis or axillæ causing red or yellow discoloration; or by B. pyocyaneus producing a blue tint. Red sweat is often due to blood exuding.

The *Treatment* of hyperidrosis consists in using 10 per cent. aluminium chloride or 1 to 5 per cent. sod. hexametaphosphate in water, dabbed on several times a day. Atropin gr. $\frac{1}{120}$–$\frac{1}{60}$ may be given hypodermically. For bromidrosis, especially of the feet, the stockings should be changed several times a day, and put into a saturated solution of boracic acid before being used again. Dusting powders relieve the slighter forms. Good applications are: chromic acid (5 per cent.), pure formalin used for four successive days a month, and diachylon plaster. If glycerin or a glucose solution is painted over, an acid reaction is produced, and no odorous decomposition occurs. In severe hyperidrosis, X-rays succeed.

GROUP XI. DISEASES OF THE SCALP AND HAIR

§ 655. In a volume on general medicine it is impossible to consider in detail all the diseases of the scalp and hair.[1] Those commonly met with will be briefly summarised.

I. Ringworm.
II. Favus.
III. Pityriasis.
IV. Alopecia.
V. Canities.
VI. Pediculosis Capitis.
VII. Hypertrichosis.
VIII. Trichoptylosis.
IX. Trichorrexis Nodosa.
X. Lepothrix.
XI. Monilethrix.
XII. Trichotillomania.

I. **Ringworm** (Synonyms: Trichophytosis Capitis, Tinea Tonsurans) is due to the invasion of the hair by a fungus (see Figs. 151, 152.) The common form is a microsporon fungus of human origin, seen in children. It can spread, when untreated, over most of the scalp. It starts as an insignificant, semi-bald, pink patch, usually overlooked, and when first seen by the physician is a white, scaly, circular patch on the scalp of children, with *broken hairs*. A few patches may occur on the glabrous skin. In another common form there are usually several patches, and often discoid or annular red lesions on the smooth skin; this is of animal origin. Adults may be infected, and recurrence from domestic pets is frequent. A trichophyton animal infection shows severe inflammatory reaction: kerion on the scalp, sycosis on the beard (§ 644). A rare form, "black dot" ringworm, looks like alopecia areata, but has black dots, which are infected hairs broken off at skin level.

Diagnosis.—The broken hair stumps are characteristic. Dabbing chloroform over the part reveals the diseased hairs, looking whitened like hoar frost. The presence of fungi is seen when broken hairs and scales are placed on a slide with a drop of liquor potassæ, and examined under a microscope (see Figs. 151, 152). Choose only infected hairs; "black dots" may have to be dug out with a needle or a comedo extractor. A culture must always be made; on that report depends the choice of

[1] See *The Hair and Scalp*, by Agnes Savill. Edward Arnold. 1951.

treatment. Wood's light reveals a greenish beaded fluorescence with *M. audouini*. Trichophyton infections and animal fungi do not fluoresce. Whether large or small spored, they cause an inflammatory reaction and often invade the smooth skin.

Etiology.—Infected hairs and scales are carried by brushes, combs, pillows, linings of hats. Ringworm therefore spreads rapidly in families,



FIG. 151.—SMALL-SPORED RINGWORM (Microsporon Audouini).

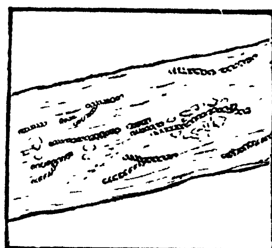


FIG. 152.—LARGE-SPORED RINGWORM (Megalosporon). — Mycelium within the hair showing dichotomous chain. Drawn by Dr. I. Muende. × 400.

schools and institutions. Dogs, cats, horses and cattle are unsuspected sources of infection in both children and adults. Sabouraud's work on fungi has recently been modified by Emmons. The main genera are microsporon and trichophyton. *M. audouini* is the chief fungus of human origin found in children under fourteen. *M. canis* is also now very common in children and adults, being couveyed from domestic pets. Trichophyton fungi may invade the hair, surround it, or be present both in and outside the shaft; endothrix, ectothrix and endo-ectothrix.

Prognosis.—*M. audouini* usually cures spontaneously at puberty. *M. canis* does not itself last long, but re-infection from apparently healthy pets is usual. Forms with severe inflammatory reactions, such as kerion, tend to spontaneous cure.

Treatment.—The head should be shaved every ten days, and a linen cap worn which can be renewed every two or three days. Epilation is necessary. When the disease is not widespread, an efficacious lotion is: picric acid, gr. 7, camphor and rectified spirit, āā fl. oz. ½. This is painted on twice daily; after three weeks the hairs are loosened and can be gently pulled out. *M. audouini* occurring long before puberty requires epilation by X-ray if a skilled radiologist is available. For small patches near puberty and for *M. canis* infection, many kinds of fungicidal ointments are used, such as ung. hyd. ammon. chlor. 5 per cent., or iodine 1 per cent.

or sulphur 5 to 10 per cent. New fungicides are on trial, such as undecylenic acid in a base which penetrates to the root of the follicle. As inflamed lesions tend to spontaneous cure, use mild antiseptics and soothing applications; gently pull out the infected hairs. Watch and prevent re-infection from pet animals.

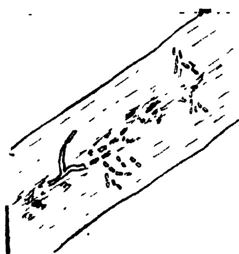


FIG. 153.—FAVUS FUNGUS.—Groups of irregular-sized spores within the hair; mycelium irregular in thickness and arrangement. Drawn by Dr. I. Muende. × 450.

Treatment by a single dose of thallium acetate is little used now. After both X-ray and thallium, till the hair falls, the scalp must be treated daily with a fungicide, such as 10 per cent. sulphur ointment, and iodine on alternate days. As the hair begins to grow again, great care must be taken that no diseased stump is left which could re-infect the new hair.

II. **Favus** occurs on the head and the body. It is rare in England. The characteristic irregular yellow crusts, with yellow, cup-shaped tops (scutula), and the mousy smell, render the diagnosis simple. The microscope reveals the spores and the mycelium of the *Trichophyton schonleinii* (Fig. 153). The culture is diagnostic. The disease develops slowly, is accompanied by redness, and leaves atrophic scars. It is less contagious than ringworm, but more intractable. It may spread to the body. Epilation is necessary.

III. **Pityriasis Sicca** (dandruff) used to be called seborrhœa sicca, from a mistaken idea that it was due to dried or altered oily secretion. Dandruff occurs in patches or generalised over the scalp, as white, dry scales between which the bottle bacillus grows freely. When this happens in those of the "seborrhœic diathesis," it is complicated by oily seborrhœa, with loss of hair, chiefly on the vertex and temples. Or there may ensue a degree of exudation, leading to the formation of yellow crusts—the *pityriasis steatoides* of Sabouraud. Or reaction occurs, a little serum exudes with staphylococcal invasion, and circinate erythema develops—*pityriasis circinata* (usually called seborrhœic dermatitis, § 627). In many cases there is a temporary loss of hair.

The *treatment* consists in washing the head once or twice a week with equal parts of soft soap and spirit, and rubbing in every night a lotion or ointment containing mercury, tar or sulphur (F. 104 to 106). Treatment must be prolonged, and recurrence is usual.

IV. **Alopecia** (Baldness) may be congenital or acquired, partial or complete, diffuse or in patches. Any bald patch should be examined to find if the skin is (A) normal or (B) atrophied.

A. The following causes should be investigated :—

1. Alopecia areata.
2. Seborrhœa oleosa, especially common in men.
3. Senile baldness in its early stage.
4. Impetigo and other superficial pustular affections.
5. Secondary syphilis.
6. Baldness over tumours, nævi, moles, etc.
7. Rare forms of ringworm.
8. Trichotillomania.
9. Monilethrix.

B. If the skin of a bald patch shows ATROPHY or SCARRING, consider :—

1. Lupus erythematosus.
2. Traumatism.
3. Folliculitis and other pustular conditions such as Kerion, Boils and Carbuncles.
4. Alopecia after the menopause.
5. Morphœa.
6. Herpes zoster.
7. Favus.
8. Pseudo-pelade.
9. Folliculitis decalvans.
10. Lichen spinulosus and plano-pilaris.
11. Ulerythema ophryogenes.
12. Lupus vulgaris and other nodular conditions.
13. Radiodermatitis.

C. If there is an EXTENSIVE area of BALDNESS, it is probably due to one of the following causes :—

1. Alopecia areata, at a late stage.
2. The advanced stage of seborrhœa oleosa, the usual baldness of men.
3. Baldness after X-ray or thallium (therapeutic epilation dose).
4. Diffuse hairfall after fevers, operation, shock, myxœdema and other causes of lowered health.
5. Congenital atrichia.
6. Ichthyosis.
7. Ichthyosis follicularis.
8. The late stage of several of the causes of baldness with atrophy.

A. *A common cause of* BALD PATCHES WITH NORMAL SKIN *is* ALOPECIA AREATA.

1. **Alopecia Areata** is a form of baldness occurring in circular patches which are smooth and white. Each patch slowly increases peripherally, and at the margin short diseased hairs may be seen, which are so characteristic as to enable us to identify the disease. The free end is of normal thickness, but presents a ragged fracture where the hair has been broken off; from this point the shaft gradually becomes thinner towards the root, which is extremely atrophied. Thus it resembles a mark of exclamation (!). The disease runs a protracted course, lasting, especially if untreated, for years. In course of time a few downy hairs begin to grow, white at first, but gradually becoming coloured. The disease is not contagious. The sympathetic endocrine balance is disturbed and permits the effect of some causal agent, such as reflex irritation from eyestrain, the teeth or nasopharynx (Jacquet), or infective foci in the tonsils or sinuses (Barber, Leslie Roberts). Any infection, especially syphilis (Sabouraud), any shock or worry, may upset the endocrine balance and precipitate an attack. In severe cases, eyebrows, eyelashes, and all the hairs on the body may fall (Alopecia totalis et universalis).

Treatment.—The patient must be warned that perseverance is necessary for a long period of time. The cause must be sought for and removed. For mild cases sulphur ointment and a weekly painting with iodine soon restores hair growth. Local and general applications of ultra-violet light are most useful. In obstinate cases success is often obtained with liquor epispasticus, or strong erythema doses with the Kromayer lamp. Many forms of counter-irritation can be used. Thyroid aids many cases; pituitary extracts have been disappointing.

The other causes of bald patches without atrophy may be briefly summarised:

2. *Seborrhœa oleosa* begins in young men, with thinning of the hair of the temples and vertex, accompanied often by oiliness. It is the usual cause of baldness in men. *Treatment* is difficult and must be prolonged. Alkalies should be taken when the urine is very acid. Œstrin therapy aids some, but must be carefully watched. 3. In SENILE BALDNESS the natural loss of hair on the vertex may in extreme age show some atrophy. 4. The bald patches seen after impetigo and other SUPERFICIAL PUSTULES rarely last long. The history aids the diagnosis. 5. In SECONDARY SYPHILIS there is rapid loss of hair in small patches over all the scalp, causing a characteristic moth-eaten appearance. A similar alopecia may occur with neuro-syphilis. The history and other symptoms aid diagnosis, and regrowth returns with antisyphilitic remedies.

B. *The bald patches show* ATROPHY *or* SCARRING. When the chief eruption occurs on the body, the causes are described in other sections, under the chief symptoms.

In *favus* the yellow crusts, mousy smell and reddened scars lead one to examine the hairs, when the fungus is found. *Pseudo-pelade, folliculitis decalvans, ulerythema ophryogenes* and *lichen of the scalp,* are such rare forms of atrophied bald patches that they are distinguished with difficulty even by the dermatologist. Their course is chronic and slow; when they have continued for years, the entire scalp may be bald and cicatricial. *Lupus vulgaris* is very rare on the scalp and has characteristic nodules (§ 646). *Sarcoid* is a rare disease with nodules of varying size which spread with circinate margin; they may be absorbed or ulcerate, leaving fine scars (§ 647). *Gummata:* see § 646. In all cases with atrophy the baldness is permanent.

C. The AREA of BALDNESS IS EXTENSIVE.

1. In alopecia totalis there is a history of alopecia areata, with loss of hair in patches which have joined and produced baldness over the whole scalp; the skin is smooth and glossy. 2. Seborrhœa oleosa may cause extensive baldness by the age of thirty; a rim of thick hair is left on the occiput and just over the ears. 3. In the baldness due to an epilation dose of thallium or X-ray, the history renders the diagnosis clear. The hair should begin to return in six weeks. 4. The diffuse hairfall after fevers, childbirth, operation or shock, rarely leads to complete baldness. The *prognosis* is excellent if treatment is given early and carried out with perseverance. In myxœdema and other conditions with chronic lowered health the outlook is good, provided the cause can be removed. One cause of baldness, which may be complete, is congenital syphilis. This usually begins in childhood, and owing to the absence of other signs the diagnosis may be unsuspected. 5. In congenital atrichia the patient has been born without hair, or there may have been a slight down. The prognosis is serious; the hair may be induced to grow a little, but falls again. Occasionally it grows longer at puberty. 6. Ichthyosis of the head is known by the scaliness of the body; it usually begins in childhood. Keratosis affects the hair follicles of the scalp, eyebrows and eyelashes. 7. Ichthyosis

follicularis is very rare. 8. As mentioned in Group B, various causes of cicatricial alopecia may in the course of years involve a large part of the scalp.

Treatment of hairfall.—In order to bring about regrowth of hair, whatever the cause of the baldness, the circulation must be encouraged by local stimulation such as massage, ultra-violet light, and the persevering use of lotions or ointments with pilocarpine, sulphur, or rubefacients such as cantharides, ammonia and acetic acid. Where there is atrophy of the skin and hair follicles, no hair can grow again. The general health must not be neglected; often small doses of thyroid are effective, even in the absence of hypothyroidism. There is gathering evidence that follicular keratosis and ulerythema ophryogenes respond to intensive doses of vitamin A. Antisyphilitic treatment is given when indicated.

V. **Canities,** grey or white hair, is usually an evidence of advancing years. In other cases it may arise in association with overwork, sudden or prolonged grief, defective general health, or neuralgia. *Premature* grey hair, beginning before thirty, is usually hereditary. *Treatment* is directed to the general health. The scalp should be examined for any local disease which, though not causal, may expedite the loss of colour. Thyroid when otherwise indicated, does good. Provided that there is no septic focus, and that the endocrine and intestinal functions are normal, para-aminobenzoic acid (a constituent of the vitamin B complex) restores colour. It should be taken in divided doses, 100 mg. with meals three or four times a day (B. Sieve).

VI. **Pediculosis Capitis** presents the following features: (1) The pediculi (Fig. 154); (2) white specks on the hairs (the eggs or "nits"), which cannot be pulled off, by which they are distinguished from dandruff (Fig. 155); and (3) itching of the scalp. If the condition is untreated, there results pustulation with formation of thick crusts, matting of the hair, and enlargement of the occipital and even the cervical glands. Lethane in white mineral oil (semprolia) and 5 per cent. D.D.T. have replaced earlier

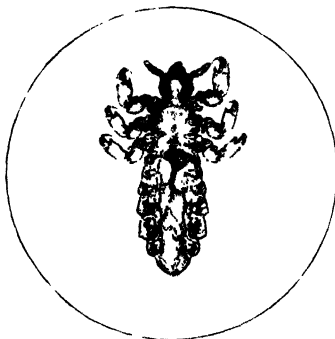


FIG. 154.—PEDICULUS CAPITIS × 10.—It differs from the pediculus corporis only in being shorter, and in its thorax and abdomen being more nearly equal in size (see § 620).

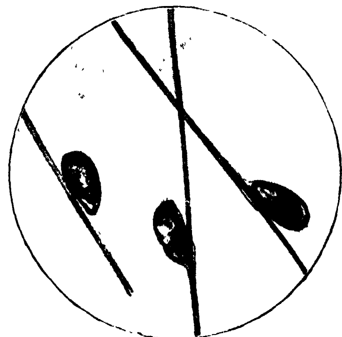


FIG. 155.—NITS (eggs) of PEDICULUS CAPITIS on hairs (magnified).

methods of treatment. Comb the hair thoroughly with a fine comb. The nits are dissolved by washing the hair with acetic acid or benzole.

VII. **Hypertrichosis** (Synonym: Hirsuties) is a growth of hair of the male type and distribution in women. It often occurs at the menopause; earlier in life it may be associated with adrenal hyperplasia or tumour, Cushing's syndrome, or with arrhenoblastoma of the ovary (which is rare). Hair grows on the face, arms, legs, abdomen or chest. *Treatment* consists of the removal by diathermy or electrolysis; this is expert work. Depilatory pastes, pumice stone, the razor and wax do not prevent regrowth but are valuable for extensive cases. X-ray treatment is efficacious, but is followed many years later by telangiectases and atrophy. Unilateral adrenalectomy in suitable cases of **virilism** enables the hair to be pulled out with little pain. Ovarian therapy is on trial.

VIII. **Trichoptylosis,** or splitting of the shafts, especially near the ends of the hairs, is often seen associated with deranged general health and local scalp disease.

IX. In **Trichorrhexis Nodosa** spindle-shaped grey swellings appear upon the hairs, due to localised splitting of the shafts. Microscopically, the nodes resemble two opposing brooms. It is due to traumatism, alkalies or dyes acting upon dry hair. It is often associated with trichoptylosis.

X. **Lepothrix** (Synonyms: Mycosis Axillaris, Trichomycosis Nodosa) is a disease affecting the hairs of the axillæ and genitals. The hairs are dry and knotty, due to adherent small yellowish concretions which may affect the whole length of the hair,

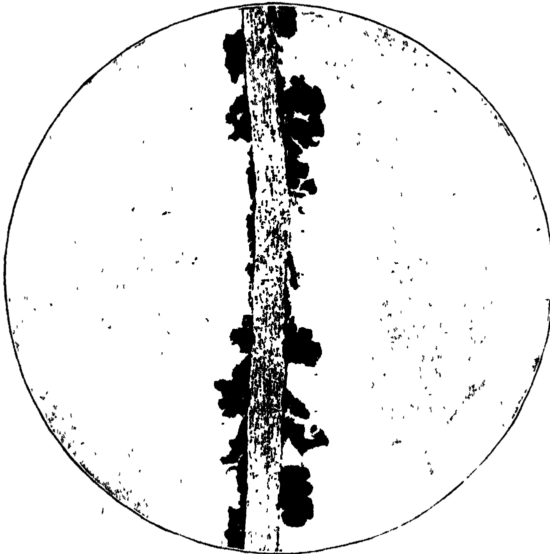


FIG. 156.—LEPOTHRIX.

but not the follicle, or may occur as separate nodules on a hair. Bacilli are found in these concretions, and the hair may be split longitudinally (Fig. 156). The sweat colours the garments yellow or brown (chromidrosis). Cleanliness and avoidance of tight-fitting clothes remedies the condition.

XI. In **Monilethrix** the hair shaft is dilated and narrowed alternately. The follicles, with short broken-off hairs, resemble Keratosis pilaris, and in a child the condition may at first sight be mistaken for ringworm. Microscopic examination shows the beaded state of the hair shaft.

XII. **Trichotillomania** is a nervous habit in which the patient continually pulls out the hairs from some region ; a bald area is thus caused.

§ **656. General Remarks on Treatment.**—Arsenic, bismuth, mercury and iodides in syphilis, iodides in other granulomata, antimony in leishmaniasis, and calciferol in lupus vulgaris are specific remedies. Ephedrine and adrenalin are used in acute urticarial conditions. When penicillin-sensitive staphylococci or streptococci are present, penicillin is particularly useful. The sulphonamide drugs are of use by mouth and *in situ*: severe general and cutaneous reactions have followed 0·5 G. thrice and 1 G. twice daily ; in other cases an eruption has followed : erythematous, morbilliform, purpuric or œdematous, especially in parts exposed to sunlight.

It is not sufficient for purposes of treatment to diagnose a case as eczema, psoriasis, lupus, etc. For *local* treatment, we must recognise *the stage of the disease* and the *precise pathological process* before us. An ointment which would cure a chronic eczema may greatly aggravate an acute weeping one. Treatment therefore depends not so much upon the name which we give to an eruption, as upon the condition of congestion, swelling, scaling, thickening, discharge, itching, etc. The *method* of application of a remedy is of as much importance as its composition. The *idiosyncrasy* of a patient and the susceptibility of his skin to various remedies must be noted ; what irritates the skin of one person is inert on that of another.

ANTIPRURITIC *lotions* are dabbed on the unbroken surface, and frequently renewed. Those commonly used are: phenol 2 to 4 per cent., liq. picis carb. 10 per cent., camphor and menthol $\frac{1}{2}$ to 4 per cent., chloretone $\frac{1}{2}$ per cent., chloral hydrate 2 per cent. For acute, congested and weeping surfaces, lotions with subacetate of lead, zinc and calamine are dabbed on freely, and allowed to dry. In chronic thickened eruptions, use *ointments* well rubbed in and covered with gauze or thin linen. In preference to lint and bandages, thin stockings with feet cut off can be used to keep in position dressings with ointments. Where there is oozing, lotions and *pastes* aid absorption of serum, whereas greasy ointments confine the exudation.

CRUSTS forming over regions soaked in serous exudation and pus must be removed before using remedies. Remove by applying lint soaked in oil, or a starch poultice. One teaspoonful of boric acid, four tablespoonfuls of cold-water starch (Orlando Jones's or Colman's rice starch) ; add cold water to make the consistency of cream. Pour on a pint of boiling water ; stir constantly, until the starch bursts and a translucent jelly forms. Or pour the cream into the boiling water. When the jelly is cold, spread on cloth in a layer about half an inch thick ; cover with muslin and apply to the part. The poultice can be used four times a day.

(1) *Sedatives* and *astringents* reduce hyperæmia, check exudation, and allay burning and throbbing. Those most used are zinc oxide and carbonate, lead, bismuth and ichthyol. When the acute stage has passed, add mercury and weak tar. Friar's balsam is useful for fissures near mucous orifices.

(2) *Reducing agents* are used for thickened, chronic eruptions. Named in order of strength, these are: wood and coal tar, carbolic, benzoic and salicylic acids, mercury, sulphur, resorcin and chrysarobin. Tar (strong or crude coal tar) is used in infantile eczema and certain types of thickened dermatitis ; never over pustules. Over 6 per cent. salicylic acid is a keratolytic ; it removes thickened horny layers and scales when used in ointment, collodion or a plaster.

(3) *Caustics* have a still more powerful action in removing thickened epidermis and scales. Liquor potassæ and soft soap are mild caustics used for this purpose before applying ointment. Strong caustics are pure carbolic, nitric and trichloracetic acids, and acid nitrate of mercury.

(4) *Dyes*, such as a 2 per cent. aqueous solution of gentian violet, are much employed, especially for staphylococcal infection, such as occurs with impetigo and eczematoid infective dermatitis. Paint over twice a week and cover with an inert powder.

(5) *Penicillin* is used for skin diseases due to penicillin-sensitive organisms, such as staphylococci and some strains of streptococci. Conditions responding to penicillin

are carbuncles, boils, the anærobic actinomycosis fungi, intertrigo, fissures and abscesses.

Superficial lesions are treated with a spray (500 units per c.c.), left uncovered, and applied thrice daily; or with a cream (200 to 1,000 units per G.) spread on with a sterilised blade two or three times a day. Most creams contain Lanette Wax SX, to which some skins are sensitive. Alternative bases are given in the British Pharmacopœia. Penicillin must be kept in a cool place, preferably a refrigerator. When lesions do not respond within a week, do not continue the application, lest penicillin resistant organisms form. *Deep-seated lesions* require large doses; the dosage is under continual revision.

PROTECTIVE MEASURES.—The *paste*, an ointment made up with a large proportion of powder (*e.g.*, F. 75), is applied spread in a thick layer on butter muslin which is bound firmly on the part. It absorbs serous exudation. *Zinc Gelatine* melted in a warm bath, then painted over the diseased skin, gives: (i.) gentle compression and support, as in hypostatic congestion; (ii.) protection from air or friction while allowing healthy growth below; (iii.) means of applying remedies. Varieties are elastoplast, visco-paste, varicosan. Remedial drugs may also be added to a basis of gelatine, tragacanth or collodion.

Treatment by X-rays, diathermy, high-frequency current, ultra-violet rays, and carbonic acid snow requires special training in the use of the apparatus.

General Treatment.—A skin disease is an external expression of an internal disorder almost as often as it is a reaction to an external irritant. Hence certain eruptions clear up on removal of a septic focus, metabolic errors such as gout, constipation or other cause of toxæmia, or the source of protein sensitisation. Patients with acute and wide-spread eruptions should be kept in bed. When the individual's threshold of resistance is lowered, or he has (in other words) become sensitised to a poison, the removal of one septic focus may give only temporary relief, because another is soon formed. Desensitisation methods are: *Autohæmotherapy*: 5 to 10 c.c. of blood are withdrawn from the patient's vein and at once injected into the muscles of the buttock; this can be repeated every five days for five or six times. *Autoserum therapy* is used in dermatitis herpetiformis and some forms of eczema. Inject into a vein, muscle or under the skin, the patient's own serum. 40 to 200 c.c. blood are withdrawn into a centrifuge tube and allowed to clot; the serum is centrifuged and injected in doses of 20 to 60 c.c. every five to ten days. *Protein therapy* is of value, *e.g.*, cow's milk, boiled, cooled and injected intramuscularly (5 to 10 c.c.) every four to seven days: typhoid and colon bacilli and peptone are also used. Marked local and general reactions may follow. *Antihistamine treatment* is indicated for certain conditions of prurigo and urticaria; see § 609. *Vaccines* are especially valuable in cases of local staphylococcal infection; they must be given in doses which do not cause severe reaction, especially in patients who are not being kept in bed. In some cases, *e.g.*, sycosis, they are more effective when given intradermally. Sometimes a course of colonic irrigation brings about rapid improvement in a skin disease which has resisted dietetic and other measures for the restoration of a normal flora; in other cases cure follows a long course of vaccines made from extracted diseased teeth, or from the stools. The health as a whole must be considered, and all forms of treatment here have their sphere—adequate mental and physical repose, endocrine preparations, diet, mountain air, massage, heliotherapy and psychotherapy.

CHAPTER XIX

THE NERVOUS SYSTEM

THE student will find the arrangement of this somewhat lengthy chapter conforming to the method pursued throughout the book. A brief account of the anatomy and physiology of the nervous system is followed by:

At the outset certain *Physiological Laws* applied to disease processes in the nervous system, and first formulated by Hughlings Jackson, may be stated:

(1) The nervous system may be visualised as consisting of a number of physiological levels, the functions of the lowest or spinal level (most automatic) being comparatively well-organised at birth, the highest or cortical (most voluntary) continually organising throughout life. The higher levels inhibit or control the lower levels.

(2) In disease the functions first acquired in development are the last to be destroyed. Thus when speech function is destroyed by a cortical lesion, gesture which is acquired chronologically before speech invariably remains. In acquired dementia the memory for recent events has gone, while the patient still remembers events of childhood.

(3) A destructive nervous lesion causes negative and positive symptoms. For example a capsular hæmorrhage will cause loss or impairment of voluntary movement dependent on destruction of the pyramidal tract—the *negative* symptom. We also observe new phenomena, not present before the onset of the lesion, muscular hypertonus and an extensor plantar response. These are *positive* symptoms due to release of intact mid-brain mechanisms which have escaped from pyramidal control.

(4) Chronic lesions of the nervous system at first irritate and later paralyse function. Thus a slowly growing meningeal tumour compressing the motor cortex will at first cause a focal Jacksonian convulsion (Irritative sign), later a monoplegia (Paralytic sign).

(5) Nerve-cells once destroyed never regenerate. Compensation occurs in cases of partial destruction, but this is never absolute.

(6) The more rapid the destruction the greater the dissolution. Acute lesions (*e.g.*, a blow on the head) produce at first widespread loss of function, complete loss of consciousness, flaccid paralysis and incontinence. These "shock phenomena" are usually transient.

APPLIED PHYSIOLOGICAL ANATOMY OF THE NERVOUS SYSTEM.

§ 667. The central nervous system consists of vast numbers of *Neurones.* A neurone is a nerve cell with its dendrites and axon. The *nerve-cells* are found in the grey matter of the cortex, basal ganglia and nuclei, the central grey matter of the spinal cord and posterior root ganglia. The *axons* are collected into bundles or tracts and run mostly in the white matter and peripheral nerves. The *nervous impulse* travels at different rates in different nerves, and may travel in both directions in a single nerve fibre. A *synapse* between a nerve-fibre and another nerve-cell will allow an impulse to pass in one direction only.

The activity of the cortical nerve cells can be studied with the Electro-encephalograph, by means of which cortical action currents are led off by electrodes placed on the intact scalp, amplified by wireless valves and recorded by a cathode-ray oscillograph. Waves of a frequency of 10 per second and 0·5 to 1·0 millivolt amplitude may be observed when the patient is at rest with the eyes closed. These waves are called alpha waves, and the ten-cycle rhythm is known as the Berger rhythm, after its discoverer (Fig. 174). Beta waves of a frequency of 25 to 50 per second are also obtained simultaneously from various parts of the cortex in normal persons. These waves can be inhibited if the individual concentrates on an arithmetical problem, or when he opens his eyes. Abnormal brain potentials accompany pathological cortical processes. In epileptics, sub-liminal discharges from cortical foci can sometimes be recorded in this way. In intracranial tumour slow waves can be picked up by the electrodes and the abnormal focus localised to a restricted area of cortex.

Neurones are extremely sensitive to oxygen-want (anoxæmia) and many nervous lesions produce their effects by alterations in the blood supply. Cerebral and spinal tumours produce their effects not so much by distortion or disruption of nerve-tracts as by *local anoxæmia.* In these cases, rapid recovery of function may follow simple decompression, even when it is impossible to remove the tumour. Certain inorganic poisons or organic toxins exert their effects selectively by picking out particular groups of neurones or muscles, *e.g.*, lead commonly affects the neurones proceeding from the sixth cervical segment, producing wrist-drop; diphtheria picks out the ciliary and the bulbar muscles. This *selective action* is also observed in infection with viruses, the virus of acute poliomyelitis affecting the anterior horn cells, that of epidemic encephalitis affecting the oculo-motor nuclei. An acute toxic lesion (*e.g.*, polyneuritis) may produce widespread recoverable paralysis without demonstrable structural changes in the neurones. Recovery, after actual destruction, is only possible in peripheral nerve lesions.

§ 668. **The Cerebral Cortex.**—The cortical MOTOR AREAS lie in the pre-central gyrus and the posterior parts of the frontal convolutions which are immediately anterior. *Here are represented not muscles but movements.* Topographically, the movements of the foot are represented at the upper end of the motor cortex; those of the leg, trunk, upper limb, hand, neck, face, lips and tongue, in that order from above downwards (Fig. 157). It will be noticed that the complicated movements of the face and hand have a relatively large cortical representation, a good example of adaptation of structure to function. Irritative lesions of these areas cause focal *Jacksonian fits.* Jacksonian fits commonly commence in one of three foci—(*a*) the thumb and index finger, (*b*) the angle of the mouth, or (*c*) the hallux, and are followed by the "paralytic sign," a transient monoplegia. Such a fit begins with clonic convulsions of one of these foci and (*a*) may remain local or, (*b*) more commonly, spreads in an orderly march in accordance with the cortical representation of the parts affected, so that face, arm and leg on one side of the body are involved and eventually the whole of the body musculature. So long as the convulsion is localised consciousness may be preserved, but when it is generalised consciousness is lost. At the posterior end of the second frontal gyrus is an area for the *conjugate movement of the head and eyes* to the contralateral side (oculogyric area). An area at the posterior end of the first

and second frontal gyri is connected with the *grasp reflex*. Stroking the palm of the contralateral hand or stretching the flexors of the fingers produces a tonic closure of the fist. Lesions extending deeply into the substance of the motor area produce a *monoplegia* or *hemiplegia*.

FIG. 157.—DIAGRAM OF THE CORTICAL AREAS (lateral aspect of hemisphere). W. in diagram = Word area.

The SENSORY AREAS lie in the posterior lip of the Rolandic fissure and extend backwards to include the superior parietal, supra-marginal, and part of the angular gyri. Stimulation of these areas will produce, as the irritative sign, a sensory Jacksonian attack, while destruction causes: (1) Loss of ability to estimate the size, shape and consistency of objects held in the contralateral hand (*Astereognosis*), and (2) Impaired ability to localise tactile stimuli (Atopognosis), and to discriminate two simultaneous touches with compass points. Sensibility to pain and temperature is intact, but there is difficulty in appreciating the intensity of stimuli. The defect in spatial recognition leads to inco-ordination of the affected hand and digits.

FIG. 158.—DIAGRAM OF THE CORTICAL AREAS (mesial aspect of hemisphere).

Further back is the VISUAL AREA (Fig. 158), represented in the upper and lower lips of the calcarine fissure on the mesial aspect of the occipital lobe; and extending, on the lateral aspect of the occipital lobe, into the angular gyrus. The right half of each visual field is represented in the left occipital cortex and vice versa.

Macular (*i.e.*, central) vision is represented at the tip of the occipital pole. The upper halves of both peripheral visual fields are represented in the lower lip of the calcarine fissure, the lower halves in the upper lip of this fissure.

Irritative lesions of the visual cortex cause visual Jacksonian attacks in the form of visual hallucinations of hemianopic distribution. In lesions far back on the occipital cortex these hallucinations take the form of moving lights, "sheets of

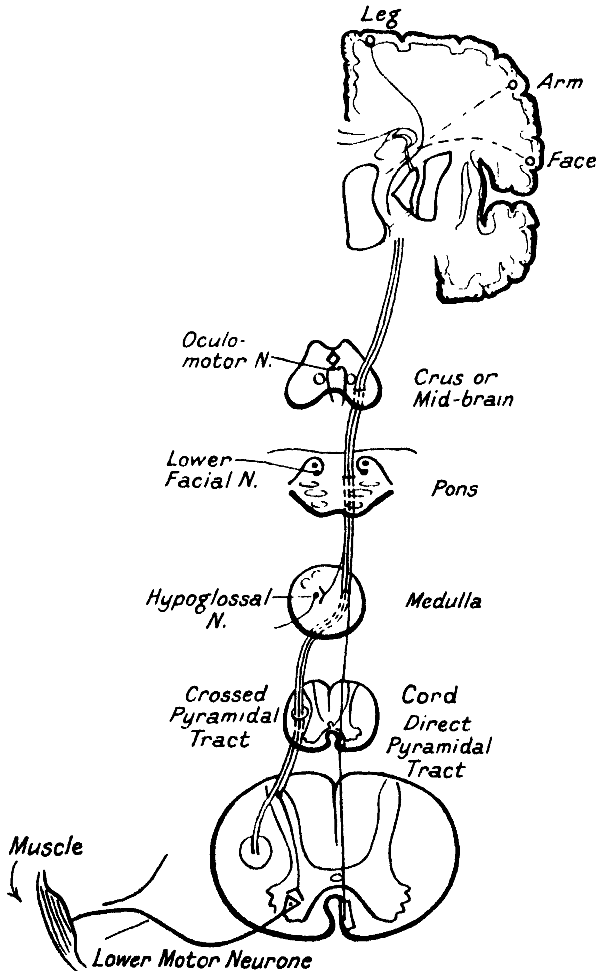


FIG. 159.—DIAGRAM OF THE PYRAMIDAL TRACTS, showing their course through the brain, brain-stem and cord.

flame," etc. With lesions involving the occipital and temporal cortex the visual hallucinations may be more complex, taking the form of scenes, "play-acting." Destructive lesions of the visual area will produce blindness of the crossed halves of each visual field (homonymous hemianopia), while partial lesions produce blindness of homonymous quadrants of the visual fields. Lesions of the angular gyrus cause impairment of stereoscopic vision and failure to recognise objects seen (visual agnosia).

The cortical AUDITORY AREAS are situated in the superior temporal gyrus. The cortical areas for TASTE and SMELL are in the uncinate gyrus on the mesial aspect of the temporal lobe. Irritative lesions of this area cause "uncinate fits," characterised by spitting and champing movements, associated with olfactory and gustatory hallucinations and a transient disturbance of consciousness or "dreamy state." The cortical areas for SPEECH are located in the left cerebral hemisphere, in right-handed individuals. They are situated in the posterior parts of the second and third frontal, the superior temporal, and in the angular gyri. Lesions of these areas cause various types of aphasia (see Fig. 175, § 743).

There are three great systems of projection fibres: the Motor, Afferent, and Visual Tracts. The Motor System consists of three groups of neurones: (1) Pyramidal Neurones, (2) Extra-Pyramidal Neurones, (3) Lower Motor Neurones.

§ 669. **The Pathway for Voluntary Movements.**—All impulses for voluntary movement are transmitted by the Pyramidal Tracts or Upper Motor Neurones. Damage to the pyramidal tract produces (1) *Impairment or loss of volitional movement* from interruption of the conduction of motor impulses. The resulting paralysis is termed a Monoplegia (paresis of one limb), Hemiplegia (paresis of face, arm and leg on one side of the body), Paraplegia (paresis of both lower limbs), or Diplegia (paresis of all four limbs). (2) *Release of extra-pyramidal motor phenomena*, viz., increase of tonus and exaggeration of the tendon reflexes, with the appearance of the extensor type of plantar response.

The Pyramidal Tract or Upper Motor Neurone (Fig. 159) extends from pyramidal cells of the pre-Rolandic cortex to the contralateral anterior horn cells of the spinal cord. From the cortical cells the fibres converge through a fan-shaped radiation, the corona radiata, to the internal capsule, which lies between the lenticular nucleus externally and the caudate nucleus and optic thalamus internally. The motor fibres occupy the genu and portion just anterior to this, and the anterior two-thirds of the posterior limb of the internal capsule. Here the fibres have undergone some rearrangement since leaving the cortex, for the order from before backwards is now face, shoulder, elbow, fingers, trunk, hip, knee and toes. Behind the motor fibres in the posterior limb of the internal capsule are the sensory and auditory fibres, and behind these the visual fibres of the optic radiations (Fig. 160).

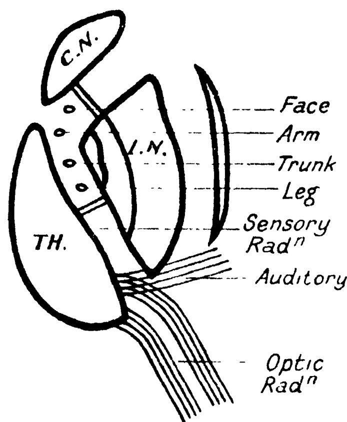


FIG. 160.—DIAGRAM OF INTERNAL CAPSULE.
(Horizontal section through the right internal capsule, showing the position of the different fibre tracts.)

From the internal capsule, the Pyramidal Tract descends through the ventral part of the crus cerebri (near the oculomotor nerve), spreading out a little in the ventrally situated formatio reticularis of the pons (the motor nucleus of the trigeminal is in the middle of the pons, and the abducens and facial nuclei in the lower part of the pons). On reaching the upper part of the medulla, the pyramidal tracts converge as the two ventrally and mesially placed pyramids. Below this, nearly all the fibres decussate to form the Crossed Pyramidal Tract which descends in the opposite lateral column of the cord. The pyramidal fibres do not end directly in the cells of the anterior horn but terminate in the region of the posterior horn,

whence short intermediary neurones connect to the anterior horn cells. A small proportion of pyramidal fibres are continued down the ipsilateral side of the cord as far as the mid-thoracic region lying near the anterior median fissure (the Direct Pyramidal Tract). These fibres eventually decussate to the opposite anterior horn cells.

All the motor nuclei of the cranial nerves receive fibres bilaterally from both pyramidal tracts, except the hypoglossal and that part of the facial nucleus connected with movements of the lower face. These receive fibres from the pyramidal tract of the contralateral side only. From the cells of the anterior horns and the cranial nerve motor nuclei the lower motor neurones arise (Fig. 159).

§ 670. **Pyramidal lesions** may occur at various levels and produce the following clinical symptoms (Fig. 159):

(*a*) CORTICAL LEVEL: Owing to the extensive distribution of motor cells on the cortex a focal lesion will produce a monoplegia which is flaccid. If the lesion extends more deeply, involving many pyramidal fibres, the symptoms will be more widespread and spasticity will be present. Motor Jacksonian fits will occur as the irritative sign. A vertical lesion over both cortical leg areas will cause paraplegia, a paralysis of both lower limbs.

(*b*) INTERNAL CAPSULE: The convergence of the pyramidal fibres here is such that a relatively small lesion will produce a complete hemiplegia. In lesions of the genu the arm is more affected than the leg. In lesions farther back, there is hemianæsthesia and perhaps hemianopia, from involvement of the sensory fibres and optic radiations (Fig. 160).

In **Hemiplegia** (§ 752) there is unilateral loss of voluntary power in the affected limbs and the lower face. The tongue is protruded towards the paralysed side. The muscles of deglutition and mastication, which have bilateral pyramidal innervation from the cortex, usually escape, as do the trunk muscles. "Clasp-knife" rigidity (§ 704) with hypertonus appears in the affected limbs, in the flexors and adductors, so that the limb is held with the arm adducted at the shoulder, flexed at the elbow and wrist, with the forearm slightly pronated. The movements of the fingers and hand are more affected than the proximal movements. In the lower limbs the hypertonus appears in the extensors and adductors, while the movement most affected is dorsiflexion of the foot. The tendon reflexes become exaggerated and ankle- and rectus-clonus may develop, the plantar response is extensor in type and the abdominal reflexes on the corresponding side disappear, the lower abdominal reflexes disappearing before the upper ones. The gait is characteristic, the paralysed limb being dragged round in a semicircle, the toes scraping the floor.

(*c*) LEVEL OF CRUS: A "crossed paralysis" results, involving the oculomotor nerve on the same side as the lesion, and a contralateral hemiplegia (Weber's Syndrome) (Fig. 159).

(*d*) LEVEL OF LOWER PONS: A "crossed paralysis" results, involving the facial and abducens nerves on the same side as the lesion, and a contralateral hemiplegia (Millard-Gubler Syndrome).

(*e*) LEVEL OF SPINAL CORD: A spastic paresis of one or both lower limbs results. Lesions above the fifth cervical segment involve upper as well as lower limbs.

To summarise: **upper motor neurone lesions** produce, on the side affected: (1) Loss of voluntary power. (2) Spasticity of the "clasp-knife" type. (3) Increased tendon reflexes with ankle- and rectus-clonus. (4) Extensor plantar responses with absent abdominal reflexes. (5) Normal electrical reactions in the affected muscles. (6) No muscular wasting.

§ 671. **Striatal Rigidity—Parkinsonism.**—The *corpus striatum* consists of the caudate and lenticular nuclei. From these important nuclei and from other masses of grey matter, notably the *Red Nucleus*, *Substantia Nigra*, and the *Sub-thalamic Body* (Corpus Luysii) of the mid-brain, and the *Vestibular* (*Deiter's*) *Nucleus* in the pons, the EXTRA-PYRAMIDAL MOTOR NEURONES arise and extend by one or more links to the anterior horn cells. The *Cerebellum* and its connections are really part of the

extra-pyramidal motor system, but for convenience these are considered later. It will be seen that the lower motor neurone is thus the "final common path" for both pyramidal and extra-pyramidal fibres (Figs. 159, 161).

The EXTRA-PYRAMIDAL MOTOR SYSTEM has no known connections with the cerebral cortex, and it probably represents a motor system phylogenetically older than the

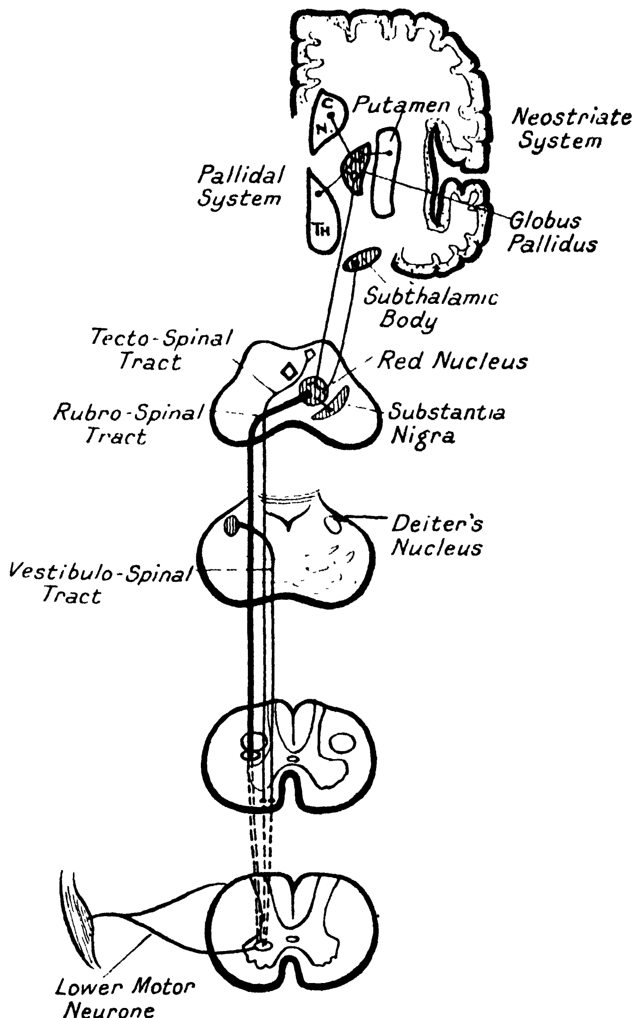


FIG. 161.—DIAGRAM OF THE EXTRA-PYRAMIDAL MOTOR SYSTEM, showing the origin of the extra-pyramidal motor tracts and their termination in the anterior horn cells.

pyramidal system. Lesions of the Extra-Pyramidal Pathways in man produce: (1) *Disturbance of muscle tone*, and (2) *Involuntary movements*, but *no true paralysis*. The reflexes are unaltered and there is no clonus, the plantar response remains of the flexor type, the abdominal reflexes are retained. *The symptom-complex most frequently encountered is Parkinsonism* or paralysis agitans, but hypotonia may also occur, with involuntary movements, athetosis, choreiform movements or tremor.

In **Parkinsonism** (§ 765) there is rigidity of both flexor and extensor muscles, the patient presenting a characteristic mask-like expression, with loss of the swinging movements of the arms on walking. On attempting to move the affected limbs passively, *e.g.*, at the wrist-joint, the examiner will encounter resistance like bending a piece of lead-pipe, or turning a cog-wheel, the so-called "lead-pipe" or "cog-wheel rigidity." The patient assumes an attitude of slight general flexion, the head and neck are bent forwards, the gait is shuffling or gliding, and the arms adducted and slightly flexed at the sides of the trunk (Fig. 4). The fingers are adducted in the "interosseal" attitude. All the movements are slow and restricted. Fine rhythmic tremor appears in the arm, leg, head or lower jaw. The reflexes are unaltered.

The anatomy of the Extra-Pyramidal Motor Neurones is complex (Fig. 161). The lenticular nucleus is subdivided into an external segment, the *putamen*, and a more important internal and smaller part, the *globus pallidus*. The globus pallidus consists of large pyramidal or multipolar cells like ventral horn cells, and receives short afferent axons from the optic thalamus, from the putamen and the caudate nucleus. It gives rise to efferent projection fibres ending in the substantia nigra and the red nucleus of the same side of the mid-brain. The whole corpus striatum is entirely unconnected with the cortex, and efferent fibres proceed only from the globus pallidus. These efferents do not pass directly to the ventral horn cells but link up with the red nucleus and vestibular and cerebellar tracts. There are three chief *Extra-Pyramidal Tracts*:

(1) *The Rubro-Spinal:* The fibres arise in the red nucleus, which is situated in the mid-brain, immediately decussate and pass down through the pons and medulla and the contra-lateral region of the cord to the anterior horn cells.

(2) *The Tecto-Spinal:* This tract arises in the mid-brain at the level of the superior corpora quadrigemina, decussates and passes down in the posterior longitudinal bundle to the anterior horn cells. It conveys impulses to the voluntary muscles as the result of stimuli from the retinæ, which have passed to the calcarine fissure, and thence to the superior corpora quadrigemina.

(3) *The Vestibulo-Spinal:* The fibres arise in the lateral vestibular nucleus of the VIII nerve (Deiter's nucleus) in the lower pons and pass, in the antero-lateral region of the cord, to the anterior horn cells of the same side. This tract is the efferent of mid-brain reflexes subserving muscular tonus.

The short axons, passing from the small cells of the caudate nucleus and putamen to the globus pallidus, are sometimes termed the *Small-Celled Neo-Striate System*. The large cells of the globus pallidus and their efferents are sometimes termed the *Large Celled Pallidal System*. The latter system is phylogenetically older, and lesions of its cells and efferents cause Parkinsonism.

§ **672. Involuntary Movements—Tremor, Chorea, Athetosis.**—Three main types of involuntary movements occur in voluntary musculature. They are never present when the muscles are completely paralysed, *i.e.*, there must be relative integrity of the pyramidal tracts. They are: (1) *Tremor*—Involuntary, rhythmical oscillations of one or more parts of the body, resulting from the alternate contraction of muscle groups and their antagonists (§ 770). (2) *Chorea*—Irregular and spasmodic involuntary movements of groups of muscles, occurring during rest, and also superimposed upon voluntary movements, which they render inco-ordinate (§ 771). (3) *Athetosis*—Involuntary movements, of a writhing or (in the face) grimacing type, slower and more stereotyped than the movements of chorea. Between the periods of increased spasm the limbs are frequently hypotonic (§ 771).

The pathogenesis of these involuntary movements is obscure. By some, they are ascribed to lesions of the short axons passing from the small cells of the caudate nucleus and putamen to the globus pallidus (the small-celled neo-striate system). In congenital athetosis and chorea, the lesions are found chiefly in the caudate nucleus and putamen. Acute focal lesions of the *sub-thalamic body or corpus Luysii* (Fig. 161),

however, are known to produce hemichorea of the contralateral half of the body, and choreiform movements are known to follow lesions of the superior cerebellar peduncle and the optic thalamus. In this connection, it should be remembered that the cerebellum is really part of the extra-pyramidal motor system. The consensus of opinion is that these involuntary movements result from lesions of the extra-pyramidal paths when the pyramidal pathways are relatively intact.

§ 673. **Flaccid Paralysis.**—*The Lower Motor Neurone* commences in the anterior horn cell, or motor cell in the brain stem, and ends in the muscle fibre. It is the final pathway for motor impulses, whether pyramidal or extra-pyramidal, and is an integral part of spinal reflex arcs subserving muscular tonus. When it is destroyed, therefore, muscular paralysis, flaccidity, and absence of tendon reflexes, ensue. The muscle, completely isolated from the central nervous system, atrophies, and contractures occur; its electrical excitability disappears (Reaction of Degeneration) (see § 709). When the damage to the lower motor neurone is short of actual destruction, these changes appear in proportionately slighter degree. To summarise: the characteristics of **lower motor neurone lesions,** are: (1) Muscular paralysis and wasting. (2) Flaccidity. (3) Loss of tendon reflexes. (4) Reaction of degeneration.

§ 674. **Sensory Pathways.**—A mixed peripheral nerve contains fibres subserving every aspect of sensibility, cutaneous and deep. The *Cutaneous* sensory impulses are those of (1) Touch, (2) Pain, (3) Temperature. The *Deep* sensory impulses are (1) Vibration of a tuning-fork on bone, (2) Sense of passive movement of joints, (3) Sense of position, (4) Deep muscular sensibility, and tendon sensibility to deep pressure. All these sensory impulses, cutaneous and deep, enter the spinal cord through the posterior roots (Fig. 162).

The fibres for *Pain, Temperature* and *Touch* are freshly relayed in the posterior horn and cross over immediately on entering the cord in the *anterior commissure* and ascend directly in the spino-thalamic tract of the opposite side to the lateral nucleus of the thalamus. In the spinal cord the spino-thalamic tract lies in the lateral column, just ventral to the pyramidal tract (Fig. 163). The fibres for *Deep Sensibility* (viz., vibration, sense of passive movement and position, deep muscular sensibility, and tendon sensibility) together with some of the fibres for touch, ascend without relay in the posterior columns of Goll and Burdach of the same side, to the ipsilateral nucleus gracilis and nucleus cuneatus in the medulla. Here they are freshly relayed and ascend in the decussation of the *mesial fillet* (arcuate fibres) to the thalamus of the contralateral side. So that, eventually, all the sensory impulses, whether cutaneous or deep, undergo a decussation either immediately on entering the cord or later, in the fillet, and terminate in the contralateral thalamus. It will be noted that there are two pathways for touch. Tactile fibres, on entering the cord, ascend both in the contralateral spino-thalamic tract and in the ipsilateral posterior columns.

In the cord we have both crossed and uncrossed sensory pathways. In the spino-thalamic tracts and in the posterior columns, the longest posterior root fibres ascending from the coccygeal and sacral segments, lie nearer the mid-line. As fibres enter at higher segmental levels they are conveyed in a lamellar fashion, so that the fibres derived from lower segments are displaced inwards by those entering at higher levels. Furthermore, the fibres for touch, pain and temperature, entering the cord to cross to the spino-thalamic tract, decussate in the anterior commissure in a diagonal fashion; the fibres for pain and temperature crossing, say, in the mid-dorsal region in the space of one segment, those for touch crossing more slowly, the decussation occupying two segments. At higher segmental levels the crossing is more and more oblique.

The *spino-thalamic tract,* containing pain, temperature and touch fibres, ascends through the formatio reticularis of the medulla to join the crossed mesial fillet, in the pons, ultimately reaching the thalamus. The fibres for temperature and pain diverge in the medulla from the tactile fibres and pass to the outer side of the olive, subsequently converging to the mid-line and joining the fillet.

The *posterior columns of Burdach and Goll* are relayed in the nucleus cuneatus and nucleus gracilis, the latter more-mesial nucleus receiving fibres from the lower

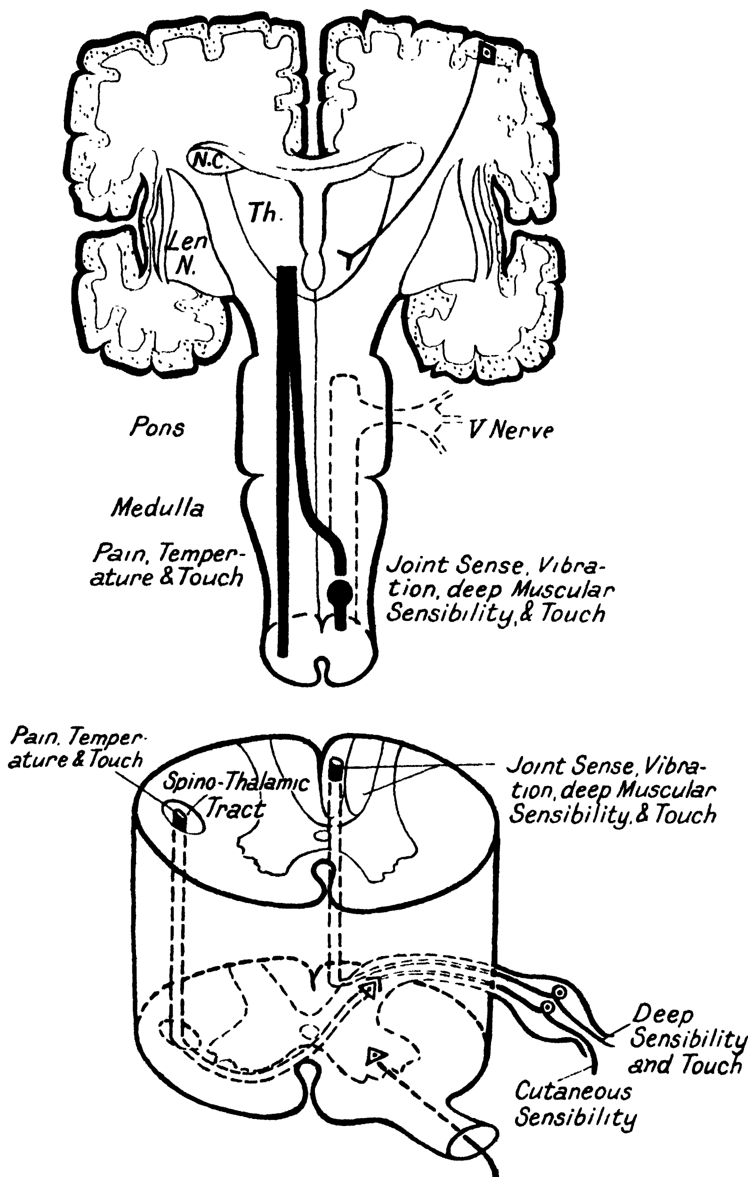


FIG. 162.—DIAGRAM OF THE SENSORY TRACTS. (After Head, Holmes, Walshe, Bing.)

limbs. Fibres concerned with deep sensibility pass upwards in these tracts and decussate, as the arcuate fibres in the medulla, to form the *mesial fillet*; this terminates in the lateral nucleus of the thalamus. The *fillet*, in the pons, passes along

the inner side of the sensory nucleus of the trigeminal nerve of the same side. The *thalamus*, situated in the lateral wall of the third ventricle, receives all the sensory impulses of the body, with the exception of the gustatory impulses, which have a direct connection with the cortex of the uncus. The thalamus registers the crude affective sensations of pain, heat, cold, producing emotions of pleasure or pain, and visceral sensations of hunger and thirst.

Inhibitory fibres run from the cortex to the thalamus, and, in certain circumstances, physical sensations are prevented from reaching the level of consciousness. For instance, we may be holding a book we are reading, quite unconscious of the pressure of the book on the hand which is holding it, until our attention is directed to this sensation. From the thalamus all sensory impulses are relayed to the *sensory cortex,* where sensation is discriminative.

Lesions of the *Sensory Cortex* do not affect crude sensations of temperature or pain. The characteristics are: (1) Loss of ability to localise tactile cutaneous stimuli (Atopognosis), (2) Defective appreciation of the size, shape and consistency of objects held in the hand (Astereognosis), (3) Light touches may be imperfectly felt, (4) there is impairment of Two-Point Discrimination (Compass-test), and (5) difficulty in appreciating the intensity of stimuli. The loss of these discriminative features of sensation may lead to inco-ordination of the affected limb. Sensory testing in cortical lesions gives great variety of response and threshold (Fig. 157).

Sub-cortical lesions produce a hemianæsthesia, affecting the contralateral face, upper and lower limbs. At the level of the sensory nucleus of the trigeminal nerve a lesion will produce a crossed hemianæsthesia, affecting the face on the same side as the lesion (from the proximity of the fillet to the ipsilateral sensory fifth nucleus) and the upper and lower limbs and trunk on the opposite side.

Lesions of the Thalamus release this structure from cortical control, with the production of characteristic symptoms of over-reaction to stimuli on the contralateral side—the "thalamic syndrome" of Déjérine and Roussy. This comprises (1) hemianæsthesia, (2) spontaneous pain on the affected side of the body, (3) over-reaction to painful or unpleasant sensory stimuli.

Central Cord Lesions (such as syringomyelia or intramedullary tumour) involve the fibres for pain and temperature, which cross in the anterior commissure, together with the touch fibres which cross in this region. Touch, as we have seen, has a double pathway; some of the fibres ascend in the posterior columns and consequently escape. The resulting cutaneous anæsthesia was termed by Charcot "Dissociated Anæsthesia," *i.e.,* there is loss of sensation to pin-prick, hot and cold (pain and temperature) while touches with cotton-wool can still be felt.

In *Hemi-section of the Cord,* posterior column sensibility is lost on the same side as the lesion; while pain and temperature are lost, and touch blunted on the opposite side. Lesions of the *posterior column* cause loss of joint sense, vibration and deep muscular sensibility. Sensitivity to light touches, as with cotton wool, is impaired. The loss of joint sense results in ataxia.

Lesions of the Posterior Roots or *Root-Entry Zone* of the cord (as met with in tabes) may produce impairment of all forms of sensation. From affection of the non-sensory afferents of spinal reflex-arcs subserving the tendon reflexes, these reflexes disappear, with loss of muscular tone.

These are also the pathways for certain **Non-Sensory Afferents** of reflex-arcs, situated at all levels in the spinal cord and brain-stem, conveying impulses which never reach consciousness, concerned with the *maintenance of muscular tonus.* They are, however, no less important than the sensory afferents and comprise:

(1) The long afferents of mid-brain reflex arcs concerned in the maintenance of tonus (see Muscular tonus, § 682).

(2) The dorsal and ventral cerebellar tracts which (with some collaterals from the posterior columns) carry to the cerebellum proprioceptive impulses for muscles and tendons important in the regulation of posture.

In a disease of the afferent system of projection fibres, such as tabes, besides sensory loss there are present also symptoms referable to destruction of these non-sensory reflex-arcs—*e.g.*, hypotonia, loss of tendon reflexes and inco-ordination.

Ascending and Descending Tracts in the Spinal Cord (Fig. 163).—The *Posterior Columns* are composed of the ascending *Tracts of Goll and Burdach*, carrying uncrossed fibres subserving Deep Sensibility (viz., Postural Sense, Sense of Passive Movement of Joints, Vibration Sense, Deep Muscular Sensibility) and the uncrossed fibres for Touch. These all pass to the nucleus gracilis and cuneatus, whence they are relayed in the mesial fillet to the contralateral thalamus.

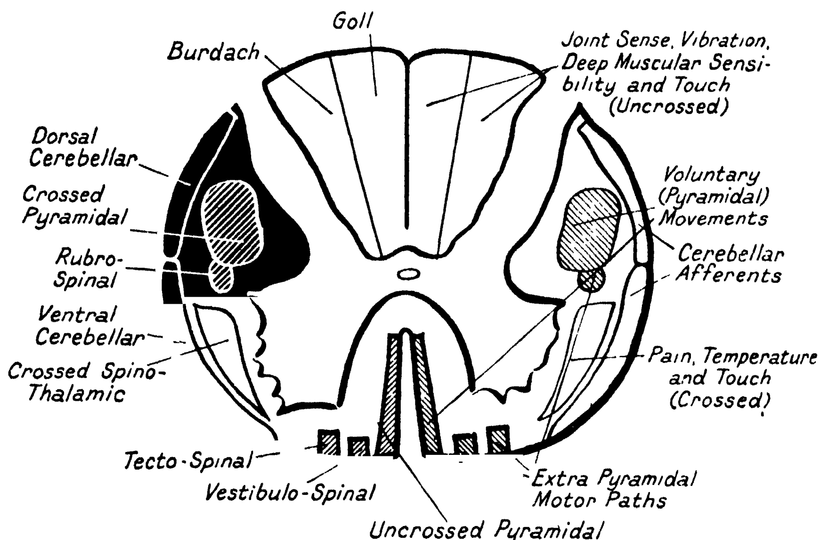


FIG. 163.—DIAGRAM OF ASCENDING AND DESCENDING TRACTS IN SPINAL CORD.
(Motor tracts shaded.) On the left half of the diagram are shown the names of the tracts, and on the right half of the diagram their functions.

The *Lateral Columns* contain the descending *Crossed Pyramidal Tract* and, just ventral to this, the *Rubro-spinal Tract*, the chief extra-pyramidal motor tract. These motor fibres are all relayed to the anterior horn cells. The periphery of the lateral columns is occupied by two ascending cerebellar tracts, the *Dorsal* (*Direct*) and *Ventral Cerebellar Tracts*, containing cerebellar afferents relayed from the cells of Clarke's Column.

The *Anterior Columns* contain the descending *Direct* (*Uncrossed*) *Pyramidal Tracts* and two extra-pyramidal motor tracts, the *Vestibulo-Spinal* and *Tecto-Spinal Tracts*. These descending motor tracts are all relayed to the anterior horn cells. The ascending *Spino-thalamic Tracts* lie in the antero-lateral region of the cord, carrying crossed fibres subserving Cutaneous Sensibility (viz., Pain, Temperature and most of those for Touch) to the fillet and ipsilateral thalamus.

Segmentation in the Spinal Cord. Each spinal segment or metamere has its anterior (motor) and posterior (sensory) roots. The *spinal nerves* are formed by fusion of one anterior and one posterior spinal root. The spinal nerve then divides into an *anterior* and a *posterior primary division*. Each of these divisions contains motor and sensory fibres, and ultimately supplies the cutaneous segment (Fig. 176) and the muscles (Table LII) developed in connection with its corresponding metamere.

§ 675. The Pathways for the Special Senses.—The peripheral organs for vision, hearing, taste and smell, are paired, and, in man, the sensory fibres proceeding from them to the cortex all exhibit a hemi-decussation, so that the cortex of one hemisphere subserves both organs in the case of each of these senses. In man, taste and smell have become intimately combined and have the same cortical representation, in the *uncus* and *hippocampal gyri.*

I. VISUAL SENSE.—For convenience the visual mechanisms are considered separately (§ 676).

II. AUDITORY SENSE.—The Eighth Cranial Nerve consists of a sensory or COCHLEAR division concerned with hearing, and a non-sensory or VESTIBULAR portion concerned with equilibrium. The *Cochlear nerve fibres* arise from cells in the SPIRAL GANGLION situated in the central pillar of the cochlea, their peripheral terminations ending in the hair cells of the organ of Corti. Centrally, the cochlear nerve passes to the brain through the internal auditory meatus and enters the lower border of the pons to terminate in the dorsally-placed *Cochlear nucleus* (tuberculum acusticum) which lies in the lower pons just external to the restiform body. From here, the fibres decussate as the *striæ acusticæ* and run in the *lateral fillet* to the Internal Geniculate Body and Inferior Corpus Quadrigeminum (*Primary Auditory Centres*). From the Primary Auditory Centres a fresh relay of fibres arises, which passes to the higher auditory areas in the superior temporal gyrus of the cortex.

Destruction of the Cochlea or the cochlear nerve produces *nerve-deafness,* irritative lesions produce *tinnitus.* Lesions of the cochlear nucleus in the pons, or the lateral fillets, will have a similar effect, but brain-stem deafness is rare. Lesions of the superior temporal gyrus do not destroy hearing because each ear has a bilateral cortical representation, but, in right-handed people, a lesion of the left superior temporal gyrus abolishes the comprehension of words and sounds heard (word-deafness).

III. GUSTATORY SENSE.—Smell and Taste are difficult to separate in man, in whom the old rhinencephalon or "smell-brain" has largely lost its function. Taste is concerned with cruder sensations of sweetness, sourness and bitterness, while smell is concerned with the appreciation of odours and flavours. Both taste and smell have the same cortical representation in the *gustatory area,* situated in the uncus and hippocampal gyri on the mesial aspect of the temporal lobe (Fig. 158).

(*a*) Taste.—The anterior two-thirds of the tongue is supplied by the lingual nerve. Fibres for taste leave the lingual nerve and enter the chorda tympani, having their cell-station in the geniculate ganglion in the aqueductus Fallopii. Thence they reach the nucleus of the nervus intermedius (dorsal nucleus of the seventh nerve, n. gustatorius) in the pons. From the posterior third of the tongue, taste-fibres pass along the glosso-pharyngeal nerve to their cell-station in the petrous ganglion, and thence they reach the dorsal nucleus of the glosso-pharyngeal nerve in the medulla. From the nuclei of the nervus intermedius and glosso-pharyngeal, fibres arise (fasciculus solitarius) and undergo a hemi-decussation passing to the uncus.

(*b*) Smell.—The olfactory nerves pass from the end-organs in the nasal mucosa through the cribriform plate of the ethmoids to the olfactory bulbs. Here fibres are relayed in the olfactory tracts. Each olfactory tract divides into a lateral and a mesial portion. The mesial portions decussate and join with the uncrossed lateral portions to terminate in the uncus.

Lesions of the olfactory bulbs or tracts produce *anosmia.* Irritative lesions of the uncus or hippocampal gyri produce *uncinate fits*—subjective sensations of taste and smell associated with champing or spitting and a transient dimming of consciousness, known as a "dreamy state." Anosmia does not occur from these cortical lesions. Loss of taste on the anterior two-thirds of the tongue occurs in lesions of the facial nerve in the aqueductus Fallopii, involving the geniculate ganglion.

§ 676. The Visual Mechanisms.—(*a*) **Visual Sense.**—The visual projection fibres are, clinically, of great importance. Each *Optic Nerve* passes backwards from

the globe into the cranial cavity and divides into a mesial and a lateral portion (Fig. 164). The mesial portions decussate at the *Optic Chiasma*, which lies just in front of the pituitary fossa, the uncrossed lateral divisions joining with the crossed mesial divisions to form the short *Optic Tracts*. The *Optic Tracts* bend round the lateral aspect of the mid-brain and terminate in the *External Geniculate Bodies or*

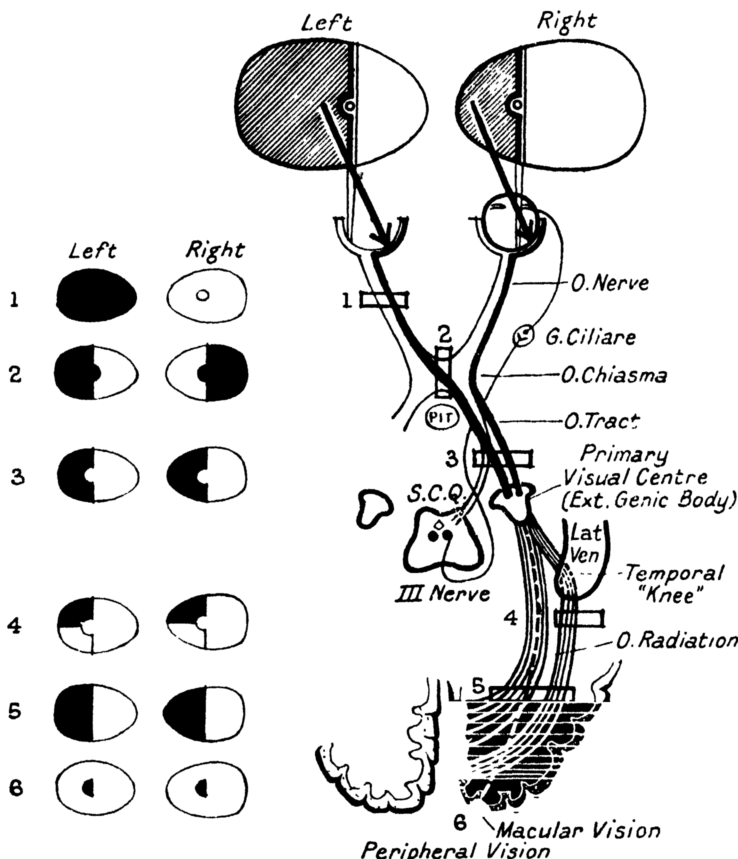


FIG. 164.—DIAGRAM OF THE VISUAL MECHANISMS.

Visual Field Defects resulting from Lesions at Different Points are represented on left of Diagram.

Lesion at 1 causes Left Optic Atrophy and Blindness in Left Eye.
" " 2 " Bitemporal Hemianopia with Involvement of Macular (Central) Vision.
" " 3 " Left Homonymous Hemianopia.
" " 4 " Left Upper Quadrantic Hemianopia from Involvement of Ventral Bundle (Temporal Knee) of Optic Radiations.
" " 5 " Complete Left Homonymous Hemianopia.
" " 6 " Left Homonymous Central Hemiscotoma.

The Path of the Pupillary Light Reflex is also shown. (Modified from Walshe.)

Primary Visual Centres. (N.B.—The Optic Thalamus has nothing to do with visual mechanisms.)

Some of the fibres in the Optic Nerves are non-visual in function and constitute afferent arcs of mid-brain reflexes concerned in the pupillary reactions to light and accommodation, and ocular movements. These non-visual fibres do not terminate in the External Geniculate Body but in the *Superior Corpus*

Quadrigeminum, which effects their connection with the ocular nuclei in the floor of the aqueduct.

From the Primary Visual Centres arise the *optic radiations*, which pass through the internal capsule behind the sensory fibres to the calcarine cortex. Each optic radiation contains fibres relayed from the corresponding halves of both retinæ. Furthermore, the dorsal bundle of the optic radiation, containing fibres relayed from the superior corresponding halves of both retinæ, passes directly backwards to the cuneus or upper lip of the fissure. The ventral bundle of the optic radiation, containing fibres from the inferior corresponding halves of both retinæ, runs first downwards into the uncus, then turning round the tip of the descending horn of the lateral ventricle ("temporal knee") runs backwards to reach the lower lip of the calcarine fissure.

The *macular fibres* also undergo a hemi-decussation in the chiasma, and are relayed from the external geniculate body to the tip of the occipital pole in the optic radiations. The tip of each occipital pole contains fibres relayed from the corresponding halves of both maculæ. The calcarine cortex subserves peripheral vision, while the cortex at the tip of each occipital lobe subserves central vision.

A lesion of one *Optic Nerve* will cause blindness in that eye. A lesion of the *Optic Chiasma* will abolish the functions of the nasal half of both retinæ and produce blindness of both temporal fields (bitemporal hemianopia) (Fig. 164). Lesions in this situation may also involve the uncrossed fibres, producing a complete blindness in one eye with a temporal hemianopia in the other. Lesions of the *Optic Tract* will abolish the functions of the corresponding halves of both retinæ, producing blindness of the contralateral halves of the visual fields (homonymous hemianopia). Lesions anterior to the superior corpus quadrigeminum and external geniculate body, causing blindness, are associated with loss of pupillary reaction to light when a pencil of light is thrown on one half of the retina, owing to the interruption of the non-visual afferents of the pupillary light reflex (Wernicke's hemianopic pupillary reaction) (Fig. 164). In blindness due to disease of the calcarine cortex, the pupils react when a beam of light is thrown on the retina.

Lesions of the *Optic Radiation* will produce homonymous hemianopia of the contralateral fields. Quadrantic hemianopia of the contralateral visual fields occurs when only a portion of the optic radiations are destroyed, the lower quadrants of the visual fields being the ones affected when the dorsal bundle is destroyed, the upper quadrants when the ventral bundle is destroyed.

Lesions of the *Visual Cortex*, when at the tip of one occipital pole, will produce a central hemiscotoma of the contralateral halves of both central visual fields. Central scotoma can also be produced by pressure lesions at the chiasma where the macular fibres lie ventrally and are delicate and vulnerable. Lesions of the upper lips of both calcarine fissures produce a horizontal hemianopia inferior, with blindness of the lower halves of both visual fields. Lesions of the left angular and supra-marginal gyri on the convex surface of the hemisphere, cause difficulty in recognising objects seen, without actual blindness, "visual agnosia" (word blindness) in right-handed people (Fig. 175).

§ 677. (*b*) **The Pupil and its Reactions.**—The cervical sympathetic is the *tonic dilator of the pupil*, the oculo-motor nerve is the *tonic constrictor of the pupil*. It has already been stated that the Optic Nerve and Optic Tract, besides containing visual fibres, also contain non-visual fibres which pass, not to the Primary Visual Centres but to the *Superior Corpus Quadrigeminum* (*Superior Colliculi*). These non-visual fibres form the afferent pathway for light impulses concerned with the *Pupillary Light Reflex* (Figs. 164 and 190). From the Superior Corpus Quadrigeminum in the mid-brain short *colliculo-ocular fibres* arise, and, surrounding the aqueduct and decussating below it, effect communication with the *Oculo-Motor Nuclei* in the floor of the aqueduct. This decussation puts each corpus quadrigeminum in connection with both oculo-motor nuclei and explains the *consensual light reaction*—when a beam of light

is thrown upon the retina, in a normal person the contralateral pupil contracts. The efferent fibres of the reflex-arc are the oculo-motor nerves, which are relayed in the ciliary ganglion and pass to the sphincter pupillæ. The Pupillary Light Reflex will be absent or diminished in disease of the Optic Nerve or Optic Tracts, mid-brain or oculo-motor nuclei, *e.g.*, Optic Atrophy will cause it. In the Argyll-Robertson phenomenon, where the pupillary light reflex is diminished or abolished and the pupillary contraction on accommodation-convergence retained, the lesion is probably in the mid-brain (usually syphilitic) in the Superior Corpus Quadrigeminum or the colliculo-ocular fibres round the aqueduct.

The reflex contraction of the pupil on *Convergence-Accommodation* is a complex affair, the pathways for which are not clearly elucidated. It is partly voluntary, and is probably a cortical reflex whose efferents are the pyramidal supra-nuclear oculo-motor fibres.

§ **678.** (c) **External Eye Movements** (Fig. 191).—Two pairs of the nuclei concerned lie in the mid-brain, the *Oculo-motor* and *Trochlear*, the other pair is in the dorsi-lateral part of the pons—the *Abducens*. The Oculo-motor and Trochlear nuclei are masses of grey matter, lying in the floor of the aqueduct of Sylvius. The *Oculo-motor fibres* sweep directly through the crus to emerge on its ventral aspect, and they are relayed in the ciliary ganglion to supply all the extrinsic muscles of the eye, except the external rectus (VI) and the superior oblique (IV). The *Trochlear fibres* decussate dorsally to the aqueduct before emerging from the dorsal aspect of the mid-brain to supply the superior oblique muscles. Round the abducens nucleus wind the fibres of the facial nerve in the floor of the fourth ventricle. The *Abducens fibres* emerge on the ventral aspect of the junction of the pons with medulla; they have a very long intracranial course, lying first in the subarachnoid space, and then perforating the dura opposite the posterior clinoid process to gain the outer wall of the cavernous sinus and orbit, supplying the external rectus muscle.

These three nuclei are all intimately connected by association fibres, and they may be conveniently regarded, not anatomically but physiologically, as a single compound nucleus (Nucleus of Edinger) whose functions, from before backwards, are :

(1) Pupillary Light Reflex.
(2) Accommodation-Convergence Reflex.
(3) Conjugate Upward and Downward Deviation of the Eyes.
(4) Conjugate Lateral Deviation of the Eyes.

The ocular nuclei receive supranuclear pyramidal fibres from the posterior part of the second frontal gyrus. Individual eye movements are not represented in the motor cortex, but irritative lesions of the second frontal gyrus produce conjugate turning of the eyes and head to the opposite side, with tonic or clonic convulsions of the contra-lateral extremities (Penfield). Lesions of the nuclei cause paralysis of *conjugate movement of both eyes*, while peripheral nerve (infranuclear) lesions produce *paralysis of individual muscles*, and are commonly unilateral. Thus, a right-sided sixth nerve nuclear lesion will produce paralysis of conjugate movement of both eyes to the right, while a lesion of the peripheral course of the right sixth nerve produces a unilateral right external rectus palsy.

Besides the pyramidal fibres, two other important sets of fibres converge upon the ocular nuclei. (1) The *posterior longitudinal bundle*, carrying fibres from Deiter's nucleus, and the spinal accessory nucleus, regulating the tonus and co-ordination of the eye muscles and the muscles which rotate the head. This is the pathway which is involved in the nystagmus produced by vestibular and cerebellar lesions. (2) The *visual cortex* in the occipital lobe sends *projection fibres* via the superior corpora quadrigemina to connect with the ocular nuclei. This is the pathway concerned in the protective blinking reflex induced by a sudden visual stimulus. Reflex protective withdrawal movements of the voluntary muscles are similarly induced through the extra-pyramidal tract leading from the superior corpus quadrigeminum to the anterior horn-cells—the tecto-spinal tract.

§ **679. Inco-ordination of Movement—Ataxia.**—Any lesion of the spinal cord, brain-stem or cerebral hemispheres, which interrupts the afferent impulses subserving sense of position and the appreciation of movement (joint-sense), will produce *Sensory Ataxia.* It will be remembered that these impulses are conveyed in the posterior columns to the contralateral thalamus and are thence relayed to the cortex. Clinically, such ataxia is well exemplified in diseases affecting the posterior columns, such as tabes or subacute combined degeneration of the cord. It may also, however, result from peripheral nerve lesions (peripheral neuritis) or disease of the sensory cortex. This form of ataxia is increased when the patient's eyes are closed, as the eyes give useful information about the position of the limbs.

In lesions of the cerebellum or its connections, ataxia is also met with. It is usually attributed to the loss of tonus which is observed in cerebellar disease and it is termed *Cerebellar Ataxia.* It is well exemplified in the phenomenon of intention tremor, to demonstrate which the patient is asked to touch his nose with his forefinger. The finger oscillates about its objective just before it is reached, but the nose is touched correctly even if the eyes are shut.

§ **680. The Mechanism of Equilibrium.** Equilibrium is dependent on perfect co-ordination of certain afferent impulses. These impulses are set up by : (1) movement of the limbs acting through the muscle-spindles, joints and tendons, (2) vision and (3) gravity, the latter acting through the vestibular apparatus.

The vestibular apparatus consists of: (*a*) The utricle and saccule containing sensitive hair-cells in contact with small crystals, the otoliths. The position of the otoliths with regard to the hair-cells varies with gravity, and impulses are set up in the saccules when the head is moved *vertically* or *forwards*. (*b*) The semicircular canals, also containing sensitive hair-cells which respond to movement of the endolymph. Impulses are set up in the semicircular canals by *angular* displacement of the head.

Impulses are constantly being received from the vestibular apparatus telling the position of the head in space. The cells of origin of the vestibular nerve are in Scarpa's ganglion in the internal auditory meatus. Fibres pass centrally to the vestibular (Deiter's) nucleus, which lies ventrally in the pons in the outer part of the floor of the fourth ventricle (Fig. 165). Efferent fibres connect this nucleus with the anterior horn cells (vestibulo-spinal tract) and the ocular nuclei (posterior longitudinal bundle). Deiter's nucleus receives afferents also from the cerebellum. Disturbances of the vestibular mechanism produce : (1) subjective vertigo, (2) hypotonia on the side of the lesion, (3) forced movements and falling, and (4) nystagmus.

In a normal subject certain disturbances (*e.g.*, sudden cessation of rotation, rocking) will cause maladjustment of postural reflexes and confusion. If a normal individual is rotated in a rotating chair which is suddenly checked and tilted backwards, the whole vestibular mechanism is so disturbed that the person may collapse or be flung out of the chair. For a time he will have no conception of how to orient himself in space. Ewald found that ablation of the vestibule causes hypotonia of the ipsilateral half of the body. A similar result will temporarily follow section of the vestibular nerve.

§ **681. The Cerebellar System** (Brain of Posture) is an integral part of the extrapyramidal motor mechanism, consisting of complex afferent and efferent systems of neurones. The *afferent* fibres come from the spinal cord (direct cerebellar tracts and fibres from the posterior columns) and the mid-brain (Deiter's vestibular nucleus and the oculomotor nuclei). The *efferent* fibres do not lead directly downward from the cerebellar cortex but are relayed from the central grey matter of the cerebellum (dentate and roof nuclei). These efferents run in three main groups : (1) *Cerebello-Rubro-Spinal :* to the opposite red nucleus and thence through the rubro-spinal tract, after recrossing to the anterior horn cells. Through these fibres the cerebellum is concerned with the tonus of the ipsilateral skeletal muscles. (2) *Cerebello-Vestibulo-spinal :* to the Vestibular (Deiter's) nucleus of the same side in the pons and thence through the vestibulo-spinal tract to the anterior horn cells. Through these fibres the cerebellum is concerned with correction for posture in voluntary movements. Through

the posterior longitudinal bundle these cerebellar efferents are brought in communication with the oculomotor nuclei in the floor of the iter, and the spinal portion of the spinal accessory nerve, controlling head movements. These connections explain disturbances of eye direction (skew deviation) and tonic neck reflexes ("cerebellar" attitude of head) met with clinically in cerebellar lesions. (3) *Cerebello-Cerebral;*

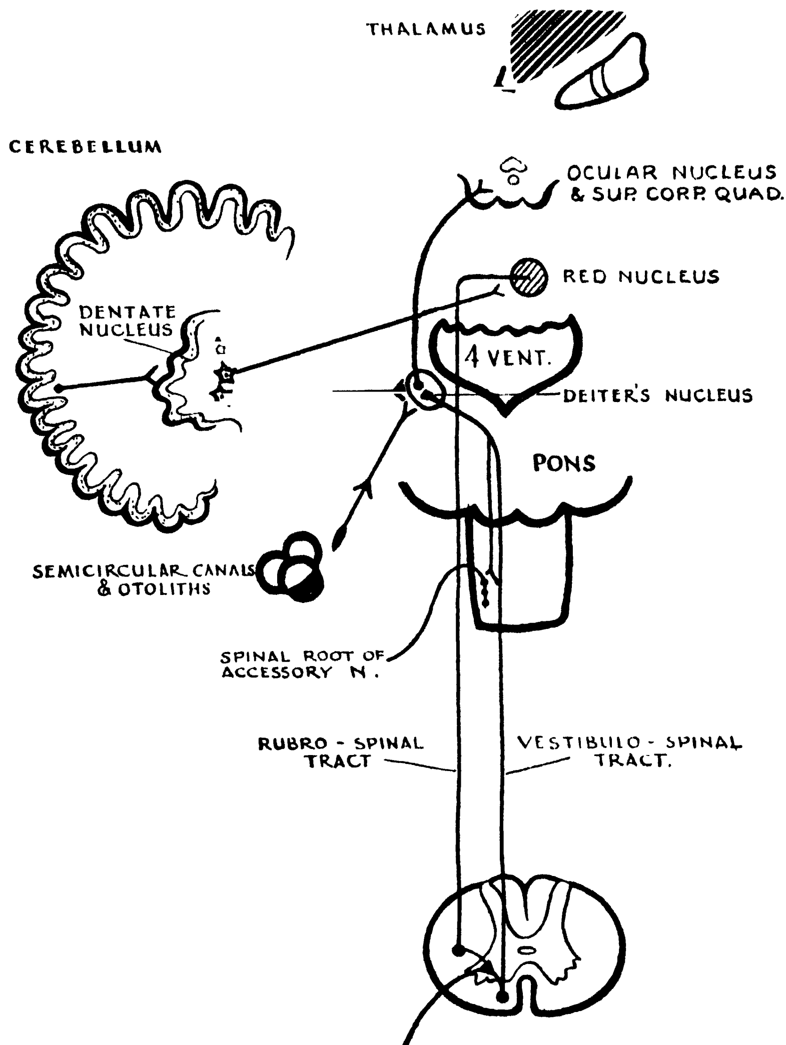


FIG. 165.—DIAGRAM OF THE CEREBELLAR AND VESTIBULAR EFFERENTS, showing the origin of these tracts and their termination in the ventral horn cells.

to connect with the opposite thalamus and the post-central and frontal cerebral cortex (Fig. 165).

The cerebellar afferents are concerned with the integration of muscle and joint and vestibular impulses. It is difficult to separate vestibular from cerebellar mechanisms as the two are intimately related.

In man, *irritative* lesions of the lateral lobes produce a vestibulo-ocular group of phenomena with notable hypotonia in the ipsilateral voluntary muscles, nystagmus, "cerebellar" attitude of the head and inco-ordination of voluntary movement. There is deviation from the straight line when the patient attempts to walk, the sound side, as it were, pushing him over to the side of the lesion. The patient pass-points to the side of the lesion. (See § 812.)

In *destructive* lesions of the cerebellum in man (*e.g.*, cerebellar atrophy) there is often absence of ocular phenomena and the only symptom may be a loss of the power of regulating pyramidal impulses (cerebellar ataxia). In patients who lost the whole of the cerebellum through war wounds, after a time there was only a general clumsiness of movement, intention tremor and slight slurring of speech. The inference is that if the cerebellum is destroyed the cerebral cortex can make itself independent of normal cerebellar adjustments.

Skew deviation of the eyes, a rare symptom, seems to depend on irritative lesions of the deep (dentate) nuclei of the cerebellum.

§ 682. **Muscular Tonus.**—The tonus or continuously braced-up condition of voluntary muscle is due to a series of proprioceptive reflexes, muscular or vestibular. The proprioceptive reflexes arise in the tendons of the flexor muscles of the limbs, *e.g.*, in the legs, as the result of pressure of the sole of the foot on the ground, and in the labyrinths, as the result of alterations in the position of the head (tonic neck reflexes). The long afferents of the proprioceptive impulses, arising in tendons, pass up the spinal cord in the antero-lateral columns (not the posterior columns) to the mid-brain, where the reflex centres are probably Deiter's nucleus and the Red Nucleus. The *Mid-brain* may be thought of as the *Brain of Tonus.*

The main efferent tract of the proprioceptive reflex-arc is the vestibulo-spinal tract to the anterior horn cells. Any influence which cuts off pyramidal impulses below the mid-brain level (*e.g.*, spinal cord compression) allows of the release of this reflex system with resultant increased tonus below the level of the lesion—*extensor rigidity.* When the long mid-brain tracts themselves are disturbed, as in the late stages of compression paraplegia, simple segmental spinal reflexes are released subserving deep reflexes and reflex muscular movements and *flexor rigidity* is produced.

The *corpus striatum* also contributes to postural tone, but the explanation of the muscular rigidity of Parkinsonism and the hypotonia of chorea and athetosis is still obscure.

The cerebellum, through its connection with the thalamus, red nucleus and Deiter's nucleus, has an important influence on the maintenance of tonus.

§ 683. **Anatomy of the Brain-Stem.**—The Brain-Stem comprises the Mid-Brain, Pons and Medulla. Here are grouped:

(1) The Sensory and Motor Pathways.

(2) The Cranial Nerve Nuclei.

(3) Important Reflex and Association Nuclei, *e.g.*, superior corpora quadrigemina, red nuclei, substantia nigra, Deiter's nucleus.

(4) Autonomic cells lying under the superior corpus quadrigeminum, connected by the *posterior longitudinal bundle* with the cervical sympathetic outflow from the antero-lateral columns of the spinal cord in the Th1 and 2 segments.

Any or all of these mechanisms may be involved in Brain-Stem lesions. Characteristically, one finds, as the result of a focal lesion, an ipsilateral cranial nerve palsy, with crossed hemiplegia or crossed hemianæsthesia, as described in § 670. For example, in lesions of the *mid-brain* there may be an oculo-motor palsy on the side of the lesion, with a contralateral hemiplegia from pyramidal involvement, signs of paralysis or irritation of the cervical sympathetic may be present (constricted pupil with enophthalmos, or the reverse of this), or, if the red nucleus and superior cerebellar peduncle are involved, contralateral involuntary movements and ataxia. Lesions of the dorsal part of the mid-brain will produce defective conjugate upward movement of the eyes. Lesions in the *pons* produce sixth or seventh nerve palsies, associated with pyramidal, sensory and cerebellar or vestibular signs.

Lesions in the *medulla* produce ninth, tenth, eleventh or twelfth nerve palsies in association with pyramidal and sensory signs, or are rapidly fatal.

(1) Motor and Sensory Pathways.—These have already been considered.

(2) Cranial Nerve Nuclei.—The student is advised to study the diagram (Fig. 166) which indicates the position of the various cranial nerve nuclei in the pons and medulla. The ocular nuclei have already been considered. The trigeminal nuclei are two, a motor and a sensory, and they are of great clinical importance, both from their extent and position. The ventral *motor nucleus* lies in the floor of the fourth ventricle and is concerned with the innervation of the muscles of mastication. The dorsal *sensory nucleus* is of wide extent, reaching from the third cervical segment of the spinal cord, extending through the tip of the posterior horns laterally in the

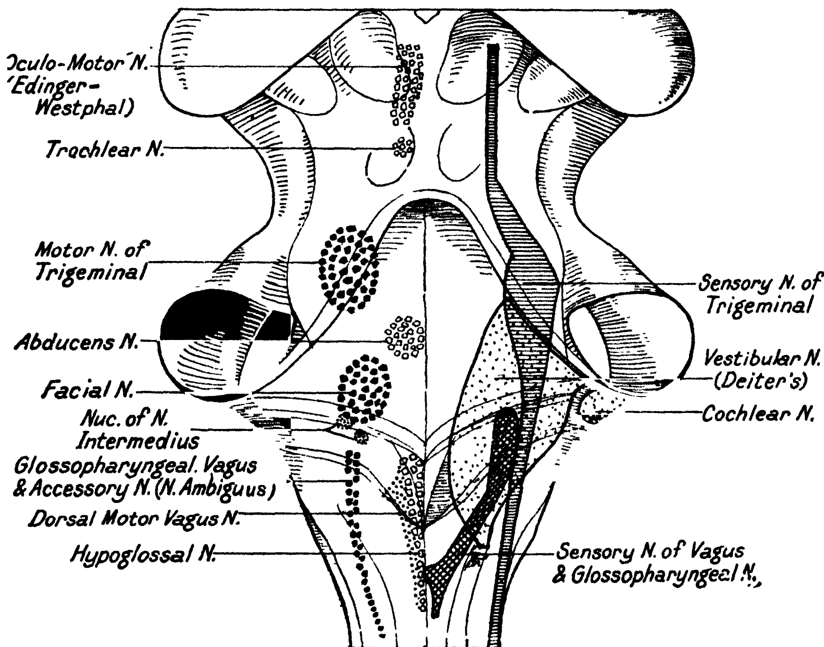


Fig 166.—Diagram of the Cranial Nerve Nuclei (Motor Nuclei on Left, Sensory on Right). (Modified from Herrick.)

medulla, higher than the superior tip of the motor nucleus in the pons. In the pons it lies lateral to the fillet. This nucleus is peculiar in that its lower extremity is connected with sensation in the upper face and cornea, while its upper extremity is concerned with sensation over the distribution of the mandibular (third) division of the fifth nerve. The sensory nucleus is concerned with common sensation in the face, scalp, cornea and conjunctiva, tongue and nasal mucous membrane. Both roots emerge from the ventro-lateral aspect of the pons, and on the sensory root is the Gasserian ganglion.

The movements of the face and tongue may be mentioned here. The *facial nucleus* lies in the lateral aspect of the pons, its nerve-fibres turning round the abducens nucleus under the floor of the fourth ventricle before emerging to supply the facial muscles. The *hypoglossal nucleus* is purely motor and concerned with the movements of the tongue. It lies lowest of all the cranial nuclei in the floor of the fourth ventricle near the mid-line. All the cranial nerve nuclei receive supranuclear pyramidal fibres from the motor cortex of both hemispheres *except* the part of the facial nucleus supply-

ing the lower facial muscles, and the hypoglossal nucleus, which receive fibres from one hemisphere only, the contralateral. In hemiplegia, due to a focal lesion in the internal capsule, all the cranial nerve nuclei escape paralysis owing to their bilateral innervation, except these two nuclei. We thus observe, in hemiplegia, a weakness of the lower face (the upper face is spared) and deviation of the tongue to the hemiplegic side when it is protruded.

The sensory *cochlear nucleus* is situated on the dorsal and outer aspect of the pons. The motor *glossopharyngeal-vagus-accessorius* nucleus (n. ambiguus) lies in the medulla and is concerned with the innervation of the muscles of the pharynx, larynx, palate, sterno-mastoid and trapezius. The motor nucleus of the spinal accessory nerve reaches as low as the third cervical segment. The *dorsal motor nucleus of the vagus* supplies the unstriped muscle of the alimentary tract and air passages. There is also a *sensory glossopharyngeal-vagus* nucleus in the medulla, concerned with common sensation from the ear, mouth, pharynx and afferents from the abdominal and thoracic viscera.

(3) Reflex and Association Nuclei of the Brain-Stem.

Superior Corpus Quadrigeminum.—This reflex centre in the mid-brain receives afferents from the optic tracts and from the visual cortex, and through its efferents the oculo-motor nerves and the tecto-spinal tracts, effects pupillary alterations, ocular movements, protective blinking and turning away from sudden visual stimuli. Lesions produce loss of upward movement of the eyes, with disorder of pupillary reactions.

Red Nucleus.—This nucleus and the substantia nigra, both in the mid-brain, subserve reflex tonus in the voluntary muscles. It is a head ganglion in a series of proprioceptive reflexes whose afferents come from (1) the cerebellum, controlling through the rubro-spinal tract the co-ordination of the limbs, and (2) from the globus pallidus, governing the performance of automatic association movements, *e.g.*, swinging the arms when walking. Lesions of the red nucleus may produce involuntary movements and ataxia. In animals, section of the mid-brain through the red nucleus, produces the phenomena of "decerebrate rigidity," due to the destruction of pyramidal control and the releasing of the extra-pyramidal mid-brain centres for reflex tonus in the voluntary muscles (see § 764).

Deiter's Nucleus. This nucleus, lying ventrally in the pons, receives fibres from Scarpa's ganglion (vestibular nerve), proprioceptive afferents from the antero-lateral columns of the cord and from the cerebellum, and transmits, through the vestibulo-spinal tract to the anterior horn cells, impulses regulating contractile tonus and equilibrium. Lesions of this nucleus produce hypotonia and loss of balance. From this nucleus also the posterior longitudinal bundle passes upwards to the ocular nuclei regulating the tonus of the external ocular muscles.

(4) Autonomic Cells and Fibres.—Disturbance of the sympathetic innervation of the eye, with irritative signs (midriasis, exophthalmos or lid-retraction) or paralytic signs (miosis, enophthalmos and pseudo-ptosis) follow upon lesions of the posterior longitudinal bundle in the mid-brain, pons, or medulla.

§ 684. **The Vascular Supply of the Brain and Spinal Cord.**—The Vertebral Arteries, before entering the foramen magnum, give off the anterior spinal and two posterior spinal arteries. These run downwards the entire length of the cord, forming, with their anastomoses, a vascular chain reinforced by branches of the intercostal and lumbar arteries, entering the vertebral column through the intervertebral foramina, and running along the anterior and posterior nerve roots. On entering the foramen magnum, the vertebral arteries give off the *posterior cerebellar arteries* and unite at the lower border of the pons to form the *basilar artery* (Fig. 167).

The *posterior inferior cerebellar* artery supplies the lateral aspect of the medulla, including the superior cerebellar peduncle, the lower part of the long sensory nucleus of the trigeminal, the fillet, the decussating arcuate fibres of the fillet, and the glosso-pharyngeal-vagus nucleus. Lesions of this artery produce "Cerebellar Apoplexy" with acute vertigo and hemiataxy, paralysis of the pharynx, soft palate and vocal

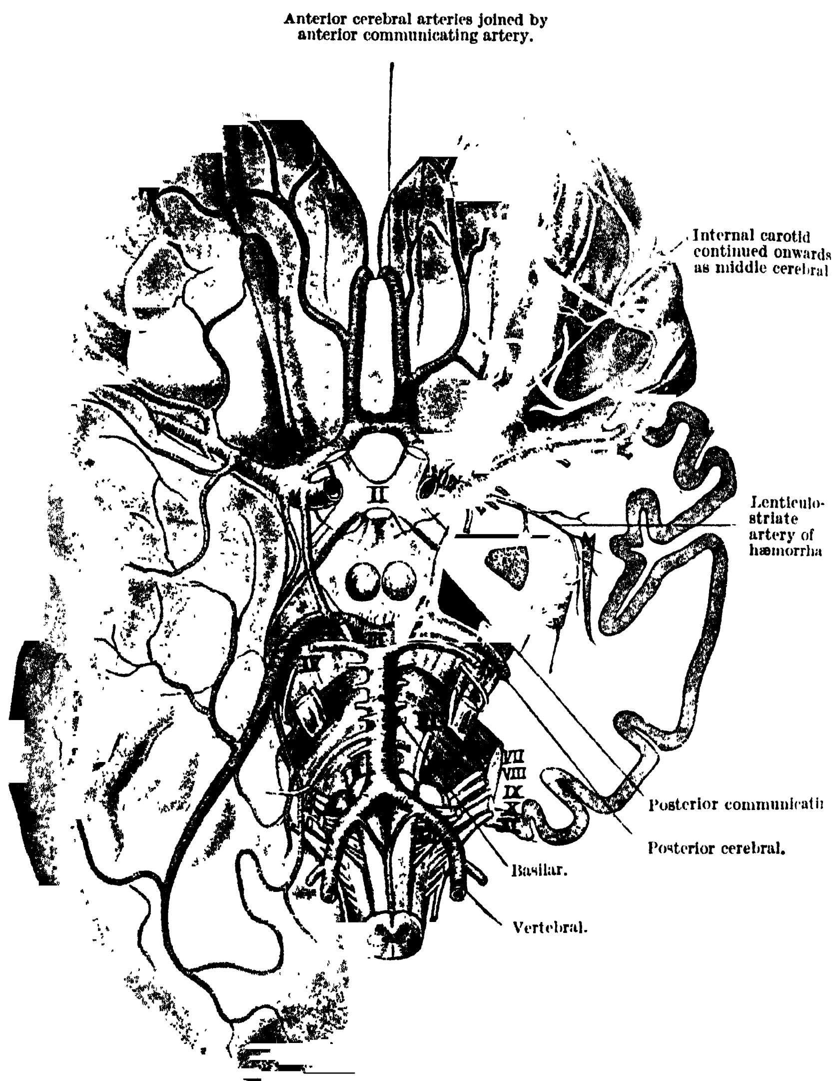


FIG. 167.—THE BASE OF THE BRAIN, showing the arterial distribution and the cranial nerves.—In the oblique section of the left hemisphere are seen from without inwards—grey matter of the island of Reil; claustrum (grey); external capsule (white); lenticular nucleus (grey); internal capsule (white) with artery of hæmorrhage; and caudate nucleus (grey). I., Olfactory lobe; II., optic chiasma; III., bifurcation of basilar artery between the third nerves; IV. (on right crus cerebri), beside fourth nerve; V. (on pons Varolii), beside fifth nerve; VI., sixth nerve (abducens); VII., facial nerve; VIII., auditory nerve; IX., glossopharyngeal nerve; X., vagus or pneumogastric; XI., spinal accessory; XII., hypoglossal nerve.

cord, with sympathetic oculo-pupillary signs, all on the side of the lesion, with a crossed dissociated hemianæsthesia of the body and sensory loss on the ipsilateral side of the face (*unilateral bulbar syndrome*).

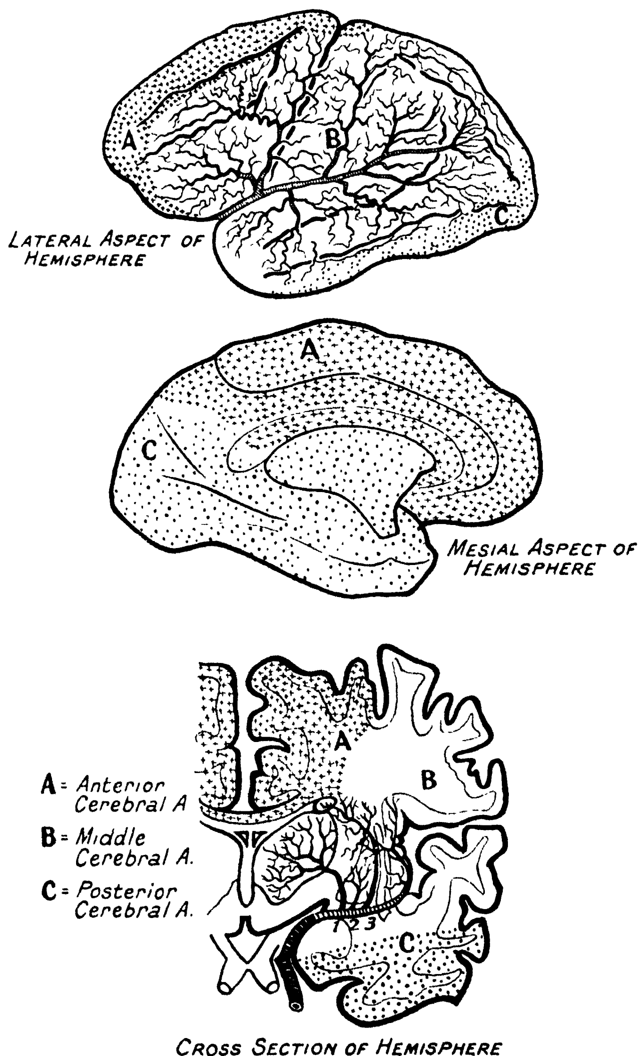


FIG. 168.—DIAGRAM OF CEREBRAL VASCULAR SUPPLY. (Modified from Bing.)
In the lowest figure:
1. Lenticulo-Optic Branch of Middle Cerebral.
2 and 3. Lenticulo-Striate Branches of Middle Cerebral.

The *basilar artery* supplies branches to the pons, and two large branches, the *superior cerebellar arteries* to the upper cerebellum, and divides at the level of the crura of the mid-brain to form the two *posterior cerebral arteries*.

The *internal carotid arteries* enter the skull just laterally to the posterior clinoid processes of the pituitary fossa and give off the ophthalmic arteries supplying the

globe and orbit. Obstruction of one internal carotid will produce a "*Carotid Hemiplegia*"—blindness, with optic atrophy on the side of the lesion, with a transient contralateral hemiplegia, the circulation through the middle cerebral being rapidly established through the Circle of Willis. The internal carotid arteries then divide into *middle* and *anterior cerebral arteries*, supplying, roughly, the anterior two-thirds of the cerebral hemispheres (Fig. 168).

The anterior, middle, and posterior cerebral arteries are connected to form the *Circle of Willis* in the subarachnoid space at the base of the brain. This circle is occasionally incompletely developed (Fig. 167).

The *anterior cerebral artery* passes anteriorly into the great median fissure, and, curving round the genu of the corpus callosum, runs backwards upon the superior aspect of this structure, supplying its anterior seven-eighths and giving off branches to supply the mesial surfaces of the hemispheres as far back as the parieto-occipital fissure, and supplying, in this territory, the cortical areas for the foot and leg at the top of the pre-central gyrus. The anterior cerebral artery sends perforating branches inwards towards the caudate nucleus. Obstruction of an anterior cerebral artery gives rise to a crural monoplegia or hemiplegia, with sensory impairment, most marked in the lower limb, with apraxia of the left upper limb from involvement of the corpus callosum, § 745 (Fig. 168).

The *middle cerebral artery* supplies most of the convexity of the cerebral hemisphere, including the motor, sensory, and speech areas of the cortex. Leaving the Circle of Willis, it gives off several small but important perforating arteries, which pierce the anterior perforated spot to supply the corpus striatum, optic thalamus, and region of the internal capsule. These are terminal arteries, without anastomoses, and they are termed the *lenticulo-striate* and *lenticulo-optic* arteries (Fig. 168). The middle cerebral artery then runs along the Sylvian fissure to supply the whole of the convexity of the cortex, with the exception of the area supplied by the anterior cerebral artery, and at the occipital pole of the hemisphere anastomoses over the cortical area for central vision with the posterior cerebral artery. The middle cerebral artery also supplies, by penetrating branches, the major part of the centrum ovale, including the temporal knee of the optic radiations and external capsule.

The *posterior cerebral artery* winds round the crus, supplying the mid-brain nuclei (corpora quadrigemina, red nucleus, etc.), and supplies the inferior mesial surfaces of the temporal and occipital lobes, the uncus, and posterior seventh of the corpus callosum, anastomosing, with the middle cerebral artery, over the convexity of the occipital pole (area for central vision). It gives off an important branch, the *calcarine artery*, to supply the cuneus and lingual gyri above and below the calcarine fissure, respectively. In obstructive lesions of the posterior cerebral artery, whilst peripheral vision is destroyed from destruction of the calcarine cortex, the anastomosis with the middle cerebral artery over the occipital pole ensures a blood supply to the area of central vision which, therefore, escapes.

The *choroid plexuses* of the lateral ventricles, which have to do with the formation of spinal fluid, are supplied by the choroid branches of the internal carotid and middle cerebral. The choroid veins drain backwards to join the great vein of Galen, draining into the anterior extremity of the straight sinus in the tentorium cerebelli.

§ 685. The Cranial Venous Sinuses.—The walls of the venous sinuses consist of dura mater. They drain blood from the brain and meninges, and cerebro-spinal fluid passes into these sinuses through the Pacchionian bodies. They empty into the internal jugular vein and communicate with the veins of the head and neck through the orbital vein and various emissary veins, the most important of which is the large mastoid emissary vein. The chief sinuses are the Superior Longitudinal, the Lateral, the Straight and the Cavernous. The basal sinuses, *e.g.*, Lateral and Cavernous, communicate freely, but the *Superior Longitudinal Sinus*, which drains the cortical veins, opens only into the Torcular Herophili, and obstruction of this sinus leads to widespread bilateral cortical necrosis, characterised, clinically, by a paraplegia from destruction of the cortical motor leg areas.

The *Straight Sinus* runs medially between the two halves of the tentorium cerebelli, draining the choroidal veins and the great vein of Galen.

The *Lateral Sinus* drains the veins of the posterior fossa and runs from the external occipital protuberance, in an arched fashion, forwards and downwards, to open into the internal jugular vein through the jugular foramen. It occupies a groove in the mastoid part of the temporal bone and communicates with the mastoid emissary vein. Obstruction causes œdema over the mastoid process.

The reticulated *Cavernous Sinuses* lie on either side of the sphenoidal air cells, draining the veins at the base of the brain and the orbital veins. They communicate with one another by means of the circular sinus, and communicate posteriorly through the superior and inferior petrosal sinuses with the lateral sinuses. Thrombosis of the lateral sinuses may thus spread into first one cavernous sinus and then the other. On the medial wall of the cavernous sinus lies the internal carotid artery, with the abducens nerve. The oculo-motor, trochlear, and ophthalmic divisions of the trigeminal nerve pass forwards, on the lateral wall of the sinus, to enter the orbit through the sphenoidal fissure. These structures are separated from the blood in the sinus only by its lining membrane (Fig 169). Occlusion or compression of this sinus causes proptosis (protrusion of the eyeball) with orbital œdema, ocular palsies, and pain and sensory loss over the first and second divisions of the trigeminal nerve.

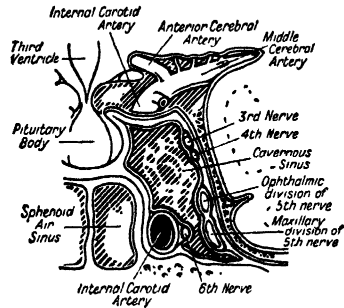


FIG. 169.—DIAGRAM OF THE CAVERNOUS SINUS.

§ 686. **The Cerebral Ventricles.**—In each cerebral hemisphere lies a *lateral ventricle* having three horns meeting in the parietal region. The anterior horn is deeply indented on its lateral surface by the head of the caudate nucleus. The temporal or descending horn is smaller and extends into the temporal lobe. The occipital or posterior horn is very inconstant in size and length. The anterior horns in their posterior two-thirds are separated only by the thin septum lucidum. The occipital horns are somewhat widely separated, while the temporal horns diverge more markedly from one another. The lateral ventricles communicate through small apertures called *interventricular foramina* (of Monro), with a single median cavity, the *third ventricle*, which lies between the optic thalami. The third ventricle communicates anteriorly with the infundibulum or pituitary stalk, and posteriorly by the long narrow iter (aqueduct of Sylvius) through the mid-brain with the *fourth ventricle* which lies between the bulb and cerebellum. The fourth ventricle opens into the subarachnoid space by three foramina, one in the median dorsal line (foramen of Magendie) and two laterally near the flocculi of the cerebellum (foramina of Luschka).

§ 687. **The Cerebro-Spinal Fluid.**—The central nervous system is enclosed in the bony case of the skull and vertebral column, and is suspended in a water-cushion of cerebro-spinal fluid. This cerebro-spinal fluid is formed by the epithelial-covered choroid plexuses of the lateral, third and fourth ventricles, by a process of dialysis. The total amount of fluid normally present within the cranio-spinal dura mater is 120–150 cubic centimetres. From the lateral ventricles the fluid passes through the foramina of Monro into the third ventricle and thence by the aqueduct of Sylvius to the fourth. It leaves the ventricular system by the medial foramen of Magendie in the roof of the fourth ventricle and the bilateral foramina of Luschka, one in each lateral recess of the fourth ventricle.

Within the subarachnoid space, the cerebro-spinal fluid bathes the whole surface of the brain and spinal cord. The fluid circulates forward through the basal cisterns, passes through the opening between the tentorium and the brain-stem and upwards over the surface of the hemispheres, to be absorbed directly into the cranial venous

sinuses by way of the arachnoid villi. These villi are invaginations of the subarachnoid space through the fibrous dural wall of the sinuses and project into their lumen. It is possible that some absorption may take place through perivascular spaces, into the cerebral capillaries, but this is of subordinate importance.

The subarachnoid space, in which the cerebro-spinal fluid circulates, is traversed by numerous delicate trabeculæ stretching from the arachnoid on the outer side to the pia on the inner. Within this space lie the cerebral and spinal blood vessels, and it is crossed by the cranial and spinal nerves. All these structures, like the walls of the subarachnoid space, are covered by flattened mesothelial cells.

Certain deep expansions of the subarachnoid space, called cisterns, exist at the base of the brain. Of these the most important are (1) the *cisterna magna*, situated between the inferior vermis and the medulla, and extending outwards on each side beneath the cerebellar hemispheres; and (2) the *cisterna basalis*, in the neighbourhood of the interpeduncular space, in which lie the Circle of Willis and the third nerves. The subarachnoid space sends important funnel-shaped prolongations along the spinal nerves as far as their foramina of exit from the dura mater, and, within the cranium, there are important prolongations (*a*) along the optic nerve as far as the exit of the nerve from the globe and (*b*) along the trigeminal nerve; so that the Gasserian ganglion is enclosed in a tiny pool of cerebro-spinal fluid, the cave of Meckel. The perilymph of the internal ear communicates through the internal auditory meatus with the cerebro-spinal fluid in the subarachnoid space.

The subarachnoid space dips into the sulci and is continued as sleeve-like channels surrounding the pial vessels into the brain substance. These perivascular spaces were first described by Virchow and Robin, and they are called *Virchow-Robin Spaces*. They subdivide with the blood vessels, and eventually communicate within the cerebral substance with pericellular spaces about the nerve-cells. The Virchow-Robin Spaces normally empty into the subarachnoid space, and, in inflammatory conditions affecting the central nervous system, they are packed with cells. Seen on a cross-section, they present the so-called "perivascular cuffing" appearance.

It is possible that the spinal fluid, when secreted, contains neither cells nor protein, these being added to it in the course of its circulation, from the perivascular spaces. It is possible also that the lymph from the peripheral nerves may pass into the subarachnoid space by way of the nerve-roots.

Normal cerebro-spinal fluid is clear and colourless. Whereas the ventricular fluid is almost non-albuminous and cell-free, the fluid obtained normally from the dependent spinal theca by lumbar puncture contains not more than 4 lymphocytes per cubic millimetre, 0·025 to 0·03 per cent. protein, 0·05 to 0·08 per cent. glucose, and 0·725 per cent. chlorides. The chloride in the spinal fluid is normally higher than that in blood plasma (0·6 per cent.); glucose, calcium, cholesterol and uric acid, are present in lesser amounts in the cerebro-spinal fluid than in the blood plasma. Spinal fluid, obtained by lumbar puncture, is normally under a pressure of 60 to 150 millimetres of water measured with a manometer (§ 919).

§ 688. **Surface Markings.**—The *Rolandic Fissure* is identified by taking a point midway between the nasion (root of the nose) and the external occipital protuberance, and drawing a line from a point 1 cm. behind this, downwards and forwards, at an angle of 67½ degrees, *i.e.*, *three-quarters* of a right angle (easily obtained by folding the square edge of a card appropriately) with the mid-line.

The *Spinal Cord* terminates in the conus medullaris, which is opposite to the 1st lumbar spinous process, while the *spinal theca* reaches as low as the level of the 2nd sacral spinous process.

The Hypothalamus and Autonomic Nervous System

§ 689. The **Pituitary** gland with its hollow infundibulum is intimately connected with the Hypothalamus. It is the most vascular gland in the body; it weighs 0·5 gram, and it lies in the bony sella turcica (Fig. 187A). The anterior lobe (pars

glandulosa) is made up of eosinophil and basophil secretory cells, and of their precursor chromophobe cells, which are non-secretory. The intermediate lobe consists of vesicles filled with colloid. The posterior lobe (pars nervosa) consists of nerve cells and neuroglia, and it produces a special internal secretion.

The *pituitary hormones* are exceedingly complex. Those formed by the eosinophil cells of the anterior lobe are probably concerned with bodily growth. The secretions

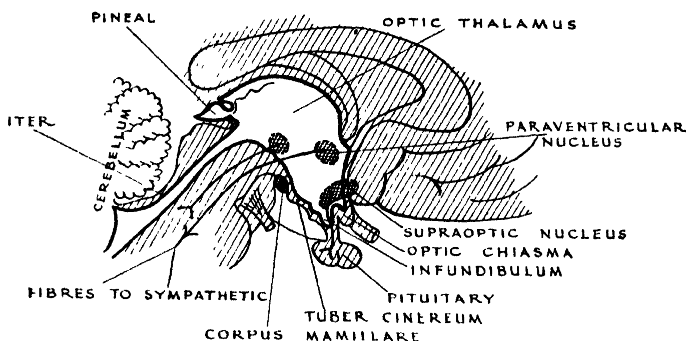


FIG. 170.—MEDIAN SURFACE OF THE HYPOTHALAMUS; shows the more important nuclei and the tracts proceeding from them.

of the basophil cells of the anterior lobe have to do with the development and activity of the gonads and mammæ. They are also concerned with the activity of the thyroid, and with carbohydrate metabolism. The posterior lobe secretion consists of two substances (*a*) vasopressin, with an " anti-diuretic " hormone regulating the constancy of the osmotic pressure of the plasma by causing water retention; and (*b*) oxytocin, which has a specific constricting effect on uterine muscle.

The **Hypothalamus** (Fig. 170) is that region of the brain forming the floor and lateral walls of the third ventricle. Various cell groups can be defined in the grey matter here, giving rise to three main groups of efferent fibres: (1) Fibres arising in the supra-optic group of cells going to the posterior lobe of the pituitary and pars intermedia. (2) A second group passing to the brain stem, and (3) Efferents from the posterior (paraventricular) group of cells controlling the autonomic nervous system.

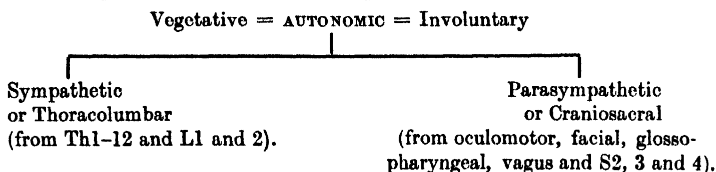
Not only does the hypothalamus regulate pituitary activity but it is the head ganglion of the autonomic nervous system; sympathetic and parasympathetic components having their representation there. Hypothalamic stimulation causes autonomic and other effects. Regulation of sleep mechanism and control of body-temperature are functions of the hypothalamus. Clinically, lesions of the hypothalamus cause (1) Diabetes insipidus, and sometimes glycosuria; (2) Genital dystrophy and amenorrhœa; (3) Infantilism, obesity, and other disorders of growth; (4) Disorders of sleep rhythm.

The *pathological causes* of these syndromes may be: Cranio-pharyngeal cysts, third ventricle and basal neoplasms, fractures of the base of the skull, vascular lesions, syphilitic and tuberculous basal meningitis and encephalitis lethargica.

Through the posterior longitudinal bundle and the vestibulo-spinal tract the hypothalamus connects with the Edinger-Westphal (oculomotor) nuclei, the salivary nuclei and the dorsal nucleus of the vagus, in the brain-stem.

§ 690. **The Autonomic Nervous System** consists of (i.) Sympathetic and (ii.) Parasympathetic Divisions, which are anatomically and physiologically separate.

The confusion of nomenclature of different writers is troublesome to the student and the following scheme may be found helpful.



The Autonomic Nervous System innervates bodily structures which are not under direct voluntary control. These include *smooth muscle* organs, the iris, and *tubular viscera* such as the bronchi, gastro-intestinal and genito-urinary tracts, the lachrymal, sweat and digestive *glands*, and the *heart* and *blood vessels*. Each of these structures has a dual innervation from the sympathetic and parasympathetic divisions, which are physiologically in a state of balanced opposition. When one division is excited, the other is inhibited. Sympathetic stimulation causes a diffuse reaction in several organs, but the result of parasympathetic stimulation is more specific and local.

I. The SYMPATHETIC Division consists of (*a*) *Ganglion cells* situated in the lateral

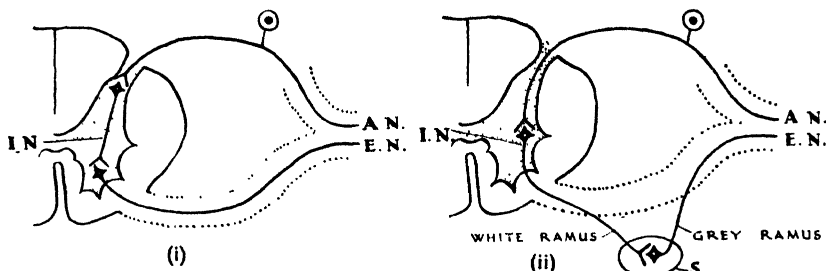


FIG. 171.—Diagram showing AFFERENT (A.N.), INTERCALARY (I.N.) and EFFERENT (E.N.) Neurones in (i.) a spinal and (ii.) in a sympathetic reflex arc. S. indicates the sympathetic ganglion of the paravertebral chain.

horn of the grey matter of the spinal cord extending from the Th1 to L2 level (lateral column); (*b*) The paravertebral *Sympathetic Chains* lying close to the vertebral column on the two sides, and (*c*) Peripheral *Sympathetic Nerves* (greater, middle and lesser splanchnic nerves) and *Collateral Plexuses* (cardiac, cœliac and hypogastric plexuses). The sympathetic chains and the peripheral plexuses are all that may be seen naked-eye by the casual dissector. In the thoracic region each ganglion of the chain is connected to its anterior spinal nerve root by two little *rami communicantes.*

Histologically, it has been shown that the sympathetic is made up of a number of reflex arcs with afferent, intercalary and efferent neurones, as in a spinal reflex arc (Fig. 171). The *afferent neurone* has its cell in the posterior root ganglion and enters the spinal cord to terminate in cells of the lateral column. From this column the *intercalary neurone* arises, and is projected out of the cord, within the anterior nerve root, which it leaves to enter the corresponding ganglion of the sympathetic chain. The *efferent neurone* arises in the ganglion and joins the anterior nerve root to be distributed with it peripherally. The sympathetic intercalary neurone is called the *white* (myelinated) ramus; the efferent neurone is called the *grey* (unmyelinated) ramus. The student will find it easy to remember that the grey ramus passes *away* from the ganglion. To the naked eye the grey and white rami are indistinguishable: the difference is histological.

The *sympathetic chains* consist of 24 ganglia: superior, middle and inferior cervical (stellate) ganglia, 12 ganglia in the thoracic, 4 in the lumbar and 5 in the sacral region. The chains fuse below in front of the coccyx (ganglion impar) and above they break

up over the internal carotid arteries. The outflow of intercalary neurones (white rami) from the spinal cord is between the Th1 and L2 segments only. These intercalary neurones, when they enter the sympathetic chain ganglia, behave in one of three ways (Fig. 172) :—(1) They may terminate here and anastomose with an efferent neurone (*e.g.*, fibres to sweat glands or blood vessels). (2) They may pass upwards or downwards in the sympathetic chain to terminate in a ganglion above or below (*e.g.*, fibres from Th1 and 2 passing to the cervical ganglia, and from L1 and 2 passing to the lumbar and sacral ganglia). (3) They may branch in the ganglion of the chain and

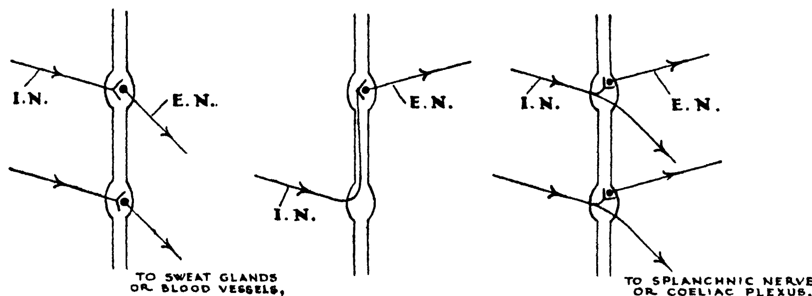


FIG. 172.—Diagram to show the three possible arrangements of intercalary neurones on entering the paravertebral sympathetic chain.

pass through it to form peripheral sympathetic nerves (*e.g.*, splanchnic) and plexuses (*e.g.*, cœliac).

It is characteristic of the sympathetic that there is always a ganglion between the cord and the viscus, blood vessel, or gland supplied by the efferent neurone. The intercalary neurone entering the ganglion is sometimes called the *pre-ganglionic* fibre, the efferent neurone the *post-ganglionic* fibre.

The cells of the lateral column are brought into relationship with the *hypothalamus* through the posterior longitudinal bundle and the vestibulo-spinal tract.

The *cervical sympathetic* fibres pass from the cord at the Th1 and 2 level and ascend in the cervical sympathetic chain to the superior cervical ganglion. Here efferent neurones arise and ascend in the carotid plexus to the ophthalmic division of the fifth nerve and run *via* the naso-ciliary nerves to the eyeball. The cervical sympathetic is the tonic dilator of the pupil. The middle cervical ganglion gives fibres to the thyroid gland and the stellate and upper thoracic ganglia furnish accelerator fibres to the heart and vaso-constrictor fibres to the upper limbs.

Destruction of the cervical sympathetic causes *Horner's syndrome*—a small pupil, narrowing of the ocular fissure from paralysis of the unstriped muscle of the levator palpebræ superioris, and apparent but not real enophthalmos. Irritative lesions of the cervical symphathetic cause opposite effects. Excision of the *stellate ganglion* will cause in addition vaso-dilatation in the upper limbs and the corresponding side of the face, with absence of sweating and gooseflesh and insignificant slowing of the heart.

Sympathetic stimulation raises blood pressure, inhibits alimentary peristalsis, causes erection of the hairs and sweating, and excites secretion of adrenalin, raising the blood sugar.

II. The PARASYMPATHETIC Division has two main outflows from the central system : (*a*) The Cranial Outflow and (*b*) The Sacral Outflow.

(*a*) The Cranial Outflow occurs through the oculomotor, facial, glossopharyngeal and vagus nerves, the ganglion cells being situated in the mid-brain in the oculomotor (Edinger-Westphal) nucleus, and in the medulla in the inferior and superior salivary nuclei and the dorsal nucleus of the vagus. Every bodily structure innervated by the sympathetic has also a parasympathetic innervation. It is through the vast ramifications of the vagus nerve that the parasympathetic is brought into communication with most of the viscera.

(*b*) The Sacral Outflow leaves the spinal cord with S2, 3 and 4 nerves in the cauda equina. They leave the spinal nerves, do not pass through the sacral sympathetic chains, but forming the nervi erigentes, run directly into the hypogastric ganglia and thence to the walls of the pelvic viscera. As with the sympathetic, there is always a ganglion between the cord and the viscus supplied. In the case of the parasympathetic, however, the ganglia lie in the walls of the viscera, so that the preganglionic fibres are long and the postganglionic fibres very short.

Stimulation of the parasympathetic causes effects antagonistic to those caused by sympathetic stimulation. The pupil is constricted, the heart retarded, the bronchioles constricted and peristalsis promoted; secretion of insulin and lowering of blood sugar occurs.

Pharmacologically, various drugs and hormones act on the autonomic nervous system. Depending on the dosage, sometimes a sympathetic, sometimes a parasympathetic effect is obtained with any one substance, but in the main the following table is true:—

	Sympathetic	*Parasympathetic*
Stimulating . . .	Adrenalin, Ephedrine	Physostigmine, Acetylcholine
Depressing	Nicotine	Atropine, Nicotine

Sir Henry Dale and his co-workers have recently established that a portion of the cerebrospinal system and the whole of the autonomic nervous system achieve their effect through chemical mediators formed at the nerve endings. These substances, normally found in the body, are acetyl-choline and adrenalin (sympathin). Thus there are two kinds of nerves, the *cholinergic* and *adrenergic*. In the case of the cerebrospinal system the motor fibres to striate muscles are cholinergic. In the case of the autonomic nervous system the parasympathetic fibres, both pre- and post-ganglionic, are cholinergic. The sympathetic fibres to the sweat glands are cholinergic but most of the sympathetic post-ganglionic fibres are adrenergic. Orbelli, using an excised skeletal muscle preparation, has shown that sympathetic stimulation diminishes fatigue, the maximum effect being reached some time after sympathetic stimulation has ceased.

Innervation of the Bladder. (1) The parasympathetic, by stimulating the detrusor muscle and relaxing the sphincter, empties the bladder of urine. Tonic activity of the sympathetic causes the opposite effect—retention of urine. The integrity of the parasympathetic reflex arc is essential for normal emptying of the bladder. The stimulus of bladder distension sets up impulses which pass in parasympathetic afferents to the conus and sacral region of the cord. The intercalary neurones emerge from the cord with the sacral outflow (S2 and 3) as the *nervi erigentes*, which anastomose with the efferent fibres in a plexus on the bladder walls (Fig. 173). Disturbances of this reflex arc, such as occur in tabes dorsalis (degeneration of the afferent fibres), or in lesions of the conus, result in retention from unopposed sympathetic activity.

Micturition can be initiated voluntarily in response to a cerebral stimulus, capable of activating the parasympathetic reflex arc. The cord pathways of this mechanism are not known. The Compressor urethræ and Bulbocavernosus muscles are under voluntary control and are innervated by the spinal pudendal nerve (S1 and 2). Incomplete spinal cord lesions cause precipitancy or retention. Complete transverse cord lesions cause reflex incontinence.

(2) The sympathetic fibres to the bladder leave the cord at the L1 and 2 segments and pass to the superior hypogastric plexus (presacral nerve, Fig. 173, G. Inf. Mes.) to anastomose with efferent fibres to the bladder. The tonic activity of the sympathetic retains urine in the bladder. Lesions of L1 or 2 roots may cause dribbling incontinence.

INNERVATION OF BLOOD VESSELS. The sympathetic vaso-constrictor fibres for the vessels of the upper limbs come from the stellate and upper two thoracic ganglia. Those for the lower limbs come from the 2nd, 3rd and 4th lumbar ganglia. They run in the spinal nerves and are distributed to the blood vessels chiefly in the periphery of the limbs where the arterial bed is richest (Woollard). Resection of these ganglia is carried out for Raynaud's disease, erythrocyanosis and circulatory disturbances

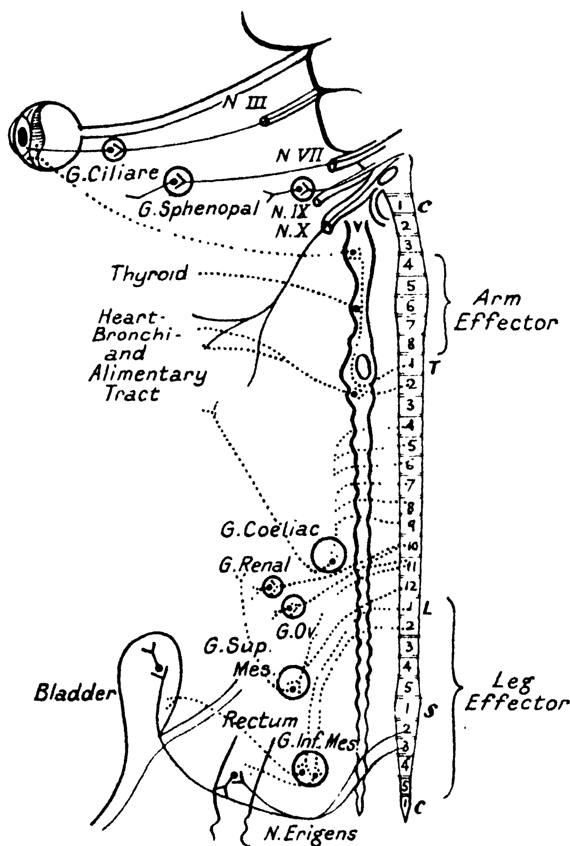


FIG. 173.—DIAGRAM OF THE AUTONOMIC NERVOUS SYSTEM.

(Sympathetic represented by dotted lines and the parasympathetic by continuous lines.)

following poliomyelitis. Dorso-lumbar splanchnectomy is practised to produce lowering of high blood-pressure.

The autonomic nervous system and the hypothalamus may be thought of as an emergency protective mechanism which may be aroused by physical or psychological factors. The physical factors are pain, extremes of temperature, hæmorrhage, or infection. The sympathetic response is by vaso-constriction or dilatation, change in the rate of the heart beat or respiration, release of glycogen and adrenalin, etc. Emotional factors can cause similar physical effects, visceral reactions to psychological stimuli.

PART A. SYMPTOMATOLOGY

§ 691. The symptoms of disease of the nervous system may be *subjective* or *objective.* The *subjective symptoms* are those of which the patient complains to you, *e.g.*, headache, pain, giddiness, sleeplessness, numbness and tingling, a useless feeling in a limb, or double vision. The *objective symptoms*, or physical signs, are those signs noted by the patient's doctor on examination, *e.g.*, papillœdema, pupillary abnormalities, external ocular palsies, dysarthria, inco-ordination, muscular wasting, spastic or flaccid paralysis, loss of tendon reflexes, etc. Subjective symptoms occur in organic as well as in functional disease, and may or may not be accompanied by physical signs. It is impossible, without a careful physical examination, to exclude the presence of organic disease on the patient's statements alone. General lassitude, such as is met with in the neuroses, may be the first symptom complained of in an organic disease such as disseminated sclerosis, and the diagnosis is made only by finding an extensor plantar response, absent abdominal reflexes, pallor of the temporal halves of the optic discs and diminished vibration sense over the tibial malleoli. Again, a patient with an organic disease may suffer from an associated neurosis.

The chief subjective symptoms of which patients complain are:

I. Nervousness and Exhaustion. Tremulousness.
II. Giddiness.
III. Pain, Numbness and Tingling.
IV. Headache.
V. Disorders of Sleep.
VI. Loss of power or inability to control one or more limbs.

I. Nervousness and Exhaustion. Tremulousness.—Patients presenting these symptoms should on no account be diagnosed as " neurotic " until after careful and painstaking physical examination. The finding of a persistent tachycardia, an enlarged thyroid and lid-retraction with raised basal metabolic rate may reveal *Hyperthyroidism.* Such symptoms may also be complained of in individuals addicted to *Alcohol* or *Drugs*, while toxic absorption from a *septic focus*, *B. coli* infection or early *pulmonary tuberculosis* may manifest themselves first by these symptoms.

TREMULOUSNESS is a common finding in *Striatal Disease*, notably Parkinsonism, following Encephalitis Lethargica, or in Idiopathic Paralysis Agitans. The general picture of rigidity and slowness of movement may escape the doctor's notice. The tremor, in the early stages of Parkinsonism, is monoplegic or hemiplegic in distribution and eye signs may be present (see § 765). The mistake is most frequently made in post-encephalitic Parkinsonism, where one meets with a " neurasthenic " type of sequela which has an organic basis. General tremulousness is a common finding in the early stages of *General Paralysis of the Insane* (an Argyll-Robertson pupil, or tremor of the face and tongue and tremulous articulation, in such cases, are often missed) (§ 902). The early symptoms of *cerebral arterio-*

sclerosis may resemble those of functional disease, but the retinal arteries show arterio-sclerotic changes and other signs of arterial degeneration are present.

§ **692.** II. **Giddiness. Vertigo.**—Vertigo may be defined as the consciousness of disordered orientation of the body in space. True vertigo implies a subjective sensation of rotation or oscillation, either of self (subjective vertigo), or of surrounding objects (objective vertigo). In many cases the postures and movements of the limbs, especially the lower limbs in standing and walking, are ill-adjusted and unsteady. The mechanism of equilibrium is described in § 680.

Examination of Cases with Vertigo.—Enquire if the patient experiences true subjective or objective sensations of rotation. Patients use the word "giddiness" to cover a variety of vague sensations of loss of confidence, viz. nausea, sinking feelings and even losses of consciousness, which are not true vertigo. Are the symptoms constant or paroxysmal ? Enquire for a history of head injury or taking of sedative drugs. Is tinnitus present ? Test the hearing, look at the ear-drums and fundi, and test the corneal reflexes. Examine for nystagmus and note its character. Test the limbs for ataxia and past-pointing, and test the tonus of the limbs. Examine the reflexes. Examine the heart, and take the blood pressure, and test the urine for albumen. Look for septic foci ; take blood for the Wassermann Reaction, and have the Eustachian tubes inflated.

A. *The Vertigo is* Paroxysmal *and* Deafness *is present.*

Ménière's Syndrome. This disease affects persons in middle life and late middle age, men more frequently than women. A previously healthy individual is suddenly seized with intense giddiness causing uncertain gait, reeling or even falling, with nausea, vomiting, and syncope. Consciousness may be lost in severe attacks, or there may be transient loss of vision or diplopia. After the attack the patient is left with (1) diminution in hearing in one ear (nerve deafness), which is progressive, and (2) continuous tinnitus. Attacks of vertigo last minutes or hours, and occur every few days or weeks. As the deafness progresses, the attacks of giddiness diminish, and finally disappear, when loss of hearing is complete. Deafness may not be present after the first few attacks, but appears later.

The condition has been ascribed to a toxic affection of the labyrinth. Cairns and Hallpike have described gross dilatation of the endolymph system in two cases examined histologically. Ménière's original case was due to a hæmorrhage into the labyrinth, but this is a rare cause. A metabolic disturbance of fluid balance has been blamed for the condition, and some cases are relieved by dehydration treatment. Syphilis is a rare cause.

Diagnosis.—The residual deafness and tinnitus distinguish the condition from *epilepsy* and from *migraine.*

Treatment.—Sources of focal sepsis must be eradicated. Over-smoking does harm. Some patients are improved by restriction of fluids to 1½ pints daily. Blistering the mastoid may help others : a small fly blister ¾ inch square is applied for twenty-four hours. If syphilis is present it must be treated. Inflation of the Eustachian tubes should be practised in all doubtful cases. Phenobarbitone (luminal) in ½-grain doses twice or three

times daily helps many cases and should be taken with religious regularity over a period of months. The patient should not be allowed to swim, drive a car or ride a bicycle until free of attacks for six months. When the attacks are rendering the patient's life a misery, destruction of the affected labyrinth by alcohol injection into the external semicircular canal may be undertaken by an expert, or surgical division of the vestibular nerve. Both these operations usually destroy hearing.

Aural vertigo. Patients with chronic nasal infection and Eustachian obstruction may suffer from paroxysms of vertigo. The nasal infection should be treated and the Eustachian tubes inflated until the obstruction is relieved. The giddiness disappears when this end is achieved. In *Otosclerosis* the attacks of giddiness are treated as in the Ménière syndrome.

B. *The Vertigo is* PAROXYSMAL *and* DEAFNESS IS ABSENT.

The vestibular reflexes may be affected by alterations in the cardiovascular system, causing a vertigo which occurs when rising from the horizontal or sitting posture, or bending down, or on prolonged standing. In *cerebral arterio-sclerosis*, the retinal vessels may show disease and the blood pressure may be raised ; in *aortic regurgitation*, signs of aortic disease will be present, and in *Stokes-Adams'* syndrome heart-block will be present. In the vasomotor instability of the *menopause*, of *convalescence* and after *hæmorrhage*, *e.g.*, from a duodenal ulcer, vertigo of this type may occur. Cases of *emphysema* may suffer in this way after a bout of coughing. Paroxysmal vertigo may occur in *migraine*, and may be associated with teichopsia and a family history of the affection. A *petit mal* attack may manifest itself by a bout of giddiness. After *head injuries*, giddiness on alteration of posture, increased by coughing or sneezing, is sometimes a severe and persistent symptom. In many of these cases there has been contusion or laceration of the labyrinth itself. Various *toxæmias* and severe *anæmias* cause occasional vertigo.

C. *The Vertigo is* CONTINUOUS.

(1) *Suppurative ear disease is present.* Suspect ACUTE LABYRINTHITIS and look for signs of CEREBELLAR ABSCESS, or INTRACRANIAL SUPPURATION.

Acute Labyrinthitis is an extension of inflammation of the middle ear into the cochlea and labyrinth. The symptoms are rapidly progressive, with pyrexia, vertigo, deafness, and tinnitus. Coarse nystagmus on looking to the side of the lesion, hemiataxia and hypotonia together with past-pointing, are present in the ipsi-lateral limbs. In progressive cases a radical mastoid operation is often necessary. Cholesteatoma in the middle ear may be associated with cerebellar abscess.

(2) PARALYSIS OF AN OCULAR MUSCLE *is present.* Diplopia resulting from squint may cause continuous vertigo from visual disorientation.

(3) *Signs of* NEUROLOGICAL DISEASE *are present.*

If the corneal reflex is diminished or absent on one side, with unilateral deafness and tinnitus, an *acoustic neurofibroma* should be suspected. Facial weakness and blunting of trigeminal sensation may be present on the side

of the lesion and hemiataxia may be present with an extensor plantar response. Irrigation of the external auditory meatus with hot and cold water will reveal impairment of labyrinthine functions. If the patient is a young adult and the vertigo is associated with intention tremor, absent abdominal reflexes, extensor plantar responses, and diminished vibration sense over the tibial malleoli, the condition is likely to be *disseminated sclerosis*, with acute lesions in the pons.

If the patient is elderly, and sudden vertigo is associated with ipsilateral hypotonia and hemiataxia, with loss of pain and temperature sensibility over the corresponding half of the face and opposite side of the trunk and limbs, the condition is a *unilateral bulbar syndrome* from thrombosis of the posterior inferior cerebellar artery. In these cases there may be unilateral paralysis of the palate, pharynx, vocal cord and tongue, together with miosis on the side of the lesion. Signs of arterial disease will be present.

Vertigo occurs with nerve deafness, tinnitus and facial paralysis, in cases of *herpes zoster* affecting the external auditory meatus, when the lesion is in the geniculate ganglion.

(4) In Psychoneuroses, feelings described by the patient as "giddiness" are frequent. This is rarely a true vertigo, but rather a feeling of insecurity or lack of confidence. The feeling may be accompanied by sensations of unreality, either of the individual or her surroundings. Such patients tend to be asthenic, with vasomotor instability, visceroptosis and low intra-abdominal pressure. The anxieties will diminish with explanation and reassurance, and the giddiness disappears as the patient acquires more confidence. An abdominal belt will help very thin patients with poor abdominal muscles.

III. **Pain, Numbness, Tingling.**

§ **693. Pain.**—A careful observer will always investigate four important features of a pain. (1) The exact *distribution and sites of radiation.* (2) Its *character and degree*—whether throbbing, burning, stabbing, aching, etc. (3) The *factors which increase the pain, e.g.*, coughing, sneezing, deep breathing, or movement in a particular direction. (4) Its *constancy, i.e.*, whether persistent or paroxysmal. Local examination of the painful part should never be omitted. A patient's own diagnosis of "rheumatism" should not be accepted without question. The lightning pains of tabes dorsalis, which commonly precede all the other symptoms of that disease by years, may be wrongly diagnosed by the doctor as rheumatic, on insufficient examination. Painful neurological conditions are dealt with in § 816.

Secondary Carcinoma of the Vertebræ gives rise to severe and persistent root pains, without marked objective neurological signs or local abnormality in the spine. Two years or more after an operation for cancer (commonly mammary carcinoma), the patient, while making some physical effort, suffers sudden intense pain in the back. These "alarm pains" become increasingly frequent and persistent, and, with increasing cachexia

and sleeplessness, may be the only symptoms present. Radiograms of the vertebræ often appear normal. Such deposits occur mostly in the lumbar vertebræ, but signs of compression of the cauda equina (see § 798) are late, and the patient may die of secondary deposits in other organs before they appear.

Dysæsthesiæ or **Paræsthesiæ** are subjective sensations of tingling, "pins and needles," crawling sensations under the skin, numbness, burning, etc. Their distribution and constancy should always be carefully noted and whether they are accompanied by local vasomotor changes (Raynaud's Disease, § 579). Such symptoms are early and transient phenomena in a limb in *disseminated sclerosis*. The "girdle sensation" of *tabes dorsalis* is a feeling as if a tight cord were tied round the waist. Pins and needles occur bilaterally in the hands, feet and legs, in cases of *subacute combined degeneration of the spinal cord* and *polyneuritis*, accompanied by sensory and reflex changes. Where nerve trunks are compressed (*e.g.*, in *the rib-pressure syndrome*), paræsthesiæ may occur before the onset of the pain. Paræsthesiæ of peripheral nerve distribution occur in *peripheral nerve lesions* of varied etiology. Pins and needles in the hands and fingers occur in *severe anæmia* and in *myxœdema*.

§ **694. Acroparæsthesia** is a condition of numbness with tingling, pins and needles, and often disagreeable burning sensations in the hands and fingers. The fingers may be clumsy but they do not alter in colour and there is no loss of tendon reflexes, true paresis, wasting, or gross sensory loss. The intrinsic muscles of the hands may be tender and there may be slight blunting of cutaneous sensibility. The patients are commonly middle-aged women and there may be a history of endocrine disorder or debility, or harder manual work than usual. The condition does not respond immediately to treatment but eventually clears up entirely. *Treatment.* Rest in bed for 2 to 3 weeks with the elbows supported on pillows will help many cases. Patients should be told to keep the hands as dry as possible. Mild sedatives and hypnotics help some. Massage and faradism to the shoulder-girdle muscles are sometimes advised.

§ **695.** IV. **Headache.**—This is a name applied to any feeling of discomfort in the head, not necessarily pain. An exact description of the type of discomfort should always be obtained. Headache is productive of great incapacity, owing to inability to concentrate, intolerance of light and noise, irritability and anxiety occasioned by the pain. The brain itself, the pia arachnoid and ependyma are insensitive, and the pain of headache arises either in the dura mater or around the venous sinuses or in the neighbourhood of the intracranial arteries. Pain in the head may arise from irritation of the roots of the fifth, ninth and tenth cranial nerves, and of the sensory roots of the upper three cervical nerves. The pain may be referred to the cutaneous distribution of the nerves of the cervical plexus, over the scalp and bones of the head (see Fig. 186). Headache is often a manifestation of a generalised increased sensitiveness of the nervous system referred to a single nerve (*e.g.*, the supraorbital) or group of nerves. To some doctors, headaches spell only constipation or eye-strain, but these are infrequent causes of all but

episodic headaches. *Anxiety* is probably the most important cause of continued headaches.

INVESTIGATION OF A CASE OF HEADACHE.—In investigating a case of headache it is important to obtain a detailed history, especially with regard to the character of the discomfort, its site and distribution, the time of onset of the headache, its periodicity, and any accompanying signs and symptoms, *e.g.*, visual spectra, paræsthesiæ in the limbs, swelling of the cheek or puffiness of the orbit during the pain. A family history of headache should always be sought for and the importance of searching for a local cause, *e.g.*, rheumatic deposits in the occipital muscles, new growths of the skull, increased intraocular tension in glaucoma, etc., cannot be sufficiently emphasised. The cause is usually revealed, not by the stethoscope, but by special examinations—the examination of the fundi and nervous system, taking the blood pressure, testing the urine, and by X-raying the skull and accessory nasal sinuses.

Headaches are due to (A) Neurological and Local Causes, (B) General Causes, (C) Reflex Causes.

A. *Neurological and Local Causes of Headache.*

These headaches have characteristic clinical distinguishing features. They are:

(I) Anxiety Headaches.
(II) Migraine.
(III) Neuralgia.
(IV) Sinus Headaches.
(V) Meningeal Headaches (including Subarachnoid Hæmorrhage.
(VI) Syphilitic Headaches.
(VII) Traumatic Headaches.
(VIII) Headaches due to Increasing or Decreasing Intracranial Pressure (including Pituitary Tumour).

(I.) ANXIETY HEADACHES.—The abnormal sensation complained of is rarely pain; usually it is described as something worse and less bearable than pain. It may be a feeling of weight or pressure, or aching tightness of the head. Other symptoms are fatigue, listlessness, restlessness, inattention, sleeplessness. Such are the headaches most commonly met with in practice.

§ **696.** (II.) MIGRAINE (Synonyms: Hemicrania, megrim, sick headache, bilious headache).—This is an affection characterised by recurring paroxysms of intense headache with, or without, visual and sensory phenomena, and nausea with, or without, vomiting.

Symptoms.—On the day before an attack the patient may feel vague malaise and know that one is impending. The following phenomena may be present in an attack: (1) Slow visual auræ of several kinds. A black spot at the side of the visual field, hemianopia, the appearance of golden X's or Z's, or a shimmering spot which opens out into a curved zig-zag "fortification" spectrum. (2) Slow sensory auræ of tingling in one hand, spreading up the arm into the lips and tongue, or down the trunk into the leg, sometimes accompanied by weakness of the affected limbs and aphasia. These auræ are characterised by the *slowness* of their spread;

they last 10 to 20 minutes and are replaced by the characteristic headache. (3) The headache is often unilateral and temporal, later becoming bilateral. When the aura is absent the headache commonly commences in the early morning and is described as a burning, throbbing pain, which increases in intensity, accompanied by (*a*) photophobia and (*b*) pain on attempting to move the eyes. (4) Nausea or even vomiting, and sometimes abdominal pain due to tonic spasm of the colon. Cases have been described of transient unilateral blindness at the height of the headache, due to spasm of the central retinal artery, seen ophthalmoscopically. Recurring external ophthalmoplegia in association with migraine headache, so called *migraine ophthalmoplégique*, has been described, but many such cases have been found, at autopsy, to have suffered from leaking "berry aneurysms" of the circle of Willis or from intracranial tumour.

The attack lasts an hour or two, or all day. The following day the patient generally feels prostrated or exhausted. Attacks recur at varying intervals, once or twice a week in severe cases, or every few months. Recurring intense headache may be the only symptom, or the slow sensory auræ or visual phenomena may recur alone, without being followed by headache.

TRIGEMINAL MIGRAINE is a type of the malady in which there is radiation of the pain to the face or supraorbital region. This should never be mistaken for tic douloureux, for the pain never runs along the lips and tongue, the pain is never started off by eating, talking, or washing the face, and "trigger zones" are absent.

CILIARY MIGRAINE is a type of migraine in which the pain is referred to the eyeball or to the retro-orbital region. Great care should be taken in these cases to exclude glaucoma or orbital growths. Injection of the supraorbital and infraorbital nerves with alcohol, as advised by Wilfred Harris, is a useful adjunct to the usual treatment for migraine in this type of case.

Diagnosis.—Migraine-like headaches occur in tumours of the occipital lobe and in chronic nephritis with uræmia. The diagnosis of migraine is made on the recurring paroxysms of headache, the unilateral character of the headache, the accompanying phenomena, and the absence of objective signs of disease. The slowness of the aura in migraine is of great value in the diagnosis from Jacksonian or other epilepsy, as the duration of an epileptic aura is but a few seconds.

Etiology.—The disease runs markedly in families; a direct inheritance can, in most cases, be traced. In some cases a history of epilepsy in the family can be ascertained, and migraine and epilepsy may occur in the same subject. The disease may begin in childhood, but commonly shows itself just before puberty, and it continues through life until past middle age. It is commoner in women than in men, and in women there is often an exacerbation in the severity and frequency of the headaches at the menopause; after this they tend to disappear, but do not always do so. The disease is probably analogous to epilepsy. In cases of recurrent unilateral migraine, small berry aneurysms ("congenital" aneurysms) are sometimes found and these may cause subarachnoid hæmorrhage by rupturing. The disease has been ascribed, without much foundation, to a recurring unilateral hydrocephalus. Amongst the factors which excite

attacks are (1) Eye-strain, (2) Dietetic indiscretions, (3) Menstruation, (4) Exposure to cold or (5) Emotional stress.

Treatment.—The nature of the malady should be clearly explained to the patient, who may fancy that he has a cerebral tumour. He should be told at the outset that it is unlikely that his headaches will disappear completely as the result of treatment, although it is possible to mitigate the severity of the attacks and lengthen the intervals between them. These patients should avoid overstrain, cold, and dietetic indiscretions. A morning dose of magnesium sulphate gr. 60, and the administration of glucose ½ oz. in orangeade, thrice daily between the attacks, and light diet, help some cases. Refractive errors should be corrected and a dentist consulted regularly. Some cases are helped by psychotherapy. The most useful measure in treatment is the continuous administration of a drug, such as phenobarbitone, gr. i., at night, and the patient should be advised to take this regularly until he has been free from headaches for a year. Later, the dose may be reduced. Other remedies are sometimes used: (1) ℞ sod. bromide gr. x., liq. trinitrin ♏ 1, liq. strychnini ♏ 5, ac. hydrochlor. dil. ♏ 10, tinct. gelsemii ♏ 5, aq. chloroformi ad fl. oz. ½; t.i.d., p.c., and (2) Thyroid extract in gr. ½ doses, once or twice a day. A cup of strong coffee or a walk in the fresh air may stave off an attack.

In the Attacks.—Once the attack has commenced little can be done to prevent its inevitable progress. Two drugs given hypodermically are sometimes successful in aborting an attack. These are: (*a*) adrenaline hydrochloride 1/1000 solution, in 3 to 5 minim doses, and (*b*) ergotamine tartrate (femergin): 0·5 c.c. (*i.e.*, 0·25 mgm.) intramuscularly, may be repeated in 2 hours, or 1 tablet (1 mgm.) of femergin may be given orally and repeated in an hour. It should not be given to pregnant women; in the non-pregnant female it may cause uterine colic if given during the menstrual periods. Alternatively, the patient should lie down in a darkened room and take (1) cibalgin gr. 5, in tablet form, every half-hour until three doses have been taken, or (2) phenacetin gr. 15, caffeine citrate gr. 5, to be repeated at half-hourly intervals for three doses, if necessary.

(III.) NEURALGIAS.—Persistent pain over the distribution of the supraorbital nerve without objective sensory impairment, *Supraorbital Neuralgia,* is a common symptom of anxiety neurosis, although it may follow injuries to the supraorbital nerve. A severe form of recurring *Frontal Neuralgia* is met with coming on in middle life, mostly in men. In many cases it does not appear to have a psychological basis, and may yield to injection of the supraorbital nerve with alcohol. Severe frontal neuralgia may also follow ophthalmic herpes (see § 856). *Occipital neuralgia* is commonly due to rheumatic infiltration in the posterior cervical muscles, but secondary carcinoma, Pott's Disease and spondylitis, may cause similar pain. In the rheumatic cases the pain may yield to massage and applications of antiphlogistine or a mustard plaster. In severe cases the nodules should be felt for and injected with 5 to 7 minims of 90 per cent. alcohol, using a hypodermic needle and syringe.

(IV.) SINUS HEADACHES.—Disease of the nasal accessory sinuses is an important cause of pain referred to the supraorbital region or to the teeth. These headaches are intermittent, and are characteristically accompanied by œdema of the orbital tissues (in frontal sinusitis) or of the face (in antral suppuration) and are relieved by a gush of pus from the nose (§ 179). Local tenderness is often present on palpating or percuss-

ing the wall of the sinus, or, in the case of the frontal sinus, on upward pressure from the orbit on the floor of the sinus. Fœtor may be present. The sinus affected does not transilluminate clearly. Suppuration in the ethmoidal sinuses is characterised by pain over the nasal bridge, behind the eyes or over the temples. Palliative treatment consists of putting the patient to bed and applying hot fomentations to the face or forehead, and an oily solution of ephedrine to the nasal mucous membrane. A brisk purge, *e.g.*, calomel gr. 3, is administered, and aspirin gr. 10 four hourly, may be given for the pain. Relief may be obtained by inhaling the vapour of ten drops of a 25 per cent. solution of menthol in spt. vini rect. in a pint of boiling water. Surgical treatment is sometimes necessary, however.

(V.) Meningeal Headaches.—The pain in meningeal irritation is intense and severe in the occipital region and in the spine, and is accompanied by rigidity of the posterior cervical muscles and spine. There is characteristic irritability and drowsiness, passing on to coma in severe cases. The occurrence of this type of headache is sufficient indication to perform a diagnostic spinal puncture. Meningeal headaches arise from a variety of causes of meningeal irritation, *e.g.*, *Meningitis*, *Meningism* (see §§ 726, 731), the leakage of blood or pus into the subarachnoid space, or irritation of the meninges by vascular tumours, especially tumours of the vermis of the cerebellum, the fourth ventricle or base of the skull. *Sunstroke* produces sudden, severe meningeal headache (see § 508). *Subarachnoid Hæmorrhage* (see § 717).

(VI.) Syphilitic Headaches.—Headaches due to syphilitic lepto- or pachymeningitis are often nocturnal, increasing in intensity. They may be accompanied by alteration in the character of the patient, and cranial nerve palsies, *e.g.*, Argyll-Robertson pupil may be found. The characteristic changes in the spinal fluid of lymphocytosis with positive W.R. confirm the diagnosis.

(VII.) Traumatic Headaches.—Cases of Unresolved Contusion of the Brain, after head injuries, are followed by characteristic (1) localised headaches intensified by exertion, noise or alteration in posture, (2) giddiness, (3) inability to concentrate. There may be accompanying localised tenderness of the scalp and slight facial weakness, pupillary abnormalities and alterations in the reflexes may be present. The symptoms are due to cerebral œdema persisting around the contused area of the cortex. Owing to the lack of elasticity of the dura the reactionary swelling does not resolve, as in a superficial contusion, say of the skin, but may persist for many months, producing a disordered state of the cerebral circulation. Contusion headaches may be prevented by keeping patients who have had moderate or severe head injuries in bed, with complete physical and mental rest, from four to six weeks, even in the absence of signs of focal organic lesions. When present, the headaches are treated by resting the patient in bed, propped into the position which affords him greatest comfort, giving bromides gr. 10 thrice daily, well diluted, and a morning saline aperient (see also § 716).

Headache, following trauma, is one of the characteristic symptoms of *Subdural Hæmatoma* (see § 827).

Patients who have suffered head injuries may develop *compensation hysteria*, especially if there is litigation for compensation (§ 889). These cases have accompanying tachycardia, loss of weight and sleeplessness, with anxiety dreams, symptoms not present in pure Cerebral Contusion.

(VIII.) Headaches due to altered Intracranial Pressure.—The headache of Cerebral Tumour, Abscess or Chronic Subdural Hæmatoma, is paroxysmal, especially evident on waking and relieved by lying down. It is accentuated by coughing, vomiting or stooping. Vomiting may be present with the headaches, papillœdema and signs of a slowly progressive local cerebral lesion, *e.g.*, a gradually increasing hemiparesis. In tumours of the posterior fossa the headache may be produced when the patient turns his head. The cause of such headaches is uncertain, but may be attributed to local pressure on the dura or blood vessels, or to direct pressure of the brain-tissue or the growth upon the trigeminal nerve or Gasserian ganglion. Where an intracranial growth is suspected, local tenderness of the scalp or skull should always

be sought for. *Pituitary Tumour* is associated with bitemporal headache and a feeling of bursting pressure behind the eyes. Other symptoms and signs will be present (see Group XII. § 829). *Lumbar puncture* headache associated with *low* intracranial pressure may be cured by intrathecal instillation of 20 c.c. warm sterile saline.

B. *General Causes of Headaches.*

Amongst the *General* or *Constitutional Causes* of Headache are: (1) *Uræmia* in any form of nephritis. It may be suspected where there is abnormality of the urine, nocturnal frequency of micturition, general œdema, high blood pressure with recurrent epistaxis, retinal hæmorrhages or albuminuric retinopathy. (2) *Arterio-sclerosis* with high blood pressure and (3) *chronic lead poisoning* (lead encephalopathy) may be attended by severe headache. Headaches arise from the toxæmias of (4) *acute specific fevers*, (5) *malaria*, (6) *gout*, (7) *diabetes*, and (8) *alcoholism*. (9) *Constipation* is an important cause of episodic headaches. (10) The headache of *anœmia* is probably mainly toxic in origin.

C. *Reflex Causes of Headaches.*

(1) *Disease of the Eye*—glaucoma; iritis; refractive errors, such as hypermetropia and astigmatism, frequently combined with emotional strain; defective convergence. Prolonged use of the eyes, *e.g.*, for moving pictures, television, or in picture galleries, "eye-strain," produces episodic headaches. (2) *Disease of the Nose, Teeth or Ears.* (3) *Ovarian Uterine, Gastric or Cardiac Disease.* (4) *Wearing tight hats.*

HEADACHES IN CHILDREN.—In young children headaches may be due to digestive disturbances or to rheumatism. The character of the stools should be enquired into, and specimens of fæces examined. A history of growing pains or chorea or tonsillitis should be sought for and the subcutaneous tissues of the elbows and shins palpated for rheumatic nodules. Eye-strain is a frequent cause of headache in school-children. Urinary infections may cause headaches in the young. A malady analogous to migraine is "cyclical vomiting" (§ 271), and until this is treated by restriction of fats, administration of glucose and alkalies, the headache and vomiting may persist. In obese children of the Fröhlich type, persistent and intractable headaches may be present, apart from intracranial tumour. Highly-strung children may suffer from anxiety headaches just as do adults, the cause lying in some factor of the child's environment, either at home or at school.

Treatment of Headaches.—Treatment consists in determining and treating the cause of the headache. The prescribing of anodyne drugs before the cause has been elucidated cannot be too strongly deprecated.

§ **697. V. Disorders of Sleep.**—The *amount* of sleep required in healthy individuals varies with the age of the person and the individual peculiarity. In general, it may be said that infants require about sixteen hours, adolescents ten hours, the middle-aged eight, and the aged five hours. Some families and individuals are notoriously poor sleepers and do not seem to suffer in health or comfort on this account. Man can exist without sleep for about the same time that he can do without food, viz., three to four weeks, but he cannot live without it.

In *normal sleep* the power to make voluntary movements is first lost, then the use of the special senses disappears, hearing being the last to go and the easiest to evoke on arousing the patient. General muscular relaxation, with ptosis, develops, the eyeballs turning upward and becoming slightly divergent. Respiration becomes slower and noisier, and tends to periodicity in the very young and the very aged. The pulse frequency lessens, the blood pressure falls with cerebral anæmia, and the general body temperature falls. Temporarily, the knee-jerks are abolished and the plantar responses become extensor. Two important features of normal sleep are: (1) Its fixed periodicity in the rhythm of sleeping and wakefulness. (2) The sleeper can be roused from sleep to normal activity, unlike the comatose or stuporose patient.

We have no precise knowledge of the *physiology of sleep*. Pavlov believes it to be a state of *active inhibition* of the cortical mechanism. Its function is to protect the nerve-cells so that they can recuperate from fatigue and recover their normal functions. In the region of the *hypothalamus* and *the grey matter of the floor of the third ventricle*, exists a nervous mechanism intimately connected with sleep. Damage to this area, by tumours or other structural disease, usually results in excessive drowsiness.

Sleep may be (A) *Diminished in Quantity* (insomnia), (B) *Defective in Quality* (disturbed sleep), (C) *Increased in Quantity* (protracted sleep). (D) *Sleep Rhythm* may be Inverted or Disturbed.

A. **Sleeplessness (Insomnia).**—The chief causes of sleeplessness are: (1) *Psychical*, but there are many other causes. Sleeplessness may arise (2) as the result of *pain* anywhere in the body, or *discomfort*, such as is caused by flatulence in the stomach or intestine, or by dyspnœa in cardiac disease or dropsy. (3) In *febrile conditions*. (4) In *organic brain disease*. In *Encephalitis Lethargica* absolute sleeplessness, lasting several days, may be met with, especially in children. Sleeplessness is an early symptom of *General Paralysis of the Insane*, when the patient is restless or excited (later, drowsiness and apathy are common). In *cerebral arterio-sclerosis*, especially when the blood pressure is high, the patient may fall off to sleep, but wakens in the early morning unable to sleep again. (5) In many forms of *chronic toxæmia, e.g., uræmia* and *alcoholism*, sleeplessness may be present in the early stages.

The *Psychical Causes*, owing to their relatively great importance, must be considered in detail. They may be divided into three groups: (1) The patient is unable to sleep because of *anxiety* (by far the largest group). (2) The patient is unable to sleep because of some *bad habit of thought*, anxious preoccupation with affairs of the past, present or future, visualising of scenes, rehearsing of conversations, etc. (3) The patient, usually a bad sleeper, is *obsessed* with the idea of sleeplessness, or "insomnia" as he calls it. (See Anxiety Neurosis, § 886.) Persistent insomnia may be the prelude to the development of a psychosis.

Treatment of Insomnia.—(1) The *factors underlying the neurosis* should be elucidated. Bad sleepers are nearly always apprehensive that the

TABLE XLV.—HYPNOTICS.

Indications.	Hypnotic.	Dosage and Method of Administration.	Untoward Effects.
(1) Sleeplessness due to mental perturbation	Aspirin	Two 5-gr. tablets crushed and taken with warm milk at bed-time	Sweating.
	Sodium Seconal	¾–1½ gr. by mouth	Fairly short action.
	Butobarbitone (Soneryl)	3 gr. by mouth	
	Sodium Barbitone (Medinal)	3 to 15 gr., in cachets	In large doses may produce confusion on waking. Possibility of habit formation.
	Allonal	2½ to 5 gr. by mouth (tab.)	
	Dial	1½ gr., in tablet form	More powerful in its effect than medinal.
	Sodium Amytal	1-3 gr. by mouth	More powerful than those above.
	Pentobarbitone (Nembutal)	3 gr., in capsules.	
(2) Sleeplessness due to pain	Aspirin, Allonal	See above	
	Pyramidon	10 gr., in tablet form, by mouth	Continued administration of Pyramidon may cause agranulocytosis.
Pain of secondary deposits in bone	In severe cases A.P.H. Powder	A.P.H. Powder = Aspirin 10 gr. Pyramidon 7 gr. Heroin 1/12 gr.	May cause confusion on waking. Possibility of habit formation.
	Veramon	6 gr. by mouth	
	Morphine	¼–⅓ gr. hypodermically	
	Pethidine	50-100 mgm. by mouth or injection	
	Physeptone	5–10 mgm. by mouth or injection	Very effective.
(3) Sleeplessness due to motor excitement, *e.g.*, early psychosis, delirium tremens (for latter never give morphine)	Paraldehyde	60-480 ♏ in equal amount of olive oil, per rectum, or 30-240 ♏ by mouth or 4 c.c. intramusc.	
	Phenobarbitone	¼ to 3 grains dissolved in hot water by mouth, or by intramuscular injection	Confusion on waking. Erythematous rash.
	Sodium Amytal	1–3 gr. Can be repeated.	
	Somnifaine	20-40 drops (8–16 ♏) by mouth, or 2 c.c. intramuscularly.	A powerful sedative.
(4) Sleeplessness in cardiac disease.	Chloralamide	25 gr. in 1 fl. oz. of brandy	
(*a*) Without dyspnœa	Potassium bromide, Chloral hydrate	20 gr., 15 gr. In a mixture	Furring of tongue. Digestive upset. Rarely confusion.
(*b*) With dyspnœa	Morphine	1/6 to ¼ gr., hypodermically	Constipation.
(5) Sleeplessness, with dyspnœa, in acute pneumonia	Morphine, Atropine	1/6 gr., 1/100 gr. Hypodermically	
(6) Sleeplessness in febrile states	Morphine, Hyoscine	¼ gr., 1/100 gr. Hypodermically	Excitement and confusion.
(7) Senile and arterio-sclerotic insomnia	Alcohol	Given as whisky or cognac with the last meal, or before retiring	
	Blue Pill	¼ gr., at night	

lack of sleep will cause insanity, or that if they take hypnotics they will become drug-addicts. Such patients should be reassured. (2) In slight

cases *physical treatment* is prescribed—comfort, quiet, warmth, change of surroundings, hot baths followed by massage, hot packs, hot drinks, last thing at night, may suffice to restore the lost sleep habit. Heavy meals and over-fatigue, emotional or intellectual strain should be avoided late in the evening. Some sleep better at the seaside, some at a higher altitude. A period of rest in bed, away from home and its distractions and worries, helps the poorly-nourished and tired-out patient. A dry biscuit and a thermos-jug of hot milk by the bedside will sometimes help those who waken in the night to fall asleep again. (3) *Hypnotics.* (Table XLV.) In more severe cases, where the continued loss of sleep is rendering the patient panicky and less and less capable of dealing with his anxieties, the insomnia should be immediately relieved by hypnotics. The *rules for prescribing a hypnotic* are: (1) It should first be explained to the patient that he is not being drugged, and that it is better for him to obtain sleep with a mild sedative than to go on having sleepless and wearying nights without it. (2) It should be prescribed nightly *for a stated initial period, e.g.*, three nights, a week, a fortnight. (3) The *hour at which it is to be taken* must be definitely stated. The additional anxiety of having to decide when to take his hypnotic is bad for the patient. (4) If possible, the patient should not know what hypnotic he is having. (5) It must be given in adequate dosage to produce sleep. (6) If the patient says that it is losing its effect, the same dose should be given in divided quantities, every quarter of an hour, before it is decided to increase the dose or change the hypnotic. *Medinal*, in doses of 5, 7½, or 10 gr., is the best of the hypnotics for use in severe cases. *Chloral hydrate*, 3–20 grains, is a useful hypnotic, and can be given in 3–5 grain doses to children. *Bromide* is not very satisfactory as a hypnotic, as in large doses it causes confusion on waking, gastro-intestinal upset and furring of the tongue. It should be given, well diluted, combined with chloral hydrate, in 10–15 grain doses. Chloral is useful in cases of cerebral arterio-sclerosis, and should be combined with a nightly dose of blue pill, ½ grain, and a morning saline aperient. In severe cases of sleeplessness the number of hours of sleep should be charted by the nurse. Patients who sleep badly, notoriously exaggerate this symptom, and it is often hard for the physician to obtain accurate statements.

B. **Defective Sleep.**—(1) *Nightmares* or *Night-Terrors* may be due to physical or psychical causes. In *children* the cause is commonly some unhappiness in the child's domestic or school environment, an over-anxious, bullying or quarrelsome parent, or an emotional trauma. Physical causes also operate, *e.g.*, thread-worms, gastro-intestinal fermentation, adenoids severe enough to impede respiration. In *adults*, night-terrors are commonly the result of a psychoneurosis; disturbed sleep and frequent waking up may also occur in advanced cardiac disease, chronic uræmia and other toxæmias. (2) *Somnambulism* or sleep-walking is a condition in which the sleeper rises apparently asleep and behaves automatically. It is commoner in children than in adults (see Hysteria, § 888).

C. **Protracted Sleep.**—Pathological drowsiness is met with in (1) Lesions affecting the hypothalamic region and the grey matter of the floor of the third ventricle, *e.g.*, Tumours of the *pituitary stalk, encephalitis lethargica* and *trypanosomiasis* (African sleeping sickness). (2) Increasing intracranial pressure, from any progressive intracranial lesion, *e.g.*, cerebral tumour. After a *head injury* the patient may sleep for several hours. (3) Chronic toxæmias, such as uræmia and diabetic ketosis, lead to drowsiness and, later, coma. (4) Obesity, such as the obesity of *myxœdema* and *pituitary disease,* is often accompanied by sleepiness. (5) After an *epileptic* fit the patient may sleep for many hours.

In trance states of psychopathic origin the patient appears to sleep but resists if one attempts to open the eyelids. Mutism is common. The condition may be acute or subacute in onset and may last for weeks or months. It is met with in schizophrenia, in confusional states and other psychoses, and in hysteria. Recurrences are frequent.

§ **698. Encephalitis Lethargica** (Synonyms: Epidemic Encephalitis, "Sleepy Sickness") is an infection of the nervous system characterised by acute and chronic phases. The acute stages of the illness are often marked by drowsiness, ptosis, squint, diplopia and a mild febrile reaction; absolute sleeplessness or inversion of the sleep rhythm is also met with. Since the first recognition of the disease in England, in 1918, the clinical picture has varied widely in different outbreaks. In the great majority of cases seen at the present time, the initial phases pass unnoticed by the patient; there is no history of an " acute attack," and the case comes to you because of some late manifestation, such as Parkinsonism (§ 765).

Symptomatology.—After an incubation period of a few days, during which the patient feels malaise, headache, pains in the limbs, coryza or constipation, the patient is conscious of persistent double vision and increasing drowsiness whilst he goes about his work. By the time he is seen by the doctor, some hours later, lethargy has set in. The patient lies in bed for days, intensely drowsy and heedless of his surroundings. There is usually mild fever. The *lethargy* is profound in the daytime. The patient can be roused by shaking to talk intelligently or to take food, if commanded, but often falls off to sleep again with unswallowed food in his mouth. At night the drowsiness gives place to muttering occupational delirium, and hallucinations may be acted by the patients.

At this stage ocular disturbances are common, or facial palsy, of the upper or lower motor neurone type, which later clears up. The *ocular changes* are varied: (1) Pupillary disturbances of all kinds, *e.g.*, irregularity and inequality, impairment of light reaction and the reaction on accommodation. The reverse of the Argyll-Robertson phenomenon may be found, viz., loss of reaction on accommodation, with preservation of reaction to light. The pupils may be strongly contracted or dilated. (2) Ptosis is common, usually bilateral; it is often missed, as it is thought to be due to the sleepiness. (3) External ophthalmoplegias of all types, either paralysis of individual muscles or paralysis of conjugate movement.

(4) Papillœdema is rare; when present, the swelling rarely measures more than one dioptre. (5) Nystagmus and photophobia.

As the long pyramidal and sensory tracts are unaffected, there are no sensory phenomena nor paralysis of the limbs. Sometimes, however, an extensor plantar response or an absent abdominal reflex is found; very rarely a hemiplegia. Widespread muscular asthenia is common, and, owing to the profound stupor, the bed is soiled with the excreta, or retention of urine may occur. The *cerebro-spinal fluid* is clear and without clot. In a third of the cases it is normal serologically; in the others it is under increased pressure, shows a few extra lymphocytes or rarely up to 100 cells per cu.mm. (always a *pure mononuclear* increase) and normal chloride and glucose content. In a few cases the fluid is hæmorrhagic, and in these clot and polymorphonuclear leucocytes may be present owing to the contained blood. The Lange curve may be of the luetic type.

Diagnosis is made on the clinical picture and spinal fluid findings. In *neoplasm of the hypothalamic region* signs of increasing intracranial pressure will show themselves, and papillœdema is usually more marked than in encephalitis lethargica.

Rarer Clinical Types of the Malady.—(1) *Type with Insomnia.*—Despite its name, lethargic encephalitis may manifest itself by succeeding nights of insomnia or by inversion of the sleep rhythm. (2) *Myoclonic Type.*—Sudden, shock-like contractions occur in individual muscles, usually the abdominal muscles or anterior thigh muscles. This may be associated with persistent hiccough. In some cases of this type severe root pains occur (§ 775). (3) *Choreiform Type.*—Choreiform movements appear in the limbs and face, like those of rheumatic chorea. Characteristic pupillary disturbances may be present. Lethargy is absent in such cases as a rule; sleeplessness and delirium are present. (4) *Akinetic Type.*—The onset is usually subacute and the temperature only slightly elevated. Features of Parkinsonism, with poverty of movement, rigidity and tremor, gradually appear. (5) Sudden Hemiplegic cases (acute subarachnoid hæmorrhage or cerebral hæmorrhage). (6) Polyneuritic cases and Cerebellar cases are described, but they are excessively rare. (7) Epidemic Hiccough is described in § 273.

Prognosis.—The lethargy usually clears up in one to three weeks. In 40 per cent. of cases the disease is fatal. The outlook is always very serious, owing to the liability to (1) residual symptoms or (2) sequelæ (Post-Encephalitis). In the report of Parsons, published by the Ministry of Health (1928),[1] one-third of the cases died, and one-third were so seriously disabled as to be unable to continue their ordinary work. The post-encephalitic phenomena may appear as long as nine years after the original signs of the disease. The commoner manifestations of POST-ENCEPHALITIS are discussed in other sections, but in order of frequency they are: (1) Parkinsonism, with its attendant bizarre symptoms (§ 765); (2) mental symptoms, especially in children (see § 907); (3) sleep disturbance, either lethargy or insomnia; (4) oculo-gyric crises and respiratory tics; (5) adiposity, polyuria and polydipsia, hyperthyroidism, extreme cachexia, excessive salivation and sebaceous secretion

[1] "Reports on Public Health and Medical Subjects," No. 49, London, 1928.

are all met with, due to hypothalamic involvement. The ocular symptoms responsible for diplopia are usually evanescent, but the pupillary abnormalities and slight ptosis are often permanent, and, in the later stages, afford valuable diagnostic marks of the disease. The spontaneous movements of chorea or myoclonus usually clear up after months, but tremor tends to stay.

Etiology.—No *age* is exempt. The virus of epidemic encephalitis is a filter-passing organism, but so far it has not been observed or cultivated.

Treatment of Acute Symptoms.—A 2½ per cent. solution of sodium salicylate in normal saline has been injected intravenously in doses of 30–50 c.c., twice or once daily, for a week or ten days. Drugs, however, are not specific and their value is unproved. The mouth, nasopharynx and nose should be swabbed or sprayed four-hourly with 1 : 1000 potassium permanganate solution. Lethargy, if profound, is treated by *lumbar puncture* daily, or by 20–30 c.c. of 15 per cent. hypertonic saline solution intravenously, injected daily. Constipation should be relieved by enemata, and bowel wash-outs may be given on alternate days, with improvement in the general condition. The patient should be kept in bed for at least two weeks after the disappearance of constitutional symptoms or focal nervous symptoms, and should not be allowed to return to his work for a further period of six months.

D. **Disturbances of Sleep Rhythm.**—In *Encephalitis Lethargica*, especially in children, the patient falls into a heavy sleep during the day but at night-time becomes wakeful and restless, often destructive, *e.g.*, tearing the bedclothes into shreds.

§ **699.** In **Narcolepsy** the patient is periodically overcome by an irresistible desire to sleep. He can be roused, but, if left undisturbed, may slumber for half an hour or longer, waking to normal consciousness. Dreams may occur. The attacks may occur several times a day and the depth of sleep varies, as in normal sleep. There is often abnormal slowness in waking up after normal sleep. Associated with this is a phenomenon known as *Cataplexy*. Whenever the patient feels a strong emotion, after hearty laughter, after anger, or when intensely interested in something, a sudden weakness overcomes him and he sinks helpless to the ground, retaining full consciousness but unable to utter a sound. The whole attack lasts only a second or two, and, during this, the knee-jerks are abolished and the plantars are extensor in type. Narcolepsy occurs as an idiopathic disease; it may occur as a sequel of encephalitis lethargica, in cases of idiopathic epilepsy, after head injuries and in cerebral tumours, especially in the hypothalamic region. Patients with obesity and genital atrophy may suffer from narcoleptic attacks.

Treatment.—The patient should be advised not to drive any vehicle, swim or ride, or work at a height from the ground. Ephedrine hydrochloride (or sulphate) in ¼ to ½ grain doses twice daily has been recommended in the treatment of this condition. Caffeine citrate 5 grains may be prescribed twice daily to enhance the effect of ephedrine. Amphetamine sulphate, 10 mgm. given on waking or at midday, will in many cases avert the sleep attacks. Continuous daily administration of this substance over long periods is not advised. All these drugs should be given as tablets on rising and at midday. If taken in the evening they are likely to interfere with the patient's normal sleeping.

VI. Subjective feelings of **Uselessness, Loss of Power** or **Inability to control** one or more limbs are met with in the slighter degrees of paresis, or when there is loss of sense of position. Such feelings, together with

other subjective sensations, *e.g.*, diplopia, precipitancy of micturition, are characteristic of organic neurological disease.

PART B. CLINICAL INVESTIGATION

§ **700.** The acquisition of a proficient technique in examining patients, and routine in applying it, is absolutely essential for solving neurological problems. The examination must be thorough and accurate, otherwise the deductions of the examiner will be erroneous. Experience in neurological disease never absolves one from the necessity of careful examination, for the case history, without the physical signs, may be entirely misleading. A sign such as hemianopia may be carelessly missed by even an experienced observer.

General Considerations.—Ambulatory patients should be examined seated on a stool in a good light. In eliciting reflexes it is advisable to have good muscular relaxation, and the patient should lie on a couch during this part of the examination. When seen in bed the patient should be asked to walk, whenever possible, on the ward or bedroom floor for inspection of the gait.

In writing case-notes on neurological cases remember the following points: (1) *Put down your observations categorically* and systematically under the various headings. (2) *Never omit to record negative as well as positive findings:* if the pupils are equal, central, circular, and react to light directly and consensually, say so. It may be as important in the final diagnosis as the finding of a fixed pupil. (3) Only certain abbreviations are permitted. These are SJ, BJ, TJ, KJ, AJ, for supinator, biceps, triceps, knee-jerk and ankle-jerk, respectively. AC and RC may be used for ankle- and rectus-clonus. + signifies a reflex elicited, + + an exaggeration of a reflex. O an absent reflex. The signs > greater than, < less than, are also permissible (*e.g.*, K.J.'s R > L). In sensory testing C.W., P.P., V.S., J.S., and T° are used for cotton-wool, pin-prick, vibration sense, joint-sense, and temperature respectively. R and L underlined, are used to indicate Right and Left; thus R.V. = 6/6, the visual acuity of the Right eye is six-sixths.

The patient's surname should be printed in block capitals at the head of the case-sheet; the address, age and occupation should be recorded, and whether married, single or widowed. No neurological examination is complete without a full examination of other systems, including the urine. Progress notes should always be made. In the majority of cases a spinal puncture with examination of the cerebro-spinal fluid will be necessary.

The ordinary methods of clinical examination—palpation, percussion, are not available in examining the nervous system. Special methods are used, requiring special tools, and the observer should provide himself with one of the heavier types of reflex-hammer, a tuning-fork (C 256) for testing vibration sense, and a good electrical ophthalmoscope. A skin pencil for marking out areas of diminished sensation is useful.

The History.—The *mode of onset* of the symptoms is of the greatest importance in diagnosing the cause of the lesion. Thus, a hemiplegia coming on in a few seconds is commonly due to a cerebral hæmorrhage or embolism, one coming on in a few minutes or hours, to a cerebral thrombosis, whilst a hemiplegia developing gradually over weeks or months is commonly caused by a slowly-forming tumour or abscess. In a chronic malady like disseminated sclerosis, the first symptom may have occurred in the patient's youth, and neurological histories often cover the major

part of the patient's lifetime. Events should be set down chronologically, using a fresh paragraph for recording the occurrence of each fresh symptom in the story. Experience is necessary to avoid inclusion of irrelevant matter. Important points may be missed if leading questions are not asked. The examiner should never omit to ask for a history of double vision, precipitancy of micturition, frequency or incontinence, since through shyness a patient may suppress important facts. A history of double vision, elicited in response to a leading question, should not be accepted unless the patient can recall what was the first object seen as a double image. It is often necessary to obtain additional history from *relatives* or *friends*, to supplement, corroborate, or amend the patient's story. In cases of epilepsy, we should, whenever possible, obtain a description of the attack from an eye-witness.

Previous History.—A history of syphilis or venereal disease—"a sore on the penis," should be inquired for in male patients. In women tact is necessary, and you have often to rely on a story of stillbirths, or the absence of pregnancies over a long period of married life. Rheumatism or other specific fevers, "influenza," aural discharge, tuberculosis, surgical operations, *e.g.*, for malignant disease, and accidents or wounds, especially to the head or spine, may all have neurological sequelæ and should always be inquired into.

Family History is particularly important in neurological disease, as some neurological diseases, *e.g.*, Friedreich's ataxia, are heredo-familial, whilst others, *e.g.*, Amyotonia Congenita, are familial. The patient should be asked regarding the occurrence of any nervous or mental trouble in his brothers or sisters, parents, uncles, aunts, cousins, etc. The health of his wife and of his family should be ascertained.

Habits and Occupation.—Inquiry should be made as to past residence abroad. If alcoholism is suspected, the patient's own statement must be accepted with "philosophic doubt." Tobacco addiction may cause a retrobulbar neuritis. The patient's occupation often has a close bearing on his illness.

THE EXAMINATION

§ **701**. SCHEME FOR ROUTINE NEUROLOGICAL EXAMINATION. Build and general appearance. Temperature. Pulse rate. Body weight.

PSYCHICAL FUNCTIONS: (*and see Chapter XX*).

Intelligence, Attentiveness, Memory, Orientation, Emotional—Phobiæ, Hallucinations, Delusions—Sleep, Delirium, Coma.

SPEECH AND ARTICULATION.

Is the patient right- or left-handed? Aphasia, Apraxia, Articulation.

CRANIAL NERVES.

Smell—Visual acuity; Fields of Vision; Optic Discs and Fundi—Pupils; External ocular movements; Nystagmus; Oculo-pupillary sympathetic phenomena—Corneal reflexes; Sensation over face; Masseters, Temporals—Facial movements and symmetry of face—Hearing—Palate; Tongue; Sterno-mastoids and Trapezii.

MOTOR FUNCTIONS.

(1) Power; (2) Co-ordination; (3) Tonus; (4) Wasting and Fibrillation, Hypertrophy of muscles; (5) Involuntary movements and Fits.

EXAMINATION OF GAIT.

SENSORY FUNCTIONS.

Cutaneous Sensibility—(1) Touch (cotton-wool); (2) Pain (pin-prick); (3) Temperature.

Deep Sensibility—(4) Joint Sense; (5) Vibration; (6) Sensibility of Muscles and Tendons to Deep Pressure.

Stereognosis, Tactile Localisation, Compass Tests.

REFLEX FUNCTIONS.

Tendon Reflexes—Biceps, Triceps and Supinator Jerks.
Knee-Jerks and Ankle-Jerks. Presence of Clonus.

Cutaneous Reflexes—Epigastric Reflexes, Upper and Lower Abdominal Reflexes. Plantar Reflexes.

Visceral Reflexes—Micturition and Defæcation.

Tonic Reflexes—Kernig's Sign.

SPINE AND CRANIUM.

Deformities or Tenderness.

TROPHIC CHANGES.

Skin—Bed-sores, perforating ulcers.

Bones and Joints—Arthropathies, pes cavus.

EXAMINATIONS OF OTHER SYSTEMS.

SPECIAL EXAMINATIONS.

(1) Cerebro-spinal Fluid—Cells, Total Protein, Globulin, Wassermann Reaction and Lange Gold Curve.
(2) Blood—Wassermann Reaction.
(3) Radiological Examinations.
(4) Electrical Reactions of Muscles.

Psychical Functions.—You should note the patient's intelligence and attentiveness, his memory for recent and remote events, and whether he is orientated in space and time. In some cases it is necessary to test the reasoning power and to record delusions (*i.e.*, erroneous beliefs impervious to reason) or hallucinations, phobiæ (*e.g.*, fear of traffic), or obsessions. The *intelligence* and *attentiveness* of the patient can be gauged roughly when taking the history from the patient. For more accurate testing, especially in the case of children, Intelligence Tests, verbal and performance, are used.[1] Memory for recent and remote events may be tested by obtaining confirmation of the patient's history from a reliable relative. *Memory span* is tested by asking the patient to repeat six digits, *e.g.*, 4—7—1—9—5—2, or to repeat a sentence of 28 syllables, *e.g.*, "Walter likes very much to go on visits to his grandmother, because she always tells him many funny stories." *Orientation* in space is tested by asking the patient, "What place is this?" "How did you come here?" etc., and in time, by asking, "What month (or year) is this?" "How long have you been here?" Aphasia or apraxia due to organic brain lesions should never be mistaken for mental disease. (See §§ 743, 745.)

§ **702. Speech and Articulation.**—(*a*) *Aphasia and Apraxia.*—The tests for these are complicated and are described in §§ 743, 745. In certain cortical lesions the

[1] The *Progressive Matrices* test (Raven) requires no vocabulary, and will be found useful for older children and adults: vide *Progressive Matrices and the Mill Hill Vocabulary Scale*, by J. C. Raven (H. K. Lewis & Co.): and *Performance Tests of Intelligence*, by J. Driver and M. Collins (Oliver and Boyd Ltd.).

patient has difficulty in translating his thoughts into words, either spoken or written, or difficulty in comprehending spoken or written speech. This is aphasia. An apraxic patient recognises objects, *e.g.*, a key or a pipe, but cannot demonstrate how to use them, although he is aware of their proper use and may not be paralysed.

(*b*) *Articulation.*—This has to do with the peripheral speech mechanism. Dysarthric patients have difficulty "in getting their tongues round words." This is tested by getting the patient to repeat certain catch-phrases, *e.g.*, "Mutual eligibility," "West Register Street," "Baby Hippopotamus."

(*c*) The occurrence of speech areas in the left cerebral cortex in right-handed people, and in the right hemisphere in left-handed people, renders it necessary that you should know whether the patient is right or left-handed.

§ **703. Cranial Nerves.**—I. Olfactory.—You ask the patient to close his eyes. *Each nostril* is tested separately, by applying a smelling-bottle of peppermint or lavender to the nostril tested and closing the other with your finger. It is sufficient if the patient can recognise differences in odour. Odours need not be named specifically. Ammonia, or substances containing it, such as smelling-salts or sal volatile should not be used, because they stimulate the fibres of the trigeminal in the nasal mucosa—(common sensation). Ask the patient "Do you smell anything ?" "Of what does this smell ?" "Is it different from this ?"

II. Optic.—You must make three examinations:

(*a*) Visual acuity.
(*b*) Fields of vision—peripheral and central.
(*c*) Ophthalmoscopic examination.

(*a*) *Visual Acuity.*—This is tested with Snellen's test types. The patient is placed 6 metres from the card and asked to read the letters from the top as far down as he can. *Each eye is tested separately* (see § 834).

(*b*) *Fields of Vision.*—You sit exactly opposite the patient, so that your eye is at a distance of about one metre from that of the patient. In examining the right field the patient's left eye is covered. You then say, "I want you to look directly at my left eye," pointing to your left eye and closing your right eye. Now, fixing the patient's pupil with a steady gaze so that you can note any deviation of the eye you are examining, hold your clenched hands almost at arms' length at a place midway between yourself and the patient. Now tell the patient, "I want you to point to anything you see moving." Move the thumb of one hand rapidly once, and if the movement is seen, the patient will point to the moving object. The moving thumbs are in this way brought from the periphery of the visual field towards the centre, testing all four quadrants of the visual field. You can thus compare the patient's field with your own; if there is a defect he does not see the moving finger at a time when you yourself see it, provided your own visual field is full. If any defect is suspected a careful perimetric chart should be made with a reliable perimeter. *Central vision* is tested with a 5 mm. white or red object, on a black rod held in the centre of the visual field, midway between one's own and the patient's eye. For convenience, a fragment of blotting-paper stuck in the nib of a pen is often used. If a central blind spot or *scotoma* is present the patient will not see the object until it is moved radially outwards. If a field defect exists it should be charted accurately by Mechanical Perimetry or Bjerrum's Screen (§ 834).

(*c*) *Ophthalmoscopic Examination.*—The optic discs and retinæ must be examined in every case of nervous disease. Every physician must know how to use an ophthalmoscope, and to recognise papillœdema, optic atrophy, and the commoner pathological appearances in the retina and its vessels (see § 848).

III, IV, VI. Oculomotor, Trochlear, Abducens.

These nerves supplying the internal and external ocular muscles are conveniently examined together. The *Oculomotor* supplies the superior, inferior and internal recti and inferior oblique, the striped muscle of the levator palpebræ superioris, and contains efferent autonomic fibres supplying the tonic constrictor fibres of the sphincter

pupillæ and ciliary muscle. The *trochlear* supplies the superior oblique muscle and the *abducens* the external rectus alone. The *Cervical Sympathetic* supplies the tonic dilator fibres of the pupil, the unstriped part of the levator palpebræ superioris and unstriped muscle at the back of the orbit.

(*a*) *Pupils.*—You must note if the pupils are equal, central, circular, oval, or irregular in outline. Observe their reactions to light, tested directly, consensually and on accommodation. Examine the external ocular movements for paresis and diplopia, look for nystagmus and note any inequality in the size of the ocular fissures, proptosis or enophthalmos (sinking of the eye into the socket). *The normal pupil* dilates to shade, and contracts briskly when light falls on the same eye (direct reflex) or on the opposite eye (consensual reflex). The *light reflex* is best tested by covering and unshading first one eye of the patient and then the other as he looks at the light. Each eye must be observed separately. Watch the effect on the pupil of shading and uncovering the opposite eye. Another way of testing the pupillary reflex to light is: turn the patient's face away from the light and (observing one pupil at a time) throw a beam of light from an electric torch first into the one eye and then into the other. Loss of direct reflex to light with preservation of pupillary contraction to accommodation constitutes the *Argyll-Robertson phenomenon.* Test the *accommodation-convergence reaction* (near reflex) of the pupils by asking the patient to look first at a distant object in the room or out of the window, and then suddenly to look at your forefinger which is held about a foot from his eyes. The normal pupil contracts briskly as the eyes converge.

Tonic pupils (Saenger) contract very slowly on accommodation. The contraction is long sustained and may last for half a minute or more. *Fixed pupils* react neither on accommodation nor to light stimuli.

(*b*) *External Ocular Movements.*—Steady the patient's chin with your left hand, in order to fix the head. Then raise the forefinger of your right hand at least one metre from the patient's eyes and say, "I want you to follow my finger closely with your eyes." Move the finger to the extreme right, and pause in order to see if there is lack of movement in one or other eye, or the presence of nystagmus. Test both eyes together, making the patient follow the finger to the extreme left, pausing again, and so also upwards and downwards. Ask in each case if the vision is clear or blurred.

(*c*) *Nystagmus* is an involuntary rhythmic oscillation of the eyeballs (§ 847) usually appearing when the gaze is directed to a fixed point, *e.g.*, the examiner's finger, at a distance from the rest-point of the eyes. This is "fixation" nystagmus, in contrast to the much rarer nystagmus which occurs when the eyes are directed straight forwards. The movement may be quicker in one direction than in the other. The *quick phase* is taken to indicate the direction of the nystagmus. Nystagmus may be *coarse* or *rapid, horizontal, vertical* or *rotatory.* You should learn to recognise the infrequent blinking component of the "Parkinsonian Mask."

V. Trigeminal.—This nerve supplies sensory fibres to the anterior part of the scalp, eyes, face, nose, mouth, and parts of the ear and tongue, as well as the dura mater (see § 856). The motor root supplies the muscles of mastication, masseter, temporals, pterygoids, mylohyoid, anterior belly of digastric, tensor palati and tensor tympani muscles.

You must make three examinations: (*a*) corneal reflex, (*b*) sensation over face, (*c*) the muscles of mastication.

(*a*) The *corneal reflex* is tested by lightly blowing on the cornea and watching for the bilateral reflex blinking, or by asking the patient to look upwards and touching the cornea from below with a long pointed strand of cotton-wool. The reflexes should be compared on the two sides. Care should be taken to touch the cornea, not the conjunctiva. (*b*) *Sensation over the face* is tested with cotton-wool, pin-pricks and tubes of hot and cold water. Where there is diminution or loss of sensation, this is mapped out with a skin pencil, working from the dull to the sensitive area. (*c*) *The muscles of mastication* are tested by asking the patient to clench his jaws, when the masseters and temporals can be palpated as they contract on the two sides. In

unilateral lesions, when the patient opens his mouth, the jaw deviates to the side of the lesion, being pushed over by the unantagonised external pterygoid muscle of the opposite side. Wasting of the masseters or temporals should be looked for.

VII. FACIAL.—Test the *voluntary movements* of both the upper and the lower face and the reflex *emotional movements, e.g.*, facial movements on smiling. Taste on the anterior two-thirds of the tongue is conveniently tested with this nerve, as taste fibres for this part of the tongue are distributed with the chorda tympani (Fig. 193).

Voluntary movements of the *upper face* are tested by asking the patient to wrinkle up his eyebrows and screw up his eyes. Slight degrees of weakness can be observed by the difference in burying of the eyelashes on the two sides, or by comparing the effort needed to open with your thumb the screwed-up eyelids. Voluntary movements of the *lower face* are tested by asking the patient to show his teeth, blow out his cheeks, or whistle. Slight degrees of facial weakness are shown by widening of the ocular fissure on the affected side, and flattening of the nasolabial fold. In upper motor neurone lesions only the lower face is affected.

Emotional movements are tested by asking the patient to smile and noting the difference in the angles of the mouth, or they may be observed during the general examination. *Taste* is best tested with a weak galvanic current; the wire electrode on the taste-sensitive areas produces a metallic taste. Or the patient is asked to protrude his tongue and to keep it out, and to nod if he tastes anything. Powdered sugar, salt, citric acid, are then rubbed on the tongue with a clean glass rod. If the patient tastes it he nods and is allowed to withdraw the tongue and describe the taste.

VIII. AUDITORY.—The sensory or cochlear component subserves *hearing*, the vestibular non-sensory component subserves reflexes concerned with equilibrium. (*a*) Before testing *hearing* examine the external auditory meatus to make sure that it is not blocked with wax. Whispered voice sounds are used, *e.g.*, "Charing Cross," "Waterloo," at a distance of one metre. Test the ears separately, the opposite ear being rapidly opened and closed by intermittently pressing the tragus into the meatus with your forefinger. Objectively a lesion of the nerve (nerve-deafness) may be differentiated from middle-ear deafness by the following tests: *Weber's Test:* You place the base of a vibrating tuning-fork (C 256) on the patient's forehead. In nerve deafness the sound is best heard by the patient on his normal side, while in middle-ear deafness, he hears it best on his affected side. The *Rinne Test:* Air-conduction is compared with bone-conduction. Normally, a vibrating tuning-fork is heard better when it is held slightly away from the meatus than when its base is placed on the mastoid of the ear tested. In nerve deafness both air and bone-conduction are reduced; in middle-ear deafness the fork is heard better when it is placed on the mastoid of the deaf ear.

(*b*) Testing the vestibular part of the nerve is described in § 860.

IX, X, XI. GLOSSOPHARYNGEAL, VAGUS, AND SPINAL ACCESSORY.

These nerves are intimately related in their central connections; they leave the skull by the jugular foramen and are conveniently tested together. (*a*) *Sensory tests:* Tickling the soft palate or posterior pharyngeal wall with cotton-wool on the end of a probe will normally produce reflex movements (Glossopharyngeal). Fibres of the glossopharyngeal nerve supply taste to the posterior third of the tongue. (*b*) *Motor Tests:* (1) *Palate:* The patient is asked to open his mouth and say, "A-Ah." When the patient phonates the soft palate will rise in the mid-line; if one side is paralysed the palate will be deviated to the sound side. Bilateral palatal palsy produces a characteristic nasal intonation, with regurgitation of fluids through the nose, on swallowing. (2) *Pharynx:* In unilateral paralysis, the posterior pharyngeal wall moves like a curtain pulled over to the sound side ("curtain movement") when the patient phonates. (3) *Larynx* (see § 164). (4) *Sterno-mastoids and Trapezii:* Examine the sterno-mastoids by asking the patient to turn his head forcibly to the right and then to the left, or by asking him to push his forehead downwards against

the resistance of the palm of your right hand. The trapezii are tested by asking the patient to shrug his shoulders up to his ears against the resistance of your hands placed lightly on his shoulders. In all cases the muscles should be inspected and palpated for wasting.

The visceral functions of this group of nerves are described in the examination of the other systems.

XII. HYPOGLOSSAL.—Ask the patient to protrude his tongue and to push it first into his right cheek and then his left. The protruded tongue is examined for spasticity, fibrillation, atrophy, or wrinkling. If one side of the tongue is paralysed and the patient attempts to protrude the organ, the tip is pushed round to the paralysed side, in a sickle-shaped curve, by the healthy side.

§ **704. Motor Functions.**—It is advisable to have the patient in pyjamas or stripped in a dressing-gown. First, you examine the upper limbs, then the trunk, and then the lower limbs, and the results are recorded in that order. You should note especially if the limbs are steady and strong; also examine their tonus, and look for wasting and fibrillation or hypertrophy of muscles.

(1) POWER.—The hand-grips are conveniently tested by crossing one's forearms and giving your three middle fingers, shaped in the form of a cone, to the patient to squeeze as hard as he can. The flexors of the fingers can be tested by asking the patient to hook the flexed fingers of his right hand round the flexed fingers of your right hand and then you attempt to extend his fingers against resistance. The dorsiflexion of the feet is similarly tested against resistance by asking the patient, when recumbent, to cock his feet upwards. You then attempt to plantar-flex them whilst he resists. In these tests the power is compared on the two sides. Each joint and each movement should be tested separately, fixing the proximal part of the limb and instructing the patient to perform various movements—flexion, extension, rotation outwards, inwards, etc., separately. If the weakness is marked, support the limb in the optimum position for action of the particular muscle or group of muscles you are investigating. Inability to relax the handgrip for some seconds is characteristic of *tonic innervation* or *myotonia*.

(2) CO-ORDINATION.—In the "finger-nose" test the patient, keeping his eyes open, moves his forefinger alternately to his nose and then to the examiner's finger which is held about one metre in front of him. He is asked to repeat this movement several times. When "intention tremor" is present the movement is jerkily performed and a coarse oscillation of the forearm and hand appears just before the objective is reached. When ataxia is present the finger misses the nose by a greater or smaller interval. You should notice if the unsteadiness is increased when the patient shuts his eyes. Co-ordination of the fine finger movements is tested with the "thumb-finger" test, the patient being asked to touch the tip of each finger in rapid succession (beginning with the fourth) to the tip of the thumb of his same hand. Other useful tests are picking up a pin, fastening the buttons of a coat, etc. In the "heel-knee" test the patient is placed in the recumbent position and is asked to place the heel of one foot on the opposite knee and to slide the heel slowly down the front of his shin. Here, too, you should notice if ataxia is increased on shutting the eyes. Other tests in the lower limbs are made with examination of the *gait*. Special cerebellar tests are described in § 812.

(3) TONUS.—*Hypotonia* is tested by shaking the relaxed limbs like a flail and comparing the resistance of the two sides, or attempting to fling the patient's hand on his chest. In another test the hands are passively dorsiflexed and the angle made with the forearm on the two sides compared; the knee-joints are passively hyperextended, etc. *Hypertonus* or rigidity is of three main types: (*a*) "Clasp-knife" rigidity; (*b*) "Cog-wheel" or "Lead-Pipe" rigidity; (*c*) Hysterical Spasm.

(*a*) "Clasp-knife" rigidity is seen typically in a residual hemiplegia. The limb is flexed at the elbow and pronated. When you attempt to undo the flexion, resistance is encountered until the elbow is almost extended, when the resistance suddenly "gives" as in the opening of a clasp-knife. This is characteristic of pyramidal disease. (*b*) "Lead-pipe" or "Cog-wheel" rigidity is characteristic of extra-pyramidal disease and is seen typically in Parkinsonism (§ 765). When you attempt to extend the elbow-joint, resistance is felt like the bending of a piece of lead-piping. Or on attempting to flex and extend the patient's wrist-joint, a feeling like turning a cogged wheel is met with. (*c*) In Hysterical Spasm the prime-movers and antagonists are simultaneously innervated by the patient so that little or no movement results—*e.g.*, when he attempts to use his hamstrings, the quadriceps, instead of relaxing reciprocally, tighten up. The spasm increases with the amount of force used to overcome it.

(4) WASTING AND FIBRILLATION. HYPERTROPHY.—By inspection and palpation you observe if the muscles are increased in bulk (hypertrophy) or wasted (atrophy). Sometimes hypertrophied muscles are feeble in their performance (pseudo-hypertrophy). The names of the affected muscles are recorded. You should carefully inspect all aspects of the limb and the back as well as the front of the patient. Whenever wasting is present you should look for *fibrillation* in the atrophied muscles. This is a flickering or quivering of muscle-fibres or bundles, best seen when the muscles are relaxed. Direct percussion of a muscle with a reflex hammer may show in certain pathological conditions the appearance of a dimple, which is persistent for some seconds—*myotonia on percussion.*

(5) INVOLUNTARY MOVEMENTS.—*Tremor* is best seen when the arms and fingers are extended. It may be coarse or fine. Tremor never exists in flaccid limbs; it usually ceases during sleep, and can often be controlled by an effort of will. *Choreic movements* are irregular, non-rhythmic, spontaneous but purposeless movements of groups of muscles. In organic disease they are more marked on one side than the other, and in the facial muscles they occur bilaterally. *Athetoid Movements* (athetosis) are slow, irregular, writhing movements, seen in hemiplegic limbs, more often in children than in adults (Fig. 177). The limbs are never completely paralysed: the movements are more marked peripherally and usually greater on one side than the other, and are bilaterally distributed in the face. The movements are intensified by emotion and by voluntary movements, and, between the accesses of mobile spasm, the limb is generally hypotonic. *Myoclonus* is a sudden shock-like contraction occurring regularly or irregularly in various muscles. *Tics* are sudden clonic jerks of a stereotyped character occurring in people of neuropathic constitution, increased by emotion.

(6) ELECTRICAL REACTIONS OF MUSCLES.—These are described in § 708.

§ 705. Examination of the Gait.

Except in patients confined rigidly to bed the gait should always be examined. It is frequently forgotten by students. The patient is asked to walk away from you to a given point, to turn round and then come back. It is advisable to pull or pin up the clothing or pyjama-trousers so that as much as possible of the legs can be seen. The patient walks on a strip of carpet with bare feet. You must look for dragging of the lower limbs, reeling or tottering, especially in turning, whether the patient deviates from the straight line and, if so, to what side he deviates, and whether he swings both arms as he walks. Where cerebellar or posterior column disease is suspected you ask the patient to heel-and-toe a straight line, to stand or hop, first on one leg and then on the other. Where myopathy is suspected the patient (usually a child) is laid flat on the floor and asked to stand upright. The various motions performed in

accomplishing this are characteristic. The following gaits can be recognised clinically:

(1) In *Spastic Gait* the toes are dragged on the ground. This occurs in pyramidal disease, *e.g.*, disseminated sclerosis, residual hemiplegia. Where extensor rigidity is combined with adductor rigidity, a "cross-legged" or "scissor-gait" is met with, the patient walking on the toes, *e.g.*, in cerebral diplegia in children.

(2) *Spastic Ataxic Gait* is met with in combined disease of the pyramidal tracts and posterior columns, *e.g.*, disseminated sclerosis, subacute combined degeneration, spinal tumour.

(3) *Ataxic or Reeling Gait* occurs in cerebellar, vestibular, or posterior column disease, *e.g.*, cerebellar tumour, Friedreich's hereditary ataxy, Ménière's disease, or tabes dorsalis.

(4) *A Festinant or Shuffling Gait,* in which the patient glides forward with little running or shuffling paces, occurs in striatal disease, *e.g.*, Parkinsonism. In this condition the normal swing of the arms is lost and the patient turns *en bloc.* It is seen in the general muscular rigidity of cerebral arterio-sclerosis.

(5) *Waddling Gait* is met with in conditions of weakness of the pelvic muscles, *e.g.*, myopathy; or from deformities, *e.g.*, congenital dislocation of the hip, or dwarfism.

(6) *High-stepping Gait* occurs in conditions of foot-drop, *e.g.*, polyneuritis; or from loss of joint sense, as in tabes, in which the feet are lifted high and the heels banged on the ground.

(7) *Jaunty Gait* is met with in chorea.

(8) *Limping Gait* may result from poliomyelitis or any injury or joint affection confined to one side.

(9) *Bizarre Gaits,* in which the patient walks with bent knees or trunk, or in zig-zag fashion, are seen in hysteria. A gingerly insecure gait, where the patient seeks support from the walls, furniture or the observers, is common in that disease.

§ **706. Sensory Functions.**—In order to make your sensory testing accurate it is desirable to gain the intelligent co-operation of the patient, and each test should be briefly explained before it is carried out. The room should be as quiet as possible and the patient's eyes closed in order to shut out extraneous stimuli.

The following rules should be observed: (1) Test each variety of sensation separately over the whole body before proceeding to the next; (2) Always compare the sensibility over corresponding areas on the two halves of the body; (3) Whenever possible chart the findings on an outline diagram; (4) Avoid suggesting the presence of a sensory change to the patient by the manner in which commands or questions are put. The following are useful approaches which suggest little to the patient: "Shut your eyes and say 'Yes' every time I touch you" (C.W.). "What does this feel like?" (P.P.). "Is there any difference in the feeling here, and here?" (P.P.).

CUTANEOUS SENSIBILITY.—(1) *Touch* is tested with a wisp of long-fibred cotton-wool. In mapping out anæsthetic areas, proceed from the area of impaired sensibility towards normal skin and from below upwards on the trunk. The point at which sensation becomes normal is recorded on the skin by a single dot made with a skin pencil. A number of these dots are made, and they can subsequently be connected, as in a graph, to outline the area. An anæsthetic area so mapped out should be recorded on the chart and shaded. Vertical hatching is used conventionally in diagrams for recording impairment of touch.

(2) *Pain* is tested with the prick of a sharp pin. In order to obtain a uniform stimulus the pin should be held with the point just projecting between the pads of the thumb and middle finger. *Dragged pin* is used to map out areas of hyperæsthesia, the pin being lightly dragged across the skin from the less to the more sensitive area. Loss of pain sensation is termed *analgesia* and is represented in outline diagrams conventionally by horizontal hatching; *hyperæsthesia* or exalted sensibility to pain is represented by a series of small crosses. The distinction should be made between "impaired sensibility" and "total analgesia." In mapping out sensory levels on the trunk remember that the segmental distribution of the spinal nerve roots runs downwards anteriorly, and upwards posteriorly, towards the mid-line. Horizontal upper levels of sensory loss are found only in hysteria and are produced by suggestion. The hysterical nature of a totally anæsthetic area may sometimes be demonstrated by Janet's "Yes-No" test. The patient is instructed to close his eyes and say "Yes" every time he feels a pin-prick and "No" every time he does not feel it. In hysteria the patient will say "No" every time he is touched over the apparently totally anæsthetic area, thus demonstrating that he is really able to feel but does not comprehend that he feels.

(3) *Temperature* is tested with tightly-corked test-tubes of cold and warm water (60° C.). The results are recorded on outline diagrams using conventional oblique hatching (Rt. to Lt. = Loss to Hot; Lt. to Rt. = Loss to Cold). Loss of sensation to temperature is called Therm-anæsthesia. In centrally situated diseases of the brain-stem and cord, such as Syringo myelia, the patient may feel the lightest touches with cotton-wool but cannot appreciate painful or thermal stimuli—this was called by Charcot "*dissociated anæsthesia.*"

DEEP SENSIBILITY.—(4) *Joint sense* comprises the sense of passive movement of the joint surfaces on one another, and the sense of position. In testing, the patient closes his eyes, or they are covered up. In order to avoid sensations of pressure, you grasp the digit you are testing, *laterally*, with your thumb and forefinger. *When testing sense of passive movement* wait a few moments, then move the joint gently, having previously asked the patient to say "Yes" when he feels any movement. In testing *sense of position* ask the patient to say "Up" or "Down" every time you move the joint. You then passively move the joint in either direction, waiting for the patient's reply after each movement. Corresponding fingers and toes are tested on the two sides.

(5) *Vibration* is tested by placing a low-pitched tuning-fork on the radial styloids and tibial malleoli. Other bony prominences may be used. Vibration sensibility may be diminished or lost very early in disease of the posterior columns or posterior roots.

(6) *Sensibility of Muscles and Tendons to Deep Pressure.*—An increased sensitiveness of the calf muscles to deep pressure of your thumb is commonly met with in polyneuritis—*i.e.*, the *deep muscular sensibility* is increased. In tabes dorsalis, as an early sign, the normal sensibility of the tendo Achillis to pressure may be diminished or lost (Abadie's sign).

Stereognosis is tested by placing various objects—*e.g.*, key, coin, rubber, in the hand or against the sole of the patient, whose eyes are closed, asking him to describe the shape, size and consistency of the object used. For true astereognosis to be present, cutaneous sensibility in the hand or foot tested should not be impaired. *Tactile localisation* (topognosis) is tested by asking the patient, who has his eyes closed, to point to where he has been touched with a pin. Normally the localisation

is exact to a fraction of an inch. *Compass Tests* are performed with small blunt-pointed calipers. The patient, who has his eyes closed, states whether he has been touched with one or two points, while the distance between the limbs of the calipers is gradually narrowed.

§ 707. Reflex Functions.

In routine examinations you must test the TENDON REFLEXES, certain CUTANEOUS REFLEXES, including the PLANTAR REFLEXES, and you must note the condition of certain VISCERAL REFLEXES, *e.g.*, micturition, defæcation. TONIC OR POSTURAL REFLEXES, *e.g.*, Kernig's Sign, are also used in clinical diagnosis.

1. **Tendon Reflexes.**—These are elicited by percussing the tendons of insertion of certain muscles.

	Method of Eliciting.	*Response.*	*Segmental Distribution.*
Biceps-Jerk.	Tapping biceps tendon.	Biceps contracts.	C5–6.
Triceps-Jerk.	Tapping triceps tendon.	Triceps contracts.	C6–7.
Supinator-Jerk.	Tapping above radial styloid.	Supinator longus contracts.	C6–7.
Knee-Jerk.	Tapping patellar tendon.	Vastus internus etc. contract.	L2–4.
Ankle-Jerk.	Tapping tendo Achillis.	Calf muscles contract.	S1–2.

In UPPER MOTOR NEURONE disease these reflexes are exaggerated and may be accompanied by sustained *clonus*, a rhythmic series of involuntary muscular contractions, produced by the sudden stretching of the tendon. In LOWER MOTOR NEURONE disease and in the MYOPATHIES, the deep reflexes are diminished or abolished in the affected muscles.

The *Biceps-Jerk* (C5–6) is elicited by supporting the patient's forearm with his elbow loosely flexed. Your thumb is placed over the biceps tendon and the thumb percussed with a hammer. The resultant jerk of the biceps is both felt and seen. The *Triceps-Jerk* (C6–7) is then investigated by abducting the patient's arm loosely and percussing the triceps tendon. The *Supinator-Jerk* (C6–7) is elicited by tapping just above the radial styloid, the patient's forearm supported in a semi-supinated position, the elbow loosely bent to a right angle. The resultant contraction of the supinator longus and flexors of the elbow is looked for. In testing the knee-jerks and ankle-jerks it is best to have the patient in the recumbent position to ensure muscular relaxation. To elicit the *Knee-Jerks* (L2–4) the recumbent patient's knees are slightly flexed, resting loosely on your arm, while you percuss the patellar tendons on the two sides and look for the resulting contraction of the vastus internus. To elicit the *Ankle-Jerk* (S1–2) the lower limb is then rotated outwards at the hip and the knee-joint slightly flexed, the sole is grasped and slightly dorsiflexed to stretch the tendo Achillis and the tendon percussed. If difficulty is encountered in eliciting the ankle-jerks in this way the patient should be asked to kneel on a *padded* chair with the calf muscles relaxed and the feet dangling over the edge. When the tendo Achillis is percussed a brisk contraction of the calf muscles results, which can be felt as well as seen if you are slightly dorsiflexing the foot with your left hand to stretch the tendon. In eliciting deep reflexes the responses on the two sides should be compared with the greatest care and noted. Always use the *minimal stimulus*, especially when testing knee-jerks. Never use much force, otherwise no accurate comparison of

the reflexes on the two sides can be obtained. In testing sluggish knee- and ankle-jerks it may be necessary to use *reinforcement*. This is achieved by asking the patient to look to the roof and clench his hands tightly, or he is asked to attempt to pull apart the interlocked fingers of the two hands. The presence of sluggish deep reflexes may thus be evident.

RECTUS-CLONUS is elicited in spastic limbs by sudden downward traction on the patella, with the knee extended. ANKLE-CLONUS is elicited with the knee passively flexed; the ankle is then suddenly dorsiflexed by light upward pressure on the sole. True clonus is always sustained and accompanied by an extensor type of plantar response.

2. **Cutaneous Reflexes.**—These are elicited by stimulating certain areas of skin or mucous membrane. The cutaneous reflexes of greatest practical importance are the Plantar Reflexes and the Abdominal Reflexes. These must be tested in every case. In upper motor neurone lesions these cutaneous reflexes disappear. This is most strikingly seen in the disappearance of the abdominal reflexes, first the lower and then the upper, in pyramidal disease.

The EPIGASTRIC (Th6–8) and UPPER (Th8–10) and LOWER (Th11–12) ABDOMINAL REFLEXES are elicited by stroking the lower anterior chest wall and the upper and lower quadrants of the abdominal wall respectively on the two sides. These reflexes may not be elicited when the abdominal wall is obese or flaccid. In the abdomen of a healthy young adult they are constantly present. In pyramidal disease these reflexes are diminished, tire easily or are lost on the affected side. The lower reflexes go before the upper. An absent abdominal reflex is an important early sign of disseminated sclerosis. In suspected disease of the cauda equina or lowest segments of the cord you have to test three other cutaneous reflexes: the Cremasteric (L1–2), Bulbocavernosus (S3–4), and Superficial Anal (S4–5).

The CREMASTERIC REFLEX (L1–2) is the reflex drawing up of the testis, on downward stroking or firm pressure, applied on the inner side of the thigh. The BULBOCAVERNOSUS REFLEX (S3–4) gives valuable information about lesions of the third sacral segment. It is elicited by placing one finger on the perinæum and pricking the dorsum of the glans penis. Normally, the bulbous urethra can be felt to contract briskly. The SUPERFICIAL ANAL REFLEX (S4–5) is obtained by watching for the contraction of the external sphincter when the skin of the perinæum is pricked.

A GRASP-REFLEX is elicited in the contralateral palm in lesions of the posterior ends of the first and second frontal gyri. To obtain this you draw your fingers across the patient's palm near the thenar eminence. The patient's fingers contract reflexly, and, when you attempt to withdraw your hand, the involuntary tonic contraction of the fingers increases, and may take seconds to relax.

The PLANTAR REFLEXES are of the utmost importance in clinical neurology, and the student should be thoroughly conversant with the correct technique of eliciting the responses. The patient should be in the recumbent position with the lower limb slightly rotated outwards at the hip and the knee slightly flexed. The feet should be comfortably warm. Normally, firm stroking along the *outer* border of the sole of the foot with a key or the end of a penholder produces flexion of the great toe—*flexor plantar response*. In pyramidal disease, however, when we stroke the outer margin of the sole there results a dorsiflexion of the great toe, associated with dorsiflexion and fanning of the other toes—*extensor plantar response* (Babinski). This extensor movement is part of a general withdrawal reflex of the whole lower limb from a painful stimulus and is always accompanied by an associated contraction of the hamstring muscles. When, therefore, the plantar responses are equivocal, the hamstrings should be palpated for contraction, while the sole of the foot is being stimulated.

	Method of Eliciting.	*Response.*	*Segmental Distribution.*
Epigastric.	Stroking lower anterior chest wall.	Epigastrium dimples.	Th6–8.
Upper Abdominal.	Stroking below costal margin.	Rectus abdominis contracts.	Th8–10.
Lower Abdominal.	Stroking above Poupart's ligament.	Obliquus abdominis contracts.	Th11–12.
Cremasteric.	Stroking inner side of thigh.	Testis is drawn up.	L1–2.
Bulbo-cavernosus.	Pricking dorsum of glans penis.	Bulbo-cavernosus contracts.	S3–4.
Superficial Anal.	Pricking skin of perinæum.	External anal sphincter contracts.	S4–5.
Plantar.	Stroking outer border of sole of foot.	Dorsiflexion of hallux with fanning of other toes, etc.	L5–S2.

The *flexor* plantar response is associated with plantigrade functions associated with standing and walking. It is a cortical reflex. In infants, who have not learned to walk, the normal plantar response is of the extensor type. This extensor reflex is of spinal origin, and, in later life when the infant learns to walk, is normally inhibited by cortical control. When the cortical control is removed by pyramidal disease the more primitive spinal extensor response is re-established. In pyramidal disease the receptive field for this reflex spreads over the skin of the whole limb, and the reflex can be obtained by pinching the skin almost anywhere on the lower limbs. An *extensor* response may be observed during sleep or deep coma from any cause, and after epileptic fits.

3. The **Visceral Reflexes** of clinical importance are those concerned with micturition and defæcation.

MICTURITION.—(*a*) Precipitancy of micturition is a frequent early symptom of cord lesions, *e.g.*, disseminated sclerosis, slow cord compression, etc. (*b*) Difficulty in commencing micturition and dribbling incontinence of urine, especially at nights, occurs in tabes dorsalis when the bladder is anæsthetic and distended. (*c*) Retention, with overflow dribbling, occurs in coma, or during the initial three weeks of spinal shock following an acute transverse cord lesion, *e.g.*, myelitis, fracture-dislocation; and in the later stages of total transverse cord lesions. (*d*) Involuntary periodic micturition may occur, in which the bladder empties reflexly but never completely. (See § 690: innervation of the bladder.)

DEFÆCATION.—Incontinence of fæces is commonly due to anæsthesia of the rectum from paralysis of the afferent nerves from the rectum, *e.g.*, in tabes or lesions of the cauda equina. In such cases, the internal anal sphincter, if felt by rectal examination, is flaccid. In spinal cord lesions, above the spinal centre in the conus, the internal sphincter retains its tone, and there is intermittent rectal incontinence, the patient being aware of the passage of fæces. Mentally confused patients are commonly incontinent.

4. Certain **Tonic** or **Postural Reflexes** are used clinically. In cases of suspected meningitis or meningeal irritation, you test for reflex tonic

contraction of the hamstrings (Kernig's Sign) and posterior cervical muscles (Brudzinski's Sign). *Kernig's Sign* is a reflex tonic contraction of the hamstring muscles, made evident by passively flexing the hip to a right angle and at the same time extending the knee. *Brudzinski's Sign* is a tonic neck reflex. When the head is passively flexed on the chest the lower limbs become flexed at the hips and knees. In hemiplegia, rotation or lateral flexion of the head towards the paralysed side may cause extension of the paralysed limbs. Movement of the head to the normal side has the reverse effect. This is *Magnus and de Kleijn's tonic neck reflex.*

Spine and Cranium.—Examine the patient's spine and cranium for *tenderness* or *deformity.* Neglect of this procedure may lead to your missing the fact that a patient's paraplegia is due to early Pott's disease.

Trophic Changes.—The *skin* should be examined for perforating ulcers and bed-sores, and the *bones and joints* for arthropathies, *e.g.*, Charcot joints; and deformities, *e.g.*, pes cavus.

EXAMINATIONS OF OTHER SYSTEMS.

The Cardiovascular, Respiratory, Alimentary and Genito-Urinary Systems should be examined and the urine tested in all cases.

OTHER SPECIAL EXAMINATIONS.

(1) *Cerebro-spinal Fluid.* It is necessary to examine the spinal fluid in many cases of neurological disease, and you should thoroughly familiarise yourself with the technique of performing *Lumbar puncture* (§ 919). Manometric observations on the spinal fluid-pressure must be made during the puncture, and the effect of compression of both jugular veins duly noted. The naked-eye appearance of the fluid should be observed in all cases. In sending fluids to the laboratory the following tests are made as routine: (1) Cell-count, (2) Total Protein, (3) Globulin content, (4) Wassermann reaction, (5) Lange's Gold Reaction (Gold Curve). Where infection of the nervous system is suspected the spinal fluid must be examined bacteriologically as well as serologically and the chlorides and glucose content estimated. *Cisternal* (or *Ventricular*) *puncture* (§ 919) should not be practised by those unfamiliar with the technique: it is not without serious danger.

(2) *The Wassermann Reaction in the Blood* is often necessary in diagnosis.

(3) *Radiological Examination* of the Skull (stereoscopic) or Vertebral Column may be necessary. The technique of encephalography and ventriculography is outlined in § 829, and that of intracisternal injection of iodised oil in § 757. These examinations should always be made by experts only.

(4) *Electro-encephalography* may, in expert hands, provide useful information in cases of head injury, suspected epilepsy or cerebral tumour (§ 667).

§ 708. Electrical Examination of Muscles. The APPARATUS REQUIRED is a faradic and a galvanic battery. In a normal case the faradic (interrupted) current causes a muscular contraction which persists as long as the current is passing. The galvanic

(constant) current causes a contraction only when the current is made or broken, not when it is passing.

To *test the faradic response*, place the large electrode on the patient's chest, on the back of the neck, or some other indifferent region, and another electrode over the motor point of the nerve or muscle to be tested. If the current is too strong for estimating the finer degrees of difference, the operator should take this second electrode in one or other of his hands, and apply his well-wetted finger to the well-wetted skin of the patient. A knowledge of the motor points of nerve and muscles is helpful, but they can be discovered by applying the electrode at different points, and noting those where contraction is most easily obtained. The motor point of a muscle is near the point of entry of its nerve; that of a nerve is generally near its most superficial part. The electrodes and the skin should be very *thoroughly* wetted with salt and warm water. The patient should be placed in a good light so that both sides of the body can be seen equally. Having thoroughly moistened the skin over the part to be tested, as well as the corresponding region of the other side, ascertain first (by gradually sliding up the secondary coil) the *minimum* current necessary to produce a minimum contraction of the muscle or muscles on the healthy side. Then test the side suspected of disease with the same amount of current, to see if the same degree of contraction is produced; if not, what strength of current is requisite. The faradic contraction of a muscle can only be obtained through its nerve, so that when the nerve is completely degenerated, the muscle (though still contracting to the direct stimulus of galvanism) fails to respond to faradism. Hence when a muscle does not respond to faradism, but does respond to galvanism, we conclude that the nerve is profoundly affected. (See § 709.)

To *test the galvanic reaction* the electrodes are placed in the same position as before. For (*a*) *quantitative* alterations, compare the two sides as before, noting what amount of current (as indicated by the number of cells used, or the number of milliampères registered by the galvanometer) is required to produce a minimal contraction on both sides. (*b*) For *qualitative* alterations begin with the kathode (negative pole) placed on or about the motor point under investigation. To distinguish the poles, place the two wires in a glass of water; a lively production of hydrogen gas appears at the kathode. Or place both wires on a piece of wetted blue litmus paper, which becomes reddened around the positive pole (anode) from the liberation of oxygen. Close the current by means of the interrupting handle; the contraction obtained is known as the Kathodal Closing Contraction (K.C.C.). Next convert the electrode on the patient into the anode or positive pole by means of your reverser, and repeat the process of closing the current. The resulting contraction is called the Anodal Closing Contraction (A.C.C.). Normally, with the same strength of current and the same degree of wetting of the skin and pressure of the electrode on the skin, K.C.C.>A.C.C., or what amounts to the same thing, a greater strength of current is required to produce A.C.C. than K.C.C.

Muscular contraction with a galvanic current is only produced at the closing or opening of the current. The normal order of the contractions is as follows:

K.C.C. > **A.C.C.** > A.O.C. > K.O.C. (O.C. = Opening Contraction).

Abnormally, A.C.C. is equal to or greater than K.C.C., and the character of the contraction is altered.

§ **709.** The **reaction of degeneration** (R.D.) differs in different stages. When a nerve is severed or is the seat of acute inflammation, after a preliminary increased response to both currents during the first two days, (*a*) the reaction is gradually lost to both currents during the ensuing ten days, the faradic reaction not being regained unless regeneration takes place.

(*b*) In the second or third week, and for some weeks afterwards, the galvanic reaction is restored, and Erb's REACTION OF DEGENERATION occurs in its complete form. It is characterised by—

(i.) No muscular contraction to faradism, however strong the current.

(ii.) A quantitative increased contraction to the galvanic current.
(iii.) The galvanic contraction, which in health is prompt and sharp, becomes sluggish, and often the response is better when the electrode is placed over the peripheral end of the muscle rather than over the motor point.
(iv.) No contraction is elicited by stimulating the motor nerve by either current.
(v.) Qualitative galvanic changes are usual: A.C.C. is equal to or greater than K.C.C.

(*c*) Two or three months later the galvanic contractility gradually disappears (unless regeneration is established), though it may happen that for one or two years A.C.C. can be obtained with a progressively increasing strength of current.

The electrical reactions of muscle are of use in diagnosis and prognosis.

(*a*) *In Diagnosis:* The presence of the reaction of degeneration in dicates that there is a lesion in the lower motor neuron. The reaction is partial or complete, according to the amount of damage to the nerve cells or fibres. When there is a *partial reaction of regeneration,* the test is difficult to carry out, and much skill and experience is required to interpret the findings. In certain diseases characteristic types of reaction occur. In *Tetany* the excitability of the nerve and muscle is increased both to faradism and to galvanism. Diminution of excitability to faradism and galvanism is found in conditions of muscular wasting, *e.g., Myopathy, Arthritic Muscular Atrophy, Disuse Atrophy,* and is usually proportionate to the wasting present. In *Myasthenia Gravis,* continued faradic stimulation rapidly tires out the affected muscles until, at last, no response can be obtained; after a period of rest recovery ensues. The galvanic reactions are unchanged. The Myasthenic Reaction is not present in all cases of the disease. During the paroxysms of *Family Periodic Paralysis,* the muscles respond neither to faradism nor to galvanism. In *Myotonia,* faradic stimulation results in a cramp-like contraction of the muscles which lasts some 5 to 30 seconds after the cessation of the current; galvanism produces a wave-like contraction which also persists after the current has been switched off.

(*b*) *In Prognosis:* In the case of *Facial Palsy* (Bell's Palsy) the electrical reactions are of prognostic value. If, in the third or fourth week after the onset, some faradic response is present, early recovery of function is indicated (*i.e.,* within three months). Where R.D. is present at the end of the fourth week after the onset, long delayed and only partial recovery is to be looked for, with the possibility of secondary contracture in the paralysed muscles.

In *Poliomyelitis* muscle-testing may be undertaken after the painful stage has subsided (usually three to four weeks after the first symptom). Affected muscles may be grouped into three classes: (*a*) Those which will recover; in these the electrical changes are but little altered. (*b*) Those which may recover; in these the electrical responses are typical, *i.e.,* the galvanic response is sluggish, though obtained with a weaker current than normally, while A.C.C.>K.C.C., and the faradic reaction is not quite lost. (*c*) Those which probably will not recover; in these there is no response either to faradism or galvanism.

PART C. DISEASES OF THE NERVOUS SYSTEM: THEIR DIAGNOSIS, PROGNOSIS AND TREATMENT

§ 710. Routine Procedure and Classification.

DIAGNOSIS.—(1) With a knowledge of the Applied Physiological Anatomy of the Nervous System and the Methods of Clinical Examination, it is possible to make an ANATOMICAL DIAGNOSIS of the localisation of the lesion.

(2) In order to make a PATHOLOGICAL DIAGNOSIS it is necessary to understand the natural history of diseases affecting nervous structures. These diseases may be classified according to the presenting clinical symptom.

A clinical classification has been used, as set forth in the following scheme:

If there is *Coma, Stupor or Lethargy*	Group I, § 711
If there are *Transient Losses of Consciousness, Fits or Convulsions*	Group II, § 719
If there is *Pyrexia with signs of Organic Nerve Disease*	Group III, § 724
If there is *Defect of Speech or Articulation* ..	Group IV, § 743
If the symptoms relate to the *Motor System* the presenting picture may be:—	
Spastic Paralysis	Group V, § 751
Parkinsonism	Group VI, § 765
Involuntary Movements	Group VII, § 770
Tonic Spasms or Cramps	Group VIII, § 777
Flaccid Paralysis or Muscular Wasting ..	Group IX, § 786
Ataxia or Inco-ordination	Group X, § 810
If the symptoms are those of a *Sensory or Painful Disorder*	Group XI, § 816
If there is *Progressive Headache and Vomiting* ..	Group XII, § 827
If the *Cranial Nerves* and *Special Senses* are affected	Group XIII, § 831

If the symptoms point to a *Psychoneurosis* or to *Unsoundness of Mind* (*Psychosis*) see Chapter XX.

GROUP I. COMA, STUPOR, OR LETHARGY.

The patient is attacked with gradually deepening and prolonged UNCONSCIOUSNESS *from which he* CANNOT BE ROUSED *by shaking or calling.* The case is one of COMA. STUPOR is a less severe degree of coma; the causes are similar. LETHARGY is a condition simulating ordinary sleep, but it may be prolonged for days or weeks.

§ 711. Coma.—In *Coma* the corneal reflexes are absent, the pupils insensitive to light, and the patient does not swallow fluids placed in his mouth. The diagnosis of such a case may present extreme difficulty, especially when no history of the patient's previous health or habits can be ascer-

tained. *If the coma is of sudden onset*, by far the commonest cause is *Cerebral Hæmorrhage*. The cause of such a hæmorrhage must be investigated.

Remember that ordinary examinations will not commonly reveal the cause of the coma. For this purpose examination of the fundi, measurement of the blood pressure, a catheter specimen of urine and spinal puncture, may all be necessary. Remember, too, that patients frequently injure themselves when they fall with a cerebral apoplexy, and the finding of a local scalp lesion, or even escaping blood or spinal fluid from the nose or ears, do not necessarily indicate a traumatic cause. If the patient's breath smells of alcohol, moreover, this does not of necessity mean that the coma is of alcoholic origin.

THE CAUSES OF COMA may be divided clinically into two groups:

A. UNILATERAL SYMPTOMS *are present.*—You may note (1) Conjugate deviation of the head and eyes. (2) Inequality of the pupils. (3) Asymmetry of the mouth and lower face, which puffs in and out with respiration, or other cranial nerve palsies, and (4) Absolute flaccidity of the limbs on one side when you test all four limbs in turn, lifting them and letting them flop on the bed. The cause is probably some Local Intracranial Lesion—viz.:

I. Cerebral Hæmorrhage, Embolism or Thrombosis.
II. Cerebral Tumour or Abscess.
III. Cerebral Compression from Traumatic or Spontaneous Extra-cerebral Hæmorrhage.

B. *The Symptoms are* BILATERAL *and* SYMMETRICAL.—The pupils are equal, the face symmetrical, and all four limbs equally flaccid. The cause is a Traumatic, or Toxic or Inflammatory Condition—viz.:

IV. Cerebral Concussion.
V. Post-Epileptic Coma.
VI. Uræmia.
VII. Diabetes Mellitus.
VIII. Insulin Hypoglycæmia.
IX. Poisoning by Opium, Alcohol, Barbiturates or Carbon Monoxide.
X. Acute Yellow Atrophy of the Liver (Cholæmia).
XI. Meningitis or Subarachnoid Hæmorrhage.
XII. Acute Encephalitis and Encephalitis lethargica.
XIII. Cerebral Malaria.
XIV. Heatstroke.
XV. Coma Carcinomatosum.
XVI. Coma Vigil of Typhus, Cholera, and Typhoid Fevers.
XVII. Anaphylactic Coma.
XVIII. Trance States and Katatonia.
XIX. Hypertensive Attacks.

CLINICAL INVESTIGATION OF COMA.—Obtain from a relative or friend any *History* available of preceding ill-health or fits, and if the coma came on suddenly or gradually. A sudden onset, *i.e.*, apoplexy, is indicative of a cerebral vascular lesion. Observe the *Age* of the patient. Coma in childhood is due to Meningitis, Epilepsy, or Intracranial Tumour or Abscess; about middle-age suspect a Cerebral Hæmorrhage.

Besides examining for unilateral signs, you should examine the scalp and skull for local trauma, noting if there is escape of spinal fluid and blood from the ears and nose (Fracture of the base). You smell the breath for acetone (Diabetes) and note the colour of the skin (jaundiced in Cholæmia, pink in Coal-gas poisoning), the presence of scars on the face and limbs (Epilepsy) or marks of hypodermic punctures (Insulin hypoglycæmia, Morphinism). You note the size of the pupils (pinpoint in Opium poisoning and Pontine Hæmorrhage) and the presence of head-retraction (Meningitis or Subarachnoid Hæmorrhage).

Next look at the optic discs (papillœdema in Intracranial Tumour and Abscess, albuminuric retinitis in Uræmia) and examine the ears for suppurative disease (Sinus Thrombosis). Estimate the blood pressure and withdraw a catheter specimen of urine, testing it for albumen and sugar. Finally, perform a spinal puncture.

Blood in the spinal fluid indicates hæmorrhage into the subarachnoid space from meningeal or ventricular hæmorrhage, or hæmorrhage from fractured base. In Meningitis, turbid spinal fluid will be present, or the fluid will show pleocytosis.

A. *There is* COMA, *and* UNILATERAL SYMPTOMS *are present.*

I. Cerebral Hæmorrhage, Embolism and Thrombosis.—The term apoplexy, or "stroke," is used to indicate a sudden loss of consciousness due to a vascular lesion—hæmorrhage, embolism, or thrombosis—within the skull. The *extent* and the *suddenness* of the vascular lesion, rather than its nature, determine the occurrence of coma. Cerebral hæmorrhage is most frequent between fifty and seventy, but it may occur even in children.

§ **712.** *Symptoms of Cerebral Hæmorrhage.*—The attack may be ushered in by a stage of headache or giddiness, lasting a few days, connected doubtless with the associated high blood pressure ; or it may come on suddenly without warning. Vomiting or a convulsion sometimes occurs. Sometimes the paralysis comes on with faintness and vertigo only ; or it may develop more gradually, followed later by unconsciousness (ingravescent apoplexy). Sometimes it comes on during sleep. In severe cases the patient is deeply comatose, cyanosed, and the breathing is stertorous, the skin is cold and covered with sweat. The muscles are completely flaccid, the flaccidity being greater on the paralysed side. The paralysed angle of the mouth drops, and the cheek flaps in and out with respiration. The patient is incontinent, and in hiscoma blisters may develop on the heels, buttocks and sacrum. The pupils and tendon reflexes are commonly absent or a larger pupil may be present on the side of the cerebral lesion. The pulse is slow and the temperature subnormal.

Diagnosis of Cerebral Hæmorrhage.—The *sudden* onset of profound coma in a person of middle age, with the presence of unilateral signs, are points of great diagnostic significance. It should be remembered that cerebral hæmorrhage frequently supervenes in the course of chronic interstitial nephritis, and therefore uræmia and apoplexy may be concurrent. The diagnostic features of the greatest value are the state of the pupils, particularly their inequality, the loss of the conjunctival reflex, and the augmented blood pressure. The diagnosis of the various *forms of vascular lesion* is given in Table XLVI. One is fairly safe in excluding cerebral hæmorrhage if the blood pressure is not high, provided that the patient is not suffering from a hæmorrhage into a vascular and malignant growth, or some severe anæmia, such as acute leukæmia. Though high blood pressure is suggestive of cerebral hæmorrhage, remember that the arterial degeneration which usually accompanies it may give rise to thrombosis as well as hæmorrhage.

As regards the *locality* of the hæmorrhage, the usual position (about

70 per cent.) is the *external* or *internal capsule*. The hæmorrhage comes from the lenticulo-striate artery, especially the left side, giving rise to hemiplegia on the side opposite to the lesion. In most of the cases of hæmorrhage into the *ventricles* there is deep coma and head retraction with paralysis or rigidity of all four limbs, and blood in the spinal fluid: the condition is fatal. Marked contraction of both pupils, convulsions and vomiting, with paralysis of all four limbs, and rapid rise of temperature to the level of hyperpyrexia, suggest hæmorrhage into the *pons*. Hurried or Cheyne-Stokes' respiration often accompanies hæmorrhage in this site, and death ensues. *Conjugate deviation of the head and eyes* towards the paralysed side is frequent when the hæmorrhage involves the motor tract.

Prognosis.—It is held by some authorities that cerebral hæmorrhage almost always goes on to a fatal termination, and that the cases of supposed cerebral hæmorrhage which recover are really cases of thrombosis. About half the cases of supposed hæmorrhage recover from the attack, some with residual paralysis; the other half die within forty-eight hours. The depth and duration of the coma are fair measures of the extent of the mischief, and therefore of the prognosis. As regards *locality*, ventricular hæmorrhage and hæmorrhage into the pons are the most serious.

Etiology.—Cerebral hæmorrhage is more frequent in the male than the female sex, and does not usually occur until after fifty. It is common in those addicted to alcohol. The rarer cases of "apoplectic seizure" in persons under forty are almost invariably due to embolism, thrombosis, or rupture of a "congenital" aneurysm. Heredity plays an important part by reason of the tendency to vascular disease which runs in families. Disease of the vessels is a necessary precursor to their rupture. High blood pressure is a most important factor in the causation of apoplexy; it predisposes to arterial disease, and may also determine the hæmorrhage. The causes of high blood pressure are given in § 87; the commonest cause is chronic interstitial nephritis. Hæmorrhage into the brain may occur in a variety of other conditions. It may occur into a rapidly-growing *glioma* and is rapidly fatal. It may be the terminal incident in *acute leukæmia, purpura*, and other blood states. It occurs in *acute infections, e.g., diphtheria* and *septicæmia, e.g.*, puerperal sepsis, and in these last conditions, embolism (mural endocarditis) and thrombosis also occur.

§ 713. **Cerebral Embolism.**—Cerebral embolism involving a fairly large artery may give rise to all the symptoms of apoplexy. Embolism is characterised by *instant* loss of consciousness. When a small cortical vessel is blocked, giddiness, or a Jacksonian attack followed by a monoplegia, may replace coma. Sometimes the posterior cerebral artery is involved, with hemianopia and sensory change, rarely the anterior cerebral or the internal carotid (*carotid hemiplegia*—§ 684). The artery usually affected is the middle cerebral. The age of the patient and the presence of cardiac disease, especially auricular fibrillation, mitral disease and ulcerative endocarditis, aid us in diagnosis. Embolism occurs from the pulmonary veins in the puerperium. During induction of artificial

pneumo-thorax a transient hemiplegia may occur from accidental introduction of air into the pleural or pulmonary veins (" Pleural hemiplegia ").

Fat Embolism follows a few hours after fracture of a long bone (Table XLVI). The fat globules lodge first in the lungs, producing cyanosis and pulmonary œdema. Some of the globules may make their way through the pulmonary capillaries to the cerebral vessels, with delirium, coma and localised cerebral palsies.

§ 714. Thrombosis of the Cerebral Arteries.—Thrombosis generally arises from a gradual occlusion of the lumen of a vessel by senile arterial disease, or by syphilitic endarteritis, in younger subjects. The picture is that of a person going about in usual health and awakening early in the morning conscious, but with a hemiplegia. In other cases, a hemiplegia develops over a period of one or more hours with little or no loss of consciousness. There is sometimes a history of previous " faints " or " attacks " indicating thrombotic lesions in tiny cerebral arteries. Thus transient hemianopia, monoplegia, hemiparesis, dysarthria, aphasia or mental confusion may occur, and these symptoms may be repeated many times, either singly or in combination, with partial recovery in the intervals between the attacks. Where the vessel occluded is large and the ensuing necrosis extensive, coma will be profound. Cerebral Thrombosis is much commoner than Cerebral Hæmorrhage or Embolism.

The *Prognosis* of cerebral embolism as regards life is usually good, though the paralysis tends to remain. Paralysis is most likely to clear up in children. If the causal condition remains, a second attack may occur. Cerebral embolism in malignant endocarditis is ultimately fatal. In thrombosis survival is likely, often for many years ; but the life of a patient must be regarded as precarious and the field of effort limited. Women seem to survive strokes better than men. Death may occur from coronary thrombosis or from cerebral hæmorrhage into an area previously softened by thrombosis. In syphilitic cases recurrence is unlikely if anti-specific treatment is thoroughly pursued.

The *Treatment of an Apoplectic Seizure.*—Perfect rest and quiet are very important. The patient should, as a rule, be left in the room where the seizure occurred—a mattress being placed on the floor, if necessary —rather than incur the movement necessary to raise him on to a bed. The head and shoulders should be raised, and the patient turned gently over to one side to prevent the tongue falling back into the pharynx. The administration of food is, as a rule, undesirable, at least by the mouth, for fear of its passing into the air passages ; alcohol must be absolutely forbidden. The patient will benefit by starvation from food for a day or two ; the lips may be moistened. The bladder should be watched, and the catheter carefully passed if necessary. In cases due to *embolism* the condition responsible for the embolism must be treated. Complete rest in bed for several weeks is essential to diminish the risk of further embolism. If embolism occurs in a patient receiving treatment for auricular fibrillation by quinidine, this drug must be discontinued. In *thrombosis* stimulants, *e.g.*, tinct. nucis vom. ♏ 10–15, t.d.s., or niketh-

amide (coramine) hypodermically—are indicated. Prolonged rest in bed is inadvisable. In *hæmorrhage*—indeed whenever the blood pressure is high —a brisk purge is indicated; two drops of croton oil or 4 to 8 grains of calomel on the tongue may be given, followed, if necessary, by a soft soap or turpentine enema. The chief indication is to prevent any extension of the hæmorrhage. If the blood pressure is very high, it is a good practice to bleed to the extent of 10 to 20 ounces. An ice-bag or a cooling lotion to the head may relieve headache.

In all cases, even when coma is present, the paralysed arm and leg should be frequently moved at all joints to prevent contracture. A pillow should be placed in the axilla, or a sling tied to the top of the bed to abduct the arm. The forearm should be frequently supinated and a light cock-up splint applied to the hand and forearm during the morning and afternoon. Passive movements should be carried out at all joints thrice daily with gentle massage, and as soon as voluntary power returns active re-educative exercises can be commenced. If left to himself the patient will walk with his leg extended, circumducting the limb to prevent his toes catching the ground. He should be taught to advance his leg by flexion at hip and knee. In sitting he should avoid the tendency to adduction of the leg and inversion of the foot.

TABLE XLVI.

DIAGNOSIS OF CEREBRAL HÆMORRHAGE, THROMBOSIS, AND EMBOLISM.

	Cerebral Hæmorrhage.	*Cerebral Thrombosis.*	*Cerebral Embolism.*
Age.	Middle and Advanced Life.	Middle and Advanced Life or any age.	Any age, but frequent in early life.
Causes.	1. Arterio-sclerosis with high blood pressure. 2. Blood diseases. 3. Acute Infections and Septicæmia.	1. Cerebral atheroma. 2. Syphilitic endarteritis. 3. Acute infections. 4. Exhausting conditions, phthisis, anæmia. 5. Cardiac enfeeblement.	1. Cardiac lesions, especially mitral stenosis, auricular fibrillation and malignant endocarditis. 2. "Fat embolism" in fracture of long bones.
Onset.	Coma usually sudden, with convulsions.	Onset usually gradual, with premonitory vertigo. Sometimes convulsions, rarely coma.	Instantaneous loss of consciousness.
Time of Onset.	During emotional excitement or physical exertion.	Often during sleep.	During exertion.

§ 715. **Thrombosis of the Cerebral Sinuses** may give rise to coma and all the symptoms of apoplexy. It may arise from caries of the skull (syphilitic or tuberculous), extension from an intracranial abscess, *e.g.*, in suppurative ear disease, and occasionally from the pressure of an aneurysm, gumma, or other tumour; or in association with meningitis. Sinus thrombosis is favoured by the feeble cerebral circulation characterising cachectic conditions (chronic diarrhœa, typhoid fever, and marasmus in children). *Septic thrombosis* and the differential diagnosis of thrombosis of the lateral, cavernous and longitudinal sinuses are described in § 738.

II. **Cerebral Tumour or Abscess.**—The symptoms may be unilateral or, less commonly, bilateral. Sudden onset of coma in cerebral abscess indicates that rupture has

occurred into the ventricle, the spinal fluid in such cases being purulent. There will be a preceding history of otitis media, suppurative frontal sinusitis or intrathoracic sepsis, and papilloedema or cranial nerve palsies may be present. *Chronic subdural hæmatoma* following a head injury, or in the aged and arterio-sclerotic, may cause similar symptoms. The onset of coma in these cases generally means a fatal issue (§ 827).

III. **Cerebral Compression from Traumatic Extra-cerebral Hæmorrhage.**—In Delayed Traumatic Apoplexy the patient recovers from his initial concussion and collapse. With the rise in blood pressure within a few hours, stupor or coma again intervene, with Jacksonian convulsions and rapidly progressive monoplegia or hemiplegia. The cause is a *rupture of the middle meningeal artery* with extra-dural hæmatoma or a subdural hæmatoma, from rupture of cortical veins. The treatment is exclusively surgical.

B. *There is* Coma, *and the Symptoms are* Bilateral *and* Symmetrical.

§ **716.** IV. **Cerebral Concussion.**—The unconsciousness follows immediately after the head injury without any latent interval. The patient is pale and collapsed, with weak pulse, shallow respiration, pale face, dilated pupils, sweating, flaccidity of the limbs and low blood pressure. In other cases he is merely dazed. In severe concussion the stupor lasts hours or days and is followed by a reactive stage ushered in by vomiting or convulsions. The temperature rises to 100° F. or higher, the pulse becomes full and bounding and the respirations deeper. There is intense headache and photophobia with hypersensitiveness to noise, and the patient lies curled up in bed with his limbs flexed, resentful of interference. This is termed "cerebral irritation," and these symptoms may last for days or weeks, perhaps accompanied by visual hallucinations. Steadily rising temperature to 104° or 105° is a sign of grave omen, indicating extensive contusion. The occurrence of *intracranial hæmorrhage* may be indicated by (1) progressive deepening of unconsciousness, (2) progressive loss of tone or power in the limbs on one side, (3) progressive slowing of the pulse, (4) progressive discrepancy in the size of the pupils—the hæmorrhage being usually on the side of the slowly dilating pupil.

Post-traumatic amnesia is that interval of forgetfulness which may elapse between the moment of impact and the time of subsequent recovery of continuous awareness of surroundings. "Islands" of memory may exist within the span of post-traumatic amnesia. As the patient recovers, the duration of post-traumatic amnesia tends to shrink. During this period rational behaviour may be carried out and subsequently forgotten.

Retrograde amnesia may also be present for the events immediately preceding the injury. Long retrograde amnesias of months or years, or total amnesia for all events prior to the injury occur usually in hysterical personalities.

Prognosis.—The duration of post-traumatic amnesia is usually a fairly reliable guide to the severity of the brain injury and may be used to estimate prognosis except in cases which are complicated by compensation hysteria. If the post-traumatic amnesia lasts minutes, the brain injury is minor. When it lasts 1 to 3 hours, the brain injury is moderate and complete recovery may be expected in from six to eight weeks. When the post-

traumatic amnesia (P.T.A.) lasts a week or more, the brain injury is severe and intellectual insufficiency or personality change may result as a temporary or permanent finding. Complete recovery in such cases may take three to six months or longer. Other factors to be taken into account in estimating the prognosis are—(1) the pre-traumatic personality of the patient, (2) associated damage to cranial nerves and other structures, and (3) the type and circumstances of the injury.

Treatment.—As soon as possible the patient should be laid flat in bed with his head to one side, and heat applied to counteract shock (§ 239). If there is a bleeding scalp-wound, hæmorrhage should be arrested by a pad and tight bandage, or by deeply suturing the scalp. Should there be a depressed fracture of the skull operation may be necessary (*e.g.*, if there is an overlying scalp wound, which might admit infection to the underlying structures). Search should be made for gross injuries of the limbs and spine, and as soon as the patient has recovered from initial shock, X-ray photographs of the skull should be obtained whenever possible. Noise and light should be excluded from the sick room. Watch the bladder for retention. Fluids should be given by mouth, with a feeding cup if the patient can swallow. The nurse should be instructed to watch for the danger signs of meningeal hæmorrhage enumerated above. Restlessness is treated by rectal paraldehyde, ♏ 120–240 given in an equal amount of olive oil, or by chloral hydrate gr. 15, potassium bromide gr. 20, three or four-hourly by mouth. Morphine and alcohol should on no account be given. Thirty-six hours after the head injury lumbar puncture should be performed and the fluid pressure estimated by a manometer. If the pressure of the spinal fluid is *above* 150 mm., fluid should be withdrawn until that pressure is reached; this may have to be repeated. Saline purgatives and 6–10 oz. of a saturated solution of magnesium sulphate run into the rectum with a tube and funnel will help to dehydrate the œdematous brain. If the pressure is *below* 150 mm. no spinal fluid should be withdrawn. The foot of the bed should be raised on 6 inch blocks.

Intravenous dextrose solution (50–100 c.cm. of a 50 per cent. solution) may be administered later to cases with raised intrathecal pressure. Given too early, it may mask symptoms of meningeal hæmorrhage. It causes a sustained fall in intracranial pressure and helps to control acidosis and shock. It should be injected very slowly, at a rate not exceeding 3 c.cm. a minute. Where return to consciousness is delayed for days or weeks (traumatic stupor), the question of decompression may have to be considered. Most cases with cerebral contusion (§ 696), even when aphasic, clear up without operation. Convalescence should be graduated with slow, progressive increase of the field of mental and physical effort. Many cases will be able to leave bed at the end of three weeks and return to work in four to six weeks' time. Others continue to suffer from liability to physical and mental fatigue, difficulty in concentration, headaches, giddiness and sleeplessness and other symptoms of unresolved cerebral

contusion. If possible the patient should be tested out in the performance of his work at home before he returns to business.

V. **Post-Epileptic Coma.**—If there is no history we may have to rely on the finding of old scars for evidence of previous attacks. The coma is usually of short duration, and the patient becomes more and more conscious. After a "congestive attack" in General Paralysis of the Insane the patient may become comatose.

VI. **Uræmia** (see § 372).—There may be albuminuric retinitis or œdema about the face and legs. A catheter specimen will show albumen, tubular or blood casts, and, in acute nephritis, blood. Uræmia produces stupor rather than coma, the deep reflexes can be obtained, there is usually no incontinence, and the plantars are flexor. Uræmic twitchings or convulsions may be observed. Deep coma with unilateral signs may occur in uræmic patients, due to cerebral hæmorrhage, to which they are especially liable.

VII. **Diabetes Mellitus** (see § 416).—The examination of the urine makes the diagnosis simple. It is usually gradual in onset.

VIII. **Insulin Hypoglycæmia** follows an overdose of insulin which leaves the blood-sugar far below its normal value. A **small overdose** gives rise to prodromal symptoms (§ 418); after a large overdose unconsciousness supervenes in half to one hour, often accompanied by fits. The diagnosis is settled by a blood-sugar examination: insulin coma only occurs if the value is below 0·045 to 0·06 per cent. Consciousness is restored by injecting adrenalin hydrochloride 1 c.c. subcutaneously, or by intravenous dextrose.

IX. **Poisoning by Opium, Alcohol, Barbiturates, Aspirin, or Carbon Monoxide.**—In all forms of narcotic poisoning the deep reflexes are lost and the plantars are extensor. In *Opium poisoning* the pupils are contracted to pin-points. In morphinists, the scars of hypodermic piqures may be seen in the arms and thighs. In *Alcoholism* the smell of the breath is fallacious. If house-surgeons would bear this in mind they would not send dying cases of apoplexy or fractured skull away from hospital as "drunks." If unilateral signs are present the case is not one of alcoholism. The diagnosis of Alcoholism may be sustained by finding alcohol in the urine, or spinal fluid. Mix a specimen with potassium bichromate solution and allow strong sulphuric acid to flow to the bottom of the test-tube. The solution turns a bright emerald green if alcohol be present in quantity. In *Barbiturate* (Medinal, Veronal, etc.) poisoning the picture resembles opium poisoning, except that the respiration rate is often raised: skin rashes are rare. There is great liability to hypostatic pulmonary œdema. The drug may be present in the spinal fluid. *Aspirin* poisoning is characterised by very profuse sweating, hyperpnœa due to acidosis, a reducing substance in the urine and a positive ferric chloride test. It is very liable to be confused with hypoglycæmic coma (§ 418). *Carbon Monoxide* poisoning occurs from coal-gas or petrol fumes, accidentally or otherwise. There is sudden collapse and unconsciousness with stertor and a cherry-red complexion. The blood-spectrum is that of carboxy-hæmoglobin.

X. **Acute Yellow Atrophy** (Cholæmia, § 333).—The associated jaundice makes the diagnosis.

XI. **Meningitis or Subarachnoid Hæmorrhage.**—The presence of head retraction and Kernig's sign point to meningeal irritation. A sudden onset of headache, and deepening coma, indicate a *Subarachnoid Hæmorrhage* from a leaking cerebral aneurysm, usually situated on the Circle of Willis.

§ 717. In **Subarachnoid Hæmorrhage** the presenting symptoms are those of *sudden* intense headache with rigidity of the neck, together with vomiting, head retraction, Kernig's sign, and bilateral extensor plantar responses.

In severe cases there may be focal or general epileptiform convulsions, with rapid coma and death. In some, the spurting arterial jet excavates the inferior surface

of the hemisphere, producing severe hemiplegia or hemianopia. The *diagnosis* closely resembles that of an acute meningitis, but (1) the onset is startlingly sudden, indicating a vascular cause; (2) signs of tuberculosis, suppurative otitis, etc., such as commonly cause meningitis, are absent; (3) retinal hæmorrhage or subhyaloid hæmorrhage and papillœdema may be seen in the fundi; (4) the diagnosis is promptly cleared up by lumbar puncture, which reveals abundant blood intimately mixed with the spinal fluid. When the spinal fluid settles, the corpuscles sink, leaving the supernatant fluid pink with the "laked" blood. During recovery the spinal fluid may be yellowish, and shows a lymphocytosis. The bleeding in many cases comes from a small *non-syphilitic aneurysm* ("congenital" aneurysm) on the Circle of Willis or a cerebral artery near this. Massive albuminuria may be present at the onset for a day or two, suggesting the diagnosis of chronic nephritis. Root-pains in the arms and trunk may be observed as the blood extends downwards in the spinal theca. Before it ruptures, the aneurysm may cause migraine or recurrent unilateral headaches. In others, it may compress and paralyse one of the oculo-motor nerves, commonly the third. Slow leakages of blood from the aneurysm cause recurrent bouts of severe supraorbital pain with gradually increasing ptosis and ophthalmoplegia. Subarachnoid hæmorrhage is a fairly common disease. It occurs at any age and especially in the third and fourth decades.

Subarachnoid hæmorrhage may also occur in (1) Cerebral Arterio-sclerosis, (2) Renal Disease with high blood pressure, (3) Intra-cerebral Hæmorrhage which leaks into the ventricles, (4) Vascular Cerebral Tumour in the neighbourhood of the ventricular system or subarachnoid space, (5) Acute Encephalitis Lethargica, (6) Hæmorrhagic blood diseases, such as Acute Leukæmia, (7) Septic embolic cerebral aneurysm in Infective Endocarditis.

The milder cases recover, but fresh leakages are liable to occur months or years later. More than a third of the cases, however, are immediately fatal. Lumbar puncture should be performed for diagnosis only; as a therapeutic measure it does more harm than good and may precipitate further bleeding. The patient should be nursed in the horizontal position; retention of urine should be looked for. Intramuscular injections of thromboplastin or normal horse serum to increase the coagulability of the blood, are indicated, with ice-bags to the head, carefully suspended, and elimination of light and noise from the sick-room. Pain is relieved by morphine, once the diagnosis is made, or by pyramidon gr. 10 and aspirin gr. 10. The patient should be kept in the horizontal position until all headache and neck stiffness have gone. There should be no return to work for three months. Recurrences are most unlikely if the patient avoids strenuous effort. In severe cases the initial illness may be followed by a transient confusional psychosis. It has been possible to demonstrate the aneurysm by arteriography, and then ligature has been successfully performed.

XII. **Acute Encephalitis** is an occasional cause of Coma (see § 740). The encephalitis may be primary, as in Lethargic Encephalitis or Polioencephalitis, or it may follow specific fevers such as measles, diphtheria, scarlet fever, or vaccination (Vaccinal Encephalitis). It may occur in Cerebral Syphilis.

XIII. **Cerebral Malaria.**—Coma, with hyperpyrexia, occurs from malignant tertian malaria (§ 510) in malarial districts. The plasmodium will be found in the blood.

XIV. **Heat-stroke** (Sunstroke) (§ 508) causes coma, with hyperpyrexia—108° F. —or above this. The spinal fluid is sterile and shows a polymorphonuclear pleocytosis in the acute stages, later lymphocytes appear. Lumbar puncture produces immediate relief of symptoms.

XV. **Coma Carcinomatosum** occurs in patients dying of secondary malignant deposits. The urine shows acetone and diacetic acid. A similar coma occurs as a terminal event in Addison's disease and other exhausting illnesses.

XVI. **Coma Vigil** is a condition occurring in cholera, typhus and severe typhoid fever, where the vital processes are at so low an ebb that the patient may be mistaken for dead.

XVII. **Anaphylactic Coma.**—Following upon the injection of a foreign protein, the patient may become comatose, as the result of severe anaphylactic shock.

XVIII. **Trance States** (§§ 697, 888) should never be mistaken for true coma. The breathing is never stertorous, the pupils react to light and the patient forcibly resists attempts to open the eyes. In **Katatonia** there is mutism, refusal of food and general diminution of activities. Such patients allow their bodies and limbs to be placed in awkward positions which are maintained indefinitely. There is apathy but no unconsciousness. The condition occurs in Schizophrenia as well as in traumatic and other stupors (§ 897).

XIX. The HYPERTENSIVE ATTACK; see § 94.

The *Prognosis* of coma is always grave, the gravity increasing with its depth and duration. The coma after head injury usually comes under the care of the surgeon. The coma of apoplexy and other vascular lesions has been already dealt with. In post-epileptic coma, if the patient does not recover within a few hours then status epilepticus is present and the condition is serious. Coma occurring with tumour of the brain or acute lesions is usually fatal. The prognosis of opium poisoning depends upon the amount of the drug administered and the promptitude and efficiency of the treatment. Uræmic coma is not so unfavourable as might be thought; cases recover with proper treatment, but in granular kidney the condition recurs sooner or later. In diabetic coma, which was formerly fatal, the patient now usually recovers with insulin and glucose.

Treatment.—For *alcoholism* and all *drug poisoning* the stomach and colon should be repeatedly washed out with warm water and warmth and cardiac stimulants applied. In addition, in *barbiturate* poisoning the subarachnoid space should be drained by lumbar puncture, and the patient propped up in bed to prevent pulmonary congestion and œdema. Nikethamide, picrotoxin, or strychnine is given if the heart or respiration be failing. The patient must be kept awake by walking him about, applying electricity to the limbs, ammonia to the nostrils, and artificial respiration. For *uræmia* eliminate the poison in the blood by colon lavage, hot packs, venesection and saline injections. *Diabetic coma* calls for skilled and prompt treatment by insulin. For *insulin coma* give dextrose intravenously and inject adrenalin and pituitrin (§ 418). For *carbon monoxide* poisoning carry out artificial respiration, and give to breathe a mixture of 7 per cent. carbon dioxide and 93 per cent. oxygen. Special inhalation apparatus is provided for this purpose. This may also be used for opium poisoning. In all poisoning cases artificial respiration may be carried out by means of a Drinker or Paul-Bragg apparatus.

§ 718. Coma in Children, apart from injury, may be due, in order of frequency, to tuberculous meningitis, post-basic meningitis, suppurative meningitis, post-epileptic stupor, cerebral tumour, syphilitic pachymeningitis, sinus thrombosis and hæmorrhage; diabetes, abscess and intracranial cysts, are rare causes. The history, mode of onset and associated symptoms, aid the diagnosis. Tuberculous meningitis is by far the most frequent cause (§ 727). Cerebral hæmorrhage or embolism occur chiefly in association with the specific fevers, such as small-pox and whooping-cough, also with rickets and scurvy. In marasmic conditions, thrombosis of the longitudinal sinus (§ 715) may ensue, together with meningeal hæmorrhage, giving

rise to convulsions followed by coma. Thrombosis of the veins of Galen and lateral sinus thrombosis, in association with ear disease, may cause coma (§ 738).

The patient remains in a condition simulating normal sleep for days or weeks—the condition is LETHARGY.

The *causes* of lethargy are similar to the causes of coma. Lethargy is a prominent symptom in *Encephalitis Lethargica* (see § 698) and in *Tumours of the Hypothalamic region.* In both tumours and encephalitis fever and ocular palsies may be present, but gross papillœdema is more common in neoplasm. Mild continued lethargy accompanied by mental confusion is suggestive of *narcotic poisoning.*

GROUP II. TRANSIENT LOSS OF CONSCIOUSNESS. FITS OR CONVULSIONS.

§ **718a.** *The patient has a sudden transient* ATTACK *of* LOSS OF CONSCIOUSNESS. The condition may be a simple "faint," SYNCOPE, or, if it follows a head injury, CONCUSSION. EPILEPSY must always be considered, either *idiopathic*, or *symptomatic* of local or general disease, *e.g.*, cerebral arteriosclerosis, cerebral syphilis, cerebral tumour, uræmia, etc.

(*a*) SYNCOPE. (Fainting.) Apart from those who faint from cardiovascular instability or insufficiency, due to disease or debility, a small proportion of "nervous" individuals are liable to faint in certain situations. Such faints are conditioned by prolonged standing in crowds and stuffy atmospheres, during medical examinations, listening to medical lectures, on feeling sudden pain, at the sight of blood, or during sudden panic. Others faint after extreme physical over-exertion (see also § 35).

The phenomena observed during some of these syncopal attacks may be indistinguishable from those seen in epilepsy; convulsions and even incontinence may occur. In such rare cases, a constitutional nervous instability is present and epilepsy may subsequently develop. In true "reflex epilepsy" the conditioning stimuli are stereotyped and often bizarre, *e.g.*, certain sounds or sudden noise will precipitate an attack: moreover, in these cases other fits, not so conditioned, will usually occur.

VERTIGO bears a superficial resemblance to fainting. For *Ménière's Disease*, see § 692. In severe aural vertigo consciousness may be lost when the paroxysm is at its height.

(*b*) As the result of HEAD INJURY the patient may be momentarily *dazed* or may *lose consciousness* (see § 716).

(*c*) § **719. Minor Epilepsy** (Synonym: Petit mal).—The attack is preceded—in about half the cases—by an aura or warning, unattended by convulsions, and often without falling, the whole lasting rarely more than half a minute to a minute. In the type of attack now under consideration, it may be that the patient merely pauses in a conversation and does not reply to questions, or there is only a vacant look, a fixity of gaze, dilated pupils or momentary pallor of the face, which none but

a close observer would notice. In more severe cases the patient drops what he is holding, or flings it from him; his head may sag for a moment and urine may be passed involuntarily, without any convulsion.

(*d*) SENILE SYNCOPE.—In the aged, transient losses of consciousness may occur from minute cerebral thromboses or myocardial failure with arterial degeneration. They indicate a need for increased rest, protection from cold and exhaustion, and small quantities of alcohol with meals and last thing at night.

Convulsions (Fits, Epilepsy) may be due to (A) IDIOPATHIC EPILEPSY, or may be (B) SYMPTOMATIC, or (C) HYSTERICAL.

A. IDIOPATHIC EPILEPSY

I. Major Epilepsy.
II. Minor Epilepsy (Petit Mal).
III. Special Varieties of Epilepsy.

B. SYMPTOMATIC EPILEPSY

IV. Cerebral Tumour.
V. Cerebral Syphilis.
VI. Cerebral Arterio-sclerosis.
VII. Hypertensive Attack.
VIII. Traumatic Epilepsy.
IX. Congenital Nervous Disease.
X. Stokes-Adams' Syndrome.
XI. Cerebral Hæmorrhage or Embolism.
XII. Asphyxia.
XIII. Forced Deep Breathing.
XIV. Toxic Convulsions.
XV. Acute Encephalitis.
XVI. Malaria.
XVII. Intracranial Cysticercus.
XVIII. Presenile Dementia.

FALLACIES.—The convulsions of STRYCHNINE POISONING or TETANUS should never be mistaken for epilepsy. In these conditions *consciousness is never lost.*

§ 720. Investigation of Cases of "Fits."—Confronted with a case giving a history of a " fit " you must decide if the condition is *organic* or *psychogenic*. Psychogenic attacks are of two main varieties: (1) Hysterical Attacks, (2) Vaso-Vagal Attacks (Gowers).

The latter type of attack occurs in BORDERLAND EPILEPTIC patients. There is, however, never true loss of consciousness, as in organic epilepsy, and the onset of the attack is slow. The patient experiences a sense of unreality, of being unable to move or to utter a single sound, with an impending feeling of imminent catastrophe or death. Associated with this are palpitation, pain under the left breast, abdominal discomfort or other visceral sensation, and rapid lowering of blood pressure, nausea or flatulence. The attacks last many minutes or hours. There is often a family history of epilepsy or migraine. The *prognosis* is good. Any psychological factors should be sought and corrected, and luminal prescribed, as for epilepsy.

The table on page 923 gives useful aid in distinguishing between hysterical attacks and organic epilepsy.

Hysterical attacks may follow true Epileptic attacks—*Hystero-epilepsy.* Epilepsy may be feigned in *Malingering.* Such attacks are carefully planned, the attack lasts indefinitely, and resembles true epilepsy only in the imitation of the convulsive movements.

When in doubt, it is best to regard anomalous attacks as epileptic until the subsequent history makes the diagnosis definite.

To obtain the necessary diagnostic information, if you have not observed the attack yourself, *it is essential to interrogate an eye-witness of the attack in addition to the patient.* In talking to the patient you should be careful not to speak of " fits " but of " attacks " or " seizures."

1. *Interrogation of the Eyewitness.*—The eyewitness should be asked to describe the attack in his own words. Questions are then put—Is there any sound before

TABLE XLVII.

	Organic Epilepsy.	*Hysteria.*
1. Periodicity and onset.	Attacks occur mostly at definite hours of the day or night.	Attacks follow upon an emotional crisis. Never during sleep.
2. Incontinence.	Incontinence of urine during attack, albuminuria found later.	Never incontinence.
3. Self-injury in attack.	Tongue, cheek, or lip-biting, with blood in the mouth.	The patient is not hurt in the seizure, although onlookers may be.
4. Movements.	Involuntary movements are clonic, if present, or have a definite march and are accompanied by conjugate deviation of the head and eyes.	The movements are purposive, and spectacular, the eyes screwed up and the hands clenched. Never conjugate deviation of the head or eyes; often convergent spasm of eyes.
5. Breathing.	Breathing stertorous, with cyanosis	Breathing never stertorous.
6. Duration.	Attack is of short duration usually.	Attack often prolonged, especially if before an interested audience.
7. Plantar reflexes.	Extensor during and immediately after the fit.	Usually absent at the toes.

he falls? What is his breathing like in the attacks, is it snoring? What is the colour of his face in the attacks? Does the patient reply to questions in his attacks? How long do the attacks last? These questions can be answered only by an eye-witness.

2. *Interrogation of the Patient.*—This should include direct questions as—Do you fall in the attacks? Do you get any warning? Has there ever been any blood in your mouth after the attack? Have you ever wet yourself in an attack by passing water? Have you ever hurt yourself in an attack? (An examination of the face, scalp, tongue or limbs may reveal cicatrices, evidences of injury in the fits.) At what time of day or night do your attacks mostly occur?

When observing a fit, push the edge of a handkerchief into the patient's mouth stand back and watch the distribution and spread of the convulsion. If the patient is in bed, throw back the bedclothes. After the convulsion is over, examine the limbs for flaccidity, examine the pupils and test the tendon reflexes and plantar responses.

§ **721. Epilepsy** may be: A, IDIOPATHIC or B, SYMPTOMATIC.

A. IDIOPATHIC EPILEPSY is a chronic malady characterised by partial or complete loss of consciousness, which is usually the essential feature of the condition. In most cases convulsive movements occur, varying in distribution and degree. Minor Epilepsy (*petit mal*) consists mainly of transitory disturbance of consciousness, Major Epilepsy (*grand mal*) is loss of consciousness with generalised convulsions.

I. **Major Epilepsy.** *Symptoms.*—A complete epileptic fit has the following characters, though they are rarely all present in their entirety: (1) In some cases, during the previous twelve to twenty-four hours, there may be *prodromata*, consisting of headache, giddiness, malaise, or alteration of character or mood. In more than half the cases this stage is absent. (2) The fit, in many cases, is immediately preceded by

an *aura* or warning—*i.e.*, a sensation or movement lasting, at most, only a few seconds, valuable as indicating the point of the cortex whence the cortical nerve-storm starts. The auræ differ infinitely in detail. There are four main groups. *Sensory auræ* are most common—*e.g.*, "a wave passing over the body," numbness, a sensation of movement, flashes of light or of colour, or singing in the ears; *motor auræ*—*e.g.*, twitching of a muscle or limb, occasionally of the trunk, and in rare cases there is a "procursive aura," in which the patient runs forward or turns round and round; *psychical auræ*—*e.g.*, various strange thoughts or hallucinations; and *somatic auræ*—*e.g.*, gastric discomfort, nausea, or fluttering in the stomach. (3) *Loss of consciousness* is the pathognomonic feature of idiopathic epilepsy. It succeeds the aura so quickly that the patient may not have time to place himself out of danger before loss of consciousness is complete. (4) *Convulsions* supervene almost at the same time as the unconsciousness. They are often ushered in with a scream. The true epileptic cry is a "weird, unearthly, hollow sound, produced by inspiratory spasm drawing in air over the nearly closed vocal cords." It comes with the onset of loss of consciousness and the onset of tonic rigidity. In the classical case there is a short stage of tonic convulsions lasting about forty seconds, followed by a stage of clonic convulsions lasting one to three minutes. In the tonic stage the breath is held, the hands are clenched, the back is rigid, and the legs are extended, the pulse is quick and may be imperceptible. The clonic movements soon involve the whole body, and are sometimes of great violence, consisting of rapid extension and flexion of the limbs, opening and shutting of eyes and jaws. The interference with respiration during both the tonic and clonic stages causes stertor and an ever-increasing blueness of the face. The tongue is often bitten. During both these stages the pupils are dilated and inactive to light, and the conjunctival reflex is absent. The light reflex may return during the clonic stage. As the convulsions pass off the respiration becomes stertorous or snoring. Urine, fæces, and semen may be voided. The saliva issues from the mouth as a frothy foam, sometimes blood-stained from injury of the tongue. (5) A stage of *stupor* or *drowsiness* succeeds the convulsions, gradually passing into a deep sleep. The temperature directly after the convulsions is raised, sometimes as much as 4° or 5° F. (6) *Periodicity of Attacks.* It is a marked characteristic of idiopathic epilepsy that the fits occur between definite hours of the day or night. They may occur solely on rising or on retiring or may be strictly nocturnal. This is of importance in treatment. Epileptic fits tend to occur just before or just after menstrual periods. They tend to occur in groups at regular intervals, and may occur several times in one day and then disappear for weeks or months.

Special Symptoms.—*Status Epilepticus* is a rare condition in which the patient has a series of fits occurring in very rapid succession for several hours or even days, consciousness not being regained in the intervals; the temperature may rise to 107° F., and the issue may be

fatal. Status epilepticus may occur with local cerebral disease, *e.g.*, in G.P.I. and Encephalitis. *Post-Epileptic Automatism* ("Masked Epilepsy").—After an attack, often a minor attack, the patient may perform automatic, irresponsible acts, dressing or undressing himself, or putting the property of others into his own pockets. These automatic phenomena of the post-epileptic state seem to be generally in inverse proportion to the severity of the seizure, which may be so slight as to escape notice. He may pass into a state of dual personality, lasting for days altogether, or sometimes longer, and during this state may perform criminal, or even homicidal acts. No recollection of these automatic actions is afterwards retained. *Epileptic Equivalents.*—Mental disturbances, usually taking the form of attacks of bad temper, sometimes appear to take the place of a fit and are spoken of as "psychic equivalents." These may, however, be part of the mental changes seen in such patients, or may be transitory post-epileptic phenomena. *Mental Changes.*—Many of the patients, but by no means all, show signs of progressive mental deterioration, which may culminate in *epileptic insanity* (§ 903). This is seen chiefly in epilepsy commencing in childhood. The patient becomes egotistical and boastful, irritable, untruthful and spiteful. Transient grandiose delusions may occur, and violent, sometimes homicidal attacks. Acute mania may occur as a transient phenomenon after an attack. These chronic changes are probably due to progressive cortical degeneration. Other cases become dull, lethargic; these symptoms disappearing after a bout of attacks. *Simple jactitation* ("the jerks"), without loss of consciousness, occurs in many chronic epileptics.

II. **Minor Epilepsy** (Petit Mal), see § 719.

§ 722. III. Special Varieties of Epilepsy.

(1) **Jacksonian Epilepsy (Focal Epilepsy).**—This variety is common in focal *cortical* lesions, but may occur in idiopathic epilepsy. (*a*) Motor, (*b*) Sensory, (*c*) Visual, (*d*) Auditory, and (*e*) Uncinate attacks are described.

(*a*) *Motor Jacksonian Fits* commence unilaterally in the thumb or index finger, the corner of the mouth or the hallux. The attack spreads in an orderly march, according to the arrangement of movements in the motor cortical area (see Fig. 157). The point of starting indicates the position of the lesion in the cortex. The onset is with tonic spasm; later, broken or clonic twitchings occur, the whole attack occupying, perhaps, twenty minutes. The movements are confined, for a long period, to one limb or one side of the body, and consciousness is retained. Sometimes Jacksonian attacks terminate in a generalised convulsion, with loss of consciousness. The Jacksonian fit may be followed by a transient local paralysis (usually a monoplegia) or, if the convulsion is right-sided, by temporary aphasia. This paralysis is known as *Todd's Paralysis*, and may last hours or even days after the fit. (*b*) *Sensory Jacksonian fits* occur in parietal cortical lesions with numbness and tingling, starting locally, and spreading in an orderly march. These may be followed by transient astereognosis. (*c*) *Visual attacks*, blinding flashes of light of hemianopic distribution, followed by hemianopia, occur in occipital cortical lesions, and (*d*) *Auditory attacks*, sudden hallucinations of sound, followed by deafness, in temporal lobe lesions. (*e*) *Uncinate fits* occur in lesions of the uncinate gyrus or lesions in its neighbourhood (*e.g.*, in pituitary neoplasms) and consist of (i.) an intensely unpleasant flavour or odour, (ii.) champing or spitting movements, (iii.) a characteristic "dreamy" state.

Causes of Jacksonian Attacks.—(1) Any irritative cortical lesion, *e.g.*, depressed fracture of skull, meningeal scarring, with local vascular changes, extra-dural and subdural hæmatoma or abscess, cerebral tumour, granuloma or angioma, cortical softening in cerebral arterio-sclerosis, cerebral syphilis, etc. (2) General Paralysis of the Insane. (3) Uræmia. (4) Infantile Hemiplegia. (5) Idiopathic Epilepsy.

(2) **Flaccid Epilepsy.** The patient may or may not lose consciousness for a moment, toppling suddenly to the ground in a limp condition, often falling so heavily that injury is caused ("drop seizures"). No convulsion occurs and no tonic or clonic contraction of muscles. The attack is usually finished in a second or two, but may last longer.

(3) **Tonic Fits.**—These occur in mid-brain lesions and in lesions of the vermis of the cerebellum, or even in the absence of discoverable local lesion. The patient loses consciousness and the attitude is one of retraction of the head, flexion of the upper and extension of the lower limbs (§ 764). There is no clonic component.

(4) **Psychomotor seizures** are periods of amnesia or automatism which alternate with or may replace grand mal attacks.

(5) **Pyknolepsy.**—In children, between the ages of 4 and 12 years, attacks of minor epilepsy occur up to the number of fifty daily. No mental deterioration occurs, and the attacks cease at puberty. The condition cannot be diagnosed with accuracy until spontaneous cessation of all fits has occurred.

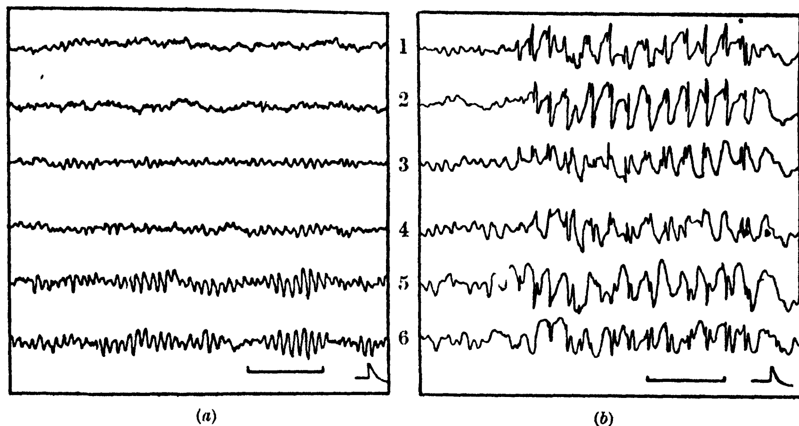
(6) **Reflex Epilepsy.**—Local or general convulsions, with loss of consciousness, start as the result of stimulation of a cutaneous epileptogenic zone. Pinching or pricking the sole of the foot may start a Jacksonian attack, and the attack may be prevented by tying a tight band round the limb above the area stimulated. This phenomenon occurs also with focal cortical lesions. Stranger still are the cases in which a sudden loud noise or bright flash of light will cause the patient to fall to the ground in a fit.

Diagnosis.—The diagnosis from *Symptomatic Epilepsy* is made by the finding of associated signs or symptoms of the causal disease (*vide infra*). In fits commencing after the age of 25 years, evidence of *G.P.I.*, *intracranial vascular disease*, *cardiac* and *renal* disease, should be sought for. It should never be forgotten that generalised fits may be the first and, for many months, the only sign of an *intracranial tumour.*

Electro-encephalography. A normal electro-encephalogram does not exclude epilepsy. Less than a third of epileptics examined in the interparoxysmal period show characteristic changes. A few show larval attacks: a gradually appearing outburst of fast waves, 30 per second. Cases of petit mal may show characteristic large voltage flat-topped waves formed by one or several rapid deflections at a rate of 3 per second. In those of unstable nervous constitution the electro-encephalogram may be abnormal although not significant of epilepsy. The records require expert interpretation.

Prognosis.—The probability of cure is greater when the patient is willing to co-operate for years in carrying out effective treatment. The outlook is better in the hereditary cases. Spontaneous cessation of epilepsy seems to occur fairly often. Attacks in infancy often disappear, to recur at puberty. The prognosis is bad when there is definite mental impairment or when the attacks start at puberty. Pregnancy affects the disease adversely. Death rarely occurs during the fit, but severe burns, often of the face, fractures and dislocations, occur comparatively frequently in chronic epileptics. Marriage cannot be advised, as the likelihood of occurrence of epilepsy or nervous instability in the family or succeeding generations, is very great. If marriage is undertaken

the condition should be explained to both partners and "protected" marriage advised.



(a) (b)

1. Right Frontal; 2. Left Fronta ; 3. Right Parietal;
4. Left Parietal; 5. Right Occipital; 6. Left Occipital.

FIG. 174.—(a) Normal E.E.G. record. The horizontal line represents 1 second and the vertical signal 50 microvolts. Alpha rhythm of 10 cycles per second and of relatively high voltage is seen arising in the occipital region.

(b) Part of a record from a patient with idiopathic epilepsy. An outburst of high-voltage waves of 3 per second, alternating with spikes, occurs simultaneously in all leads and lasts for 4 seconds.

(Reproduced by permission of Dr. Denis Williams.)

Etiology.—Both sexes are equally affected. The disease is only rarely heredo-familial, but a history of nervous instability in the family—migraine, eccentricity, alcoholism, insanity, is common.

Treatment.—Three factors will precipitate attacks: (1) Constipation, (2) Overeating, (3) Emotional excitement or worry. These should be avoided. Often, simple restriction of diet, and of eating between meals (many chronic epileptics are gluttonous) and correction of constipation, will diminish the frequency and severity of the attacks. If the occupation can be chosen, an open-air one should be advised. Epileptics should be excluded from all work involving danger. Possible safe occupations are farming and gardening, or indoor work at home. Most epileptics, however, lose their employment. Riding, swimming, and driving a car or riding a bicycle, should be forbidden. Alcohol is contra-indicated. *Where there is an epileptic in the family all fires should be protected with metal screens.*

Diet.—In severe cases a white diet is advisable, red meat is allowed occasionally. In children, a ketogenic diet alone may reduce or entirely stop the attacks. This treatment is not effective in adults. Fat is increased, while carbohydrate and protein are cut down so as to produce ketosis. Fats are given as bacon, suet-pudding, extra rations of butter or margarine, toast dipped in bacon-fat, etc. Not every child can be made to take such a diet. *Drugs.*—The treatment of epilepsy is by medicines. This should be impressed on the patient, who will often

foolishly break the continuity of his treatment after a bout of attacks, attributing his seizures, not to his malady, but to the "drugs" he is taking. In other cases, depression or some other emotional condition is attributed to the medicine, which is stopped by the patient or the parents. If these things are explained at the outset, the likelihood of this happening is less. *In prescribing for epilepsy remember to time the dose of the medicine to precede the occurrence of the fits.* If the fits are all nocturnal, give the medicine last thing at night. If the fits all occur just after the patient gets up in the morning, give the medicine an hour before rising. In female patients, whose attacks occur round about the menstrual periods, the dose of the medicine should be increased, perhaps doubled, just before those times, and a brisk saline purgative, *e.g.*, Seidlitz powder, given in the morning. *Bromide* is probably the most useful drug. It has the disadvantage of causing unsightly, pustular skin eruptions—"bromide acne." This is obviated, to some extent, by giving arsenic in the medicine and restricting salt in the diet. The following mixture may be prescribed once to three times a day: potassium bromide gr. 10–15, liq. arsenicalis ♏ 1–2, tinct. belladonnæ ♏ 5, aquam ad ½ fl. oz. Insoluble *phenobarbitone B.P.* (*luminal*) in tablet form or the soluble sodium phenobarbitone may be given in ½ gr. to 1 gr. doses, once to three times a day. This drug may cause erythematous rashes with dizziness and drowsiness, if employed in larger doses. Patients acquire tolerance to phenobarbitone. It is a useful adjunct to bromide; it is effective in very small doses and can be given in tablet form, which is convenient. In most cases it should be prescribed with a daily dose of bromide, not alone. For cases who do not tolerate bromide, *borax* or potassium borotartrate may be given in 10–15 gr. doses. *In Epilepsy, if the administration of drugs is suddenly stopped, Status Epilepticus may ensue.* The medicines must be continued with religious regularity until the patient has been free from attacks for five or six years. *Sodium-diphenyl-hydantoinate* (*Sodium phenytoin, Epanutin*) is used, particularly for cases of grand mal, but only after all other remedies have been tried first. It is of value in the treatment of psychomotor attacks. Capsules contain 1½ gr. (for infants ¾ gr.). In giving epanutin one dose of phenobarbitone is replaced by a capsule of epanutin for the first week, and the phenobarbitone gradually replaced by epanutin up to 3–5 capsules daily. Spongy gums, tremors, ataxia, diplopia, and scarlatinal or morbilliform rashes are some of the toxic symptoms observed after its use. Should albumen or urobilin appear in the urine during its administration, the dose should be diminished or the drug stopped. *Tridione* (0·3 G.) in some cases will control petit mal attacks (not grand mal), but this drug is toxic and should not be given unless the patient is under continuous expert supervision. Frequent blood counts are necessary as it may cause agranulocytosis.

Treatment in Attacks.—Put the knotted edge of a handkerchief or a wooden pencil between the teeth. Loosen the clothing round the neck,

remove spectacles and false teeth and prevent the patient damaging himself in the clonic stage. *Treatment of Status Epilepticus.*—(1) Give immediately morphine gr. ¼–½, alone or with hyoscine hydrobromide gr. 1/100. (2) Wash out the colon by means of a soap and water enema, followed later by lavage with water or saline. (3) If the fits have not ceased, give 4 drachms of paraldehyde per rectum with an equal quantity of olive oil and repeat in six hours if necessary. Temperature is lowered by tepid sponging. (4) The patient should be fed (nasally, if necessary) with glucose and milk feeds to combat the acidosis. Inhalations of chloroform may be necessary to control the attacks.

§ **723.** B. SYMPTOMATIC EPILEPSY.—Epilepsy may be the presenting symptom, particularly in patients where the first fit occurs after the age of 25 years, of an intracranial tumour, cerebral syphilis, cerebral arterio-sclerosis, or even of cardiac or renal disease. Intracranial cysticercus cellulosæ may cause epileptiform attacks. In all these conditions the attacks may be focal Jacksonian attacks (motor, sensory, visual, auditory, gustatory) or generalised convulsions. The Jacksonian attacks of focal organic cortical lesions are sometimes distinguishable from those of idiopathic epilepsy by the fact that the paralysis following the fit (Todd's paralysis) becomes progressively greater and greater after each seizure, until a permanent residual monoplegia develops.

IV. CEREBRAL TUMOUR (§ 828).—For weeks or months generalised epileptic convulsions may be the only sign of cerebral tumour. With cortical neoplasms, focal Jacksonian attacks occur as the irritative sign, the type of attack varying with the site of the lesion.

V. **Cerebral Syphilis.**—The "congestive attacks" of G.P.I. may be *focal* (followed by transient paresis or aphasia) or *general*. In any form of cerebral syphilis epilepsy may occur. The finding of Argyll-Robertson pupils, cranial nerve palsies, or a history of syphilitic headaches or history of infection, indicate the diagnosis, which is made by examining the spinal fluid. The spinal fluid Wassermann reaction will be positive in nearly all the cases. When the syphilitic disease is mainly vascular (*e.g.*, syphilitic hemiplegia from endarteritis), not meningeal, the Wassermann reaction may be positive in the blood and negative in the spinal fluid.

VI. **Cerebral Arterio-sclerosis** (Synonym : Arterio-sclerotic Epilepsy).—Epilepsy, due to anoxæmia or the formation of mural thrombi in the diseased vessels, occurs in patients with high blood pressure, retinal arterio-sclerosis and hæmorrhages, and a history of "strokes."

VII. The HYPERTENSIVE ATTACK is described in § 94. I. *c*.

VIII. **Traumatic Epilepsy.**—Following a severe head injury (often with sepsis), generalised epileptiform convulsions may occur. In cerebral contusions and depressed fracture, with meningeal and cortical injury, vascular brain scars form and exert traction on the cortical areas, with resulting Jacksonian convulsions. In this latter group surgical removal of the scar may alleviate the condition, otherwise the treatment is as in idiopathic epilepsy.

IX. In CONGENITAL DISEASE of the central nervous system, *e.g.*, syringomyelia, cerebral diplegia and hydrocephalus, fits may occur.

X. STOKES-ADAMS' SYNDROME, in incomplete heart-block, is described in § 69.

XI. CEREBRAL HÆMORRHAGE OR EMBOLISM may be ushered in by fits, followed by a flaccid hemiplegia.

XII. Asphyxia, from a foreign body in the larynx, may give rise to unconsciousness, with generalised convulsions, especially in children.

XIII. Forced Deep Breathing will induce epileptic fits in certain individuals.

XIV. Toxic Convulsions occur as the result of (*a*) metabolic poisoning, *e.g.*, *Uræmia* (test the urine, estimate the blood pressure and look for albuminuric retinitis). *Eclampsia* (when the fits occur before, during, or after labour and are associated with albuminuria and signs of toxæmia), *Acute Yellow Atrophy of the Liver* (with jaundice), *Insulin hypoglycæmia* (look for prick-marks on the limbs) or (*b*) *Drug Poisoning, e.g.*, Alcohol, Absinthe, Neoarsphenamine, Lead Poisoning, Belladonna, Hydrocyanic Acid.

XV. Acute Encephalitis, and

XVI. Malaria, especially Cerebral Malaria, may be attended by epileptiform fits.

XVII. **Intracranial Cysticercus Cellulosæ** (*T. Solium*) is a rare cause of focal or generalised convulsions, especially in those who have lived abroad (§ 316). Cysticerci may be present in the subcutaneous tissues or muscles as hard, oval, pea-like bodies. If calcified, the nodules show up in radiograms of the skull or skeletal muscles.

XVIII. Presenile Dementia may first be manifested by recurrent fits in a person past middle age (§ 903).

§ 724. **Infantile Convulsions.**—In circumstances causing rigors in an adult, convulsions occur in infants. Convulsions in infants may also be due to local disease of the brain or its membranes, or to toxic causes. Recognition of the convulsion is easy. In determining the cause a consideration of the child's age is important.

A. Convulsions in the First Three Months of Life.
1. Birth Injuries, *e.g.*, Tentorial laceration.
2. Cerebral Diplegia and Cerebro-Macular Degeneration.
3. Congenital Hydrocephalus.
4. Congenital Syphilis.

B. Convulsions from Six to Eighteen Months of Life.
5. Rickets with Tetany.
6. Gastro-intestinal Causes—Over-feeding, Unsuitable Food, Constipation.
7. Acute Infective Disorders—Broncho-pneumonia and Measles, Tonsillitis, Bacilluria.
8. Cerebral and Meningeal Causes—Meningitis, Polioencephalitis and other forms of Encephalitis.
9. Reflex Causes—Teething, Round-worms, Rectal irritation from Thread-worms.
10. During Cerebral Venous Congestion—in Whooping-Cough.

C. Convulsions after the age of Two Years.
11. Epilepsy is the commonest cause where the child is otherwise apparently healthy.

The commonest causes are rickets, gastro-intestinal disorders and acute infective diseases. Epilepsy may start at an early age and may be the cause of unexplained convulsions in infancy.

Treatment.—The rectal temperature should be taken and an attempt made to

(1) *Control the Convulsion.*—Immerse the infant in a warm mustard bath for ten to fifteen minutes, then place in a warm bed and give an enema, or insert a rectal glycerine suppository. The administration of chloroform, by inhalation, will immediately control the fits. Chloral

hydrate gr. 4, and sodium bromide gr. 5, may be injected through a catheter into the bowel and repeated in an hour. Next

(2) *Investigate the Cause.*—(*a*) Question the mother or nurse about the *diet*, with special attention to the question of over-feeding, unsuitable food or constipation. (*b*) Search for a history or signs of rickets and tetany. (*c*) Make a general physical examination of the child for rash, any source of sepsis (throat and ears), examine the chest, look for head retraction squint or Kernig's Sign, and lastly (*d*) Examine the stools and urine.

GROUP III. PYREXIA, WITH SIGNS OF ORGANIC NERVE DISEASE

Pyrexial disorders of the nervous system are not numerous, and most of them are due to acute inflammation of the meninges. In this group are considered certain conditions, all of which present *neurological symptoms* with PYREXIA and its attendant symptoms.

CLINICAL INVESTIGATION.—(1) Obtain a complete *Personal and Family history*, especially with regard to tuberculosis, previous suppurative ear disease, bronchiectasis, pneumonia, empyema, etc. In tuberculous meningitis there is often a history of preceding weeks of malaise. History of a rigor or fit may indicate the formation of a cerebral abscess. (2) The *age* of the patient is of some importance. Post-basic meningitis is almost confined to infants under one year. (3) Look for *signs of meningeal irritation*, *e.g.*, stiffness of the posterior cervical muscles, positive Kernig's Sign, etc. (4) Make as detailed a *neurological examination* as possible with special reference to the cranial nerves, tendon reflexes and plantar responses, signs of paresis in the limbs. Examine the fundi for optic neuritis (intracranial abscess), hæmorrhages (subarachnoid hæmorrhage) or tuberculous deposits (tuberculous meningitis). (5) Examine the *scalp, cranial bones, mastoid and other air sinuses, the ears, throat and lungs*, for evidence of infection. Œdema of the face, scalp or neck should be noted. Suppurative sinus disease is accompanied by facial œdema, extradural abscess by Pott's puffy tumour of the scalp, and sinus thrombosis by orbital œdema or œdema of the neck. (6) In nearly all cases it is necessary to examine the *spinal fluid* for Pressure, Cells, Organisms, Total Chlorides, Glucose, Total Protein, etc. (7) The *temperature* should be recorded on a four-hourly chart.

The following Clinical pictures may be encountered:

A. A patient with a *Chronic Nervous Disease* or an *Acute Non-Infective Nervous Disease* shows *Transient Pyrexia* .. § 725
B. A patient shows *Pyrexia* with signs of *Meningeal Irritation* § 726
C. A patient shows *Pyrexia* with subsequent *Flaccid Paralysis*.. § 732
D. A patient shows *Pyrexia* accompanied by *Rapidly Ascending or Descending Paralysis* .. .. § 734
E. A patient with a *Suppurative Focus* in the *Middle Ear, Mastoid, Accessory Air Sinuses, Thorax or elsewhere,* develops *Pyrexia* with *Localising Nervous Symptoms* .. § 737
F. Following upon *Vaccination* or an *Acute Specific Fever*, a patient develops *Pyrexia* and *Nervous Symptoms* .. § 740
G. A patient develops mild *Pyrexia*, with *double vision, ptosis, ocular palsies and lethargy* .. .. § 698

A. *A patient with a Chronic Nervous Disease or an Acute Non-Infective Nervous Disease, shows* TRANSIENT PYREXIA.

§ **725.** In **chronic diseases of the nervous system** transient fever may occur from INFECTION OF THE BLADDER OF INFECTED BED-SORES. Patients with the remitting type of DISSEMINATED SCLEROSIS will often give a history of "influenzal attacks" which are doubtless pyrexial attacks associated with the laying down of fresh plaques. Fever of 102°–104° F. may occur in the "congestive attacks" of GENERAL PARALYSIS OF THE INSANE, and it should not be forgotten that there are many such cases with RESIDUAL MALARIAL INFECTION following malarial treatment. In **acute** cerebral lesions of any kind, notably INTRA- or EXTRA-CEREBRAL HÆMORRHAGE OF CEREBRAL CONTUSION, the temperature is subnormal during the initial stages of collapse, but subsequently rises above normal. In hæmorrhage into the *pons* hyperpyrexia is frequent, and may be as much as 108° F. Convulsions occur, affecting chiefly the legs, with coma, bilateral extensor responses and strongly contracted pupils. Death occurs rapidly. The occurrence of "nervous fever" has, in fact, no foundation. Lesions of the *hypothalamic region* and the *tuber cinereum* may cause hyperpyrexia.

We are here, however, chiefly concerned with infective conditions of the nervous system, mostly of acute onset, where there is a combination of nervous symptoms and pyrexia.

B. *A patient shows* PYREXIA *with signs of* MENINGEAL IRRITATION. The condition to be suspected is MENINGITIS.

§ **726.** *The Clinical Picture of* **Meningeal Irritation.**—There is intense occipito-cervical headache with photophobia and great sensitiveness to noise, pain in the back, rigidity of the posterior cervical muscles and spine, and retraction of the head. The patient is irritable, later drowsy, and finally comatose. He lies on his side curled up and may be resistive if disturbed. In the early stages retention of urine is a common feature and should always be looked for. There are bouts of restlessness, with a monotonous, rather high-pitched wailing cry.

The only signs may be the characteristic headache and stiffness of the neck (§ 707). *Kernig's Sign* is inability to extend the knee with the thigh flexed at a right angle on the abdomen; it depends on spasm of the hamstrings. *Brudzinski's Sign* is the appearance of similar flexion of the lower limbs and sometimes also the upper limbs, when the head is acutely flexed on the chest.

In meningococcal and tuberculous meningitis other features complicating this clear-cut clinical picture may appear, depending on the spread of the inflammatory process. The knee- and ankle-jerks may disappear, signs of hemiparesis, pupillary abnormalities, squints and other cranial nerve palsies are often found.

Types of Meningitis (Lepto-meningitis).

MENINGOCOCCAL MENINGITIS (Syn. Cerebrospinal fever). See § 503.

§ **727. Tuberculous Lepto-meningitis** is usually part of a general miliary tuberculosis, the meningeal over-shadowing the other signs. Metastatic infection can also occur by erosion into a vein of caseating material from a mediastinal or mesenteric gland, or from a focus in bone or lung. It is commonest in children under the age of five years, running an acute and

TABLE XLVIII.—CAUSES OF MENINGEAL SYMPTOMS.

	Mode of Onset.	*Clinical Picture.*	*Signs of Disease Elsewhere.*	*Spinal Fluid.*
Subarachnoid Hæmorrhage. (§ 717).	Sudden, with intense headache and deepening coma. History of migraine.	Meningeal irritation, with ocular palsies, hemiparesis, root-pains, massive albuminuria.	Usually none. Sometimes hyperpiesis, renal or blood diseases.	Pink or yellow from laked blood. Sterile.
Tuberculous Meningitis (§ 727).	History of preceding weeks of malaise. Family or personal history of Tuberculosis.	Meningeal irritation with pupillary abnormalities, squint, absence of deep reflexes and abdominal retraction.	Signs of Miliary Tuberculosis.	Clear or yellow, with "web-clot" containing Tubercle Bacilli. Diminished glucose and chlorides.
Benign Lymphocytic Meningitis (§ 728).	Subacute in young adults.	Meningeal irritation but patient otherwise in good health.	None.	Always clear and free from clot. Glucose and chlorides not reduced.
Meningococcal Meningitis (§ 503).	Gradual onset. Perhaps rash.	Intense head-retraction in post-basic variety.	None, or signs of Cerebro - spinal fever.	Turbid fluid with flocculi. Meningococci.
Acute Poliomyelitis (§ 732. 1).	Acute Onset, during epidemics.	Meningeal irritation. Later, flaccid paralysis.	None.	Clear, sterile, with lymphocytosis and normal chlorides.
Pneumococcal and Pyogenic Meningitis (§ 729), or *Leaking* Cerebral Abscess (§ 737).	Acute Onset.	Meningeal irritation with ocular palsies and disappearance of deep reflexes. Localising signs in abscess.	Suppurative focus in mastoid, lungs, or elsewhere.	Purulent fluid, with causal organism. Fluid may be sterile in leaking abscess.
Meningism (§ 731).	Acute Onset.	Meningeal irritation only.	Bronchopneumonia or Apical Pneumonia, acute infectious diseases, Baciluria, Hysteria.	Normal.

fatal course, but it also occurs in adults when it tends, in some cases, to assume a more chronic form.

Symptoms.—In children the early symptoms are most insidious. The child loses appetite, becomes thinner, peevish and apathetic. A slight irregular fever and constipation are common and occasional vomiting occurs. Complaints of pain or discomfort in the neck should never be ignored. Such symptoms *persist* over a period of several weeks and then become rapidly worse. It is in this early stage that treatment may help the patient. In the *irritative stage* the meningeal signs are well-marked with headache, neck rigidity, vomiting and photophobia. Persistent severe vomiting suggests hydrocephalus. Later, signs of *compression* appear—drowsiness, abdominal retraction, flaccidity and loss of tendon reflexes, followed by *coma*. In adults the disease may run a course of weeks or months. Cases of *benign lymphocytic meningitis* closely simulate the tuberculous variety in the cerebro-spinal fluid findings, but in these recovery is usually complete and the chlorides never fall as they do in tuberculous meningitis.

Diagnosis.—Examination of the cerebro-spinal fluid should not be delayed when the disease is suspected. The encouraging results from streptomycin treatment are in cases diagnosed and treated early. At least two adequate samples of fluid should be collected by spinal puncture and the fluid should be examined for cells, total protein, globulin, chlorides, and glucose. Centrifuged deposits should be searched for tubercle bacilli, cultures should be made and a guinea pig injected. In the early stages the fluid is clear and perhaps under pressure. On standing and cooling a "spider web" clot may form, in the meshes of which tubercle bacilli may be entangled. If the cells are more than 300 per cu. mm. the fluid may be opalescent. Up to 30 per cent. of the cells may be polymorphs, but lymphocytes predominate. The chloride level frequently falls to 650 or 600 mgm. per cent. and may be as low as 500 mgm. per cent. These low chloride readings are highly characteristic of the disease (normal is 720–750 mgm. per cent.). The glucose is diminished. Other tests are less helpful but may have to be carried out, viz., (1) Mantoux test (see § 521), (2) radiograms of chest, (3) choroidal tubercles are found in the ocular fundi early in the disease in as many as half the cases, when the spread is a miliary one.

Course and Prognosis.—The discovery of streptomycin by Waksman (1944) has modified the course and perhaps the prognosis of a disease which was almost invariably fatal. In children untreated cases are invariably fatal within three weeks of the appearance of meningeal symptoms. In cases treated with streptomycin the mortality under the age of three years is still very high, but in adults 30 to 40 per cent. are "in good clinical condition" six months after the onset. A few of the remitted cases are left with permanent optic atrophy, squint, dysphasia or mental disorder, but these complications are exceptional. Relapses occur and it is still too early to assess the final results.

Treatment.—Symptomatic treatment consists of repeated spinal puncture to relieve headache, with ice-bags to the head, chloral hydrate, codein and other analgesic drugs by mouth or by nasal tube to control pain and restlessness; with avoidance of noise and bright light. In stuporose patients a nasal tube for feeding is essential.

Streptomycin is available in England at special hospital centres. Combined intramuscular and intrathecal injections are used. *Intramuscularly* the daily dose advised is 0·02 G. per lb. of body weight, given twelve hourly in divided doses, for three to six months. *Intrathecally* the daily dose advised is 0·05 to 0·1 G. in one dose, given six hours after the last intramuscular injection. If spinal block develops, the streptomycin must be given into the ventricles through burr holes made in the skull.

§ 728. Benign Lymphocytic Meningitis. The *Signs* are those of meningitis running a sub-acute course in young adults. Constitutional disturbance is usually slight. At first the illness may be mistaken for tuberculous meningitis or acute poliomyelitis, owing to the large number of lymphocytes found in the sterile spinal fluid. The chloride content of the

fluid, however, does not fall as in tuberculous meningitis and signs of muscular paresis are absent. The disease may be due to a virus similar to that producing chorio-lymphocytic meningitis in mice. In some cases the symptoms come on after acute catarrhal infections of the upper respiratory tract. The illness runs a benign course but an excess of lymphocytes may persist in the spinal fluid for weeks after all clinical signs have disappeared. *Treatment* is symptomatic.

§ 729. **Pyogenic and other forms of Acute Lepto-meningitis.** Most of these cases are secondary to a focus of infection outside the nervous system. The organisms concerned are the *Pneumococcus*, *Streptococcus*, *Staphylococcus*, *Gonococcus* and *H. influenzæ*.

Pneumococcal Meningitis may be secondary to otitis media, empyema, pneumonia, pneumococcal peritonitis or arthritis. The spinal fluid is sometimes too thick to flow through a lumbar puncture needle: it is greenish-yellow and contains abundance of polymorphonuclear leucocytes amongst which the pneumococcus is found. The condition develops rapidly, coma supervenes early and before the introduction of sulphonamides the disease was nearly always fatal. In many cases there is an associated blood infection.

Streptococcal Meningitis may be secondary to middle-ear or mastoid disease, erysipelas, or other infections of the scalp, face or cranial air sinuses. *Staphylococcal* cases are secondary to skin or bone infections. *Gonococcal* cases occur. *B. Pyocyaneus Meningitis* may be caused through accidental infection during intrathecal administration of penicillin. *Influenzal Meningitis* is a rare cause of purulent meningitis in children.

The C.S.F. findings in such cases are shown in Table LXI.

Treatment of Acute Lepto-meningitis. Before giving either sulphonamides or penicillin, endeavour to identify the causal organism in the C.S.F. This usually means withdrawing at least two adequate samples of spinal fluid and its laboratory examination as soon as possible afterwards. If this is made a rule, much subsequent confusion will be avoided. Oral *Sulphadiazine* is the sulphonamide most commonly used in treatment. It is active against the meningococcus, pneumococcus, streptococcus hæmolyticus and staphylococcus aureus. Give an initial dose of 4 G. by mouth (by nasal tube, or intramuscularly, if the patient is unconscious) followed by 2 G. doses by mouth four-hourly, for four or five days; then half-doses for three days. The patient must be wakened up for his four-hourly doses through the night (see Tables XXVIII and XXIX). Systemic *penicillin* does not pass the blood-brain barrier and is therefore only an adjuvant to treatment with sulphadiazine. Give 120,000 units intramuscularly daily for four or five days. It is safe to do this in an unidentified meningitis if sulphadiazine is given as well. Penicillin should never be the only form of chemotherapy in acute meningitis. Very rarely it may be necessary to give intrathecal penicillin when the causal organism is penicillin-sensitive and is not reacting to other treatment. Penicillin intrathecally is to some extent an irritant and may cause headache, stupor

and fits. Pure crystalline penicillin should be used and the greatest care taken to avoid bacterial contamination, for B. pyocyaneus meningitis is very difficult to cure. Give 10,000 units in 10 c.c. warm sterile saline intrathecally, and repeat in twelve hours. Often these two doses are sufficient: if not, the dose may be repeated once daily for three days. If there is a spinal block, the penicillin may have to be given by ventricular or cisternal puncture, burr holes being drilled in the skull for the ventricular administration. In all cases when the patient is unconscious, a nasal tube should be passed into the stomach so that adequate fluids and feeds can be given.

The primary focus of the meningitis may have to be dealt with surgically.

Anthrax Meningitis is a rapidly fatal form. The spinal fluid is hæmorrhagic. **Blastomycotic meningitis** is a very rare form secondary to infection of the skin or lungs with organisms of the yeast group.

§ **730. Diagnostic Spinal Puncture in Meningitis.**—In all cases of the meningeal syndrome, a diagnostic spinal puncture will be necessary to determine whether there is meningeal infection or whether the syndrome is due to SUBARACHNOID HÆMORRHAGE OR MENINGISM. In meningeal infection the fluid analysis will determine the *type of meningitis* and its *bacterial cause.* This laboratory information is essential before treatment can be started. It is reasonable to give oral sulphonamides and systemic penicillin until the bacteriology can be determined as accurately as possible (see Tables XLVIII, LXI).

Recurrent meningeal infection may take place from a focus of infection in the extra-dural or sub-dural space or from a leaking intra-cerebral abscess.

§ **731. Meningism** is a condition of meningeal irritability characterised clinically by the picture of meningeal irritation but with normal spinal fluid. The condition closely simulates meningitis and occurs especially in children, in *Broncho-pneumonia* and *Apical Lobar Pneumonia*, at the onset of *Acute Specific Fevers*, *Otitis media* and during severe *Pyelitis.* In adults meningism occurs in *Apical Pneumonia*, *Typhoid*, *Acute Tuberculosis* and without fever in *Hysteria* (hysterical pseudo-meningism).

C. *A patient shows* PYREXIA *with subsequent* FLACCID PARALYSIS. *The condition is either* I. ACUTE POLIOMYELITIS; II. ACUTE INFECTIVE RADICULITIS; *or* III. HERPES ZOSTER.

§ **732.** I. **Acute Poliomyelitis** (Synonyms: Infantile Paralysis, Acute Anterior Poliomyelitis and Polioencephalitis) is an acute specific fever of sudden onset, resulting in loss of power and rapid wasting of one or more groups of muscles. No age is exempt, but the disease usually affects children between the ages of two and five years. During the first year of life there appears to be relative immunity from infection and the disease is uncommon after middle life. The *incubation period* is probably about twelve days. Two stages are recognised: (i.) the pre-

paralytic stage and (ii.) the paralytic stage. The virus seems to invade the nervous system exclusively, and may reach the motor nerve cells before any symptoms appear.

Symptoms.—The symptoms are those of a mild febrile attack and last a few days, rarely more than seven. Paralysis may or may not follow. (1) The *Pre-Paralytic Stage* passes unnoticed in the majority of cases. Pyrexia, general malaise and fretfulness occur, with pain in the limbs. The muscles are tender to pressure and the joints painful when the limbs are moved. Local symptoms: (i.) redness of the tonsils and fauces, (ii.) coryza, with streaming of the eyes or (iii.) gastro-intestinal upset, with anorexia, vomiting and diarrhœa. In more severe cases there is delirium and stupor. Rigidity of the posterior cervical muscles and spine appears, with severe headache and irritability, pains in the limbs, muscular twitchings, positive Kernig's sign, and in severe cases, incontinence. In the early stages the *blood* shows a polymorphonuclear leucocytosis, which may be marked (30,000 per cu. mm.).

The *spinal fluid* is now found to be under increased pressure, clear or opalescent, and it shows a lymphocytosis (occasionally, polymorphs are found), in numbers varying with the severity of the meningeal reaction. The albumen content is increased. The glucose and chloride content is normal; by the end of a fortnight the cell-count is usually normal. The combination of lymphocytosis in the spinal fluid with polymorphonuclear leucocytosis in the blood is quite characteristic.

In some cases (50 per cent. of cases in epidemics) the patient recovers without paralytic symptoms (*Abortive* or *Meningeal* types of the disease). Convalescent serum, even if administered early and intrathecally, does not avert paralysis.

(2) *The Paralytic Stage.*—(*a*) *Spinal Type.*—In infants the paralysis appears two or three days after the onset of the symptoms; in adults the paralysis appears earlier. The paralysis varies in degree: it is (i.) flaccid; (ii.) the corresponding tendon reflexes are lost; (iii.) the affected muscles are very tender, but there is no sensory impairment. (iv.) The paralysis most often affects the legs, and at first appears widespread, but residual paralysis is usually much less. (v.) There may be retention of urine, which soon passes off. Ascending and descending paralysis may occur, especially in adults, and may be rapidly fatal from respiratory paralysis. (*b*) In the *Brain-stem type* the motor nuclei of the brain-stem are affected. This causes facial paralysis, ocular palsies with nystagmus, motor trigeminal or bulbar palsies. The facial paralysis in these cases is usually of the peripheral type and tends to recovery. (*c*) *Cerebral, Cerebellar and Neuritic* forms are sometimes described. The *Cerebellar type* is said to produce cerebellar ataxy without nystagmus; rapid and complete recovery ensues as in other comparable acute cerebellar lesions. Poliomyelitis affecting the cerebrum is called *Polioencephalitis.* In this, mental confusion and restless delirium persist for some days after the pyrexia has subsided. Polioencephalitis is believed

to be a cause of infantile hemiplegia and perhaps some cases of epilepsy.

Diagnosis.—Careful clinical examination usually discloses swelling and redness of joint or joints in *acute rheumatism* and enlargement of the epiphysis in *syphilitic epiphysitis.* In both these diseases the tendon reflexes are brisk, not lost as in poliomyelitis. Examination of the spinal fluid settles the question. In *infantile scurvy* the affected limbs are tender and swollen (§ 545). The ascending and descending types of poliomyelitis closely resemble *acute febrile polyneuritis* and *Landry's paralysis*, but are distinguished by the spinal fluid findings. In *encephalitis lethargica* (§ 698) clot and polymorphonuclear cells are not found in the fluid. In *acute myelitis* there are sensory loss, bed-sores and incontinence (§ 736). In the convalescent stage of flaccid paralysis there is rarely any difficulty in diagnosis. The diagnosis is made certain by spinal puncture. The differential diagnosis from *tuberculous meningitis* is made by the finding of normal amounts of chloride and glucose in the spinal fluid, the absence of tubercle bacilli, and the polymorphonuclear leucocytosis in the blood in Poliomyelitis. Clear fluid and clot are common to the two conditions.

Etiology.—Noguchi and Flexner in 1913 isolated a neurotropic virus believed to be the cause of the disease. It is found in the brain and spinal cord of cases dead of the disease, and also in fæces, the intestinal canal and naso-pharynx. Unfortunately the only method of demonstrating the virus is by injection of emulsions of suspected material into certain monkeys, thus producing disease in the inoculated animals. The disease is believed to spread from healthy carriers and also from one infected individual to another, but whether by infected fæces or by "droplet" infection is unknown. The roles of water, milk and fomites in the spread of the disease is not understood, but patients' fæces should be treated as infective. The virus reaches the nervous system from the naso-pharynx or intestinal canal and the main incidence of the disease is on the motor cells of the spinal cord and brain stem.

Prognosis.—(1) The slight unrecognised forms of Poliomyelitis cause many orthopædic deformities. (2) The mortality in epidemics ranges from 11 to 20 per cent. and is greatest in the spreading form of the disease. (3) The severity of the general or the meningeal symptoms bears no relationship to the amount of residual paralysis, which may be severe when the general symptoms are slight, and *vice versa.* (4) The amount of paralysis present at the beginning of the paralytic stage is commonly much greater than the ultimate residual paralysis, except in the ascending or descending form, where further extension may occur for a few days. (5) Muscles incompletely paralysed will certainly recover. (6) The final prognosis as to ability to use the affected limbs or limb depends on whether the muscles affected perform essential functions in walking or prehension, and on certain orthopædic considerations. No case should be regarded as hopeless until months or years have elapsed, as recovery is often long

delayed. One attack confers immunity. The infectivity varies in degree in different epidemics. For Electro-prognosis, see § 709.

Treatment.—Hexamine gr. 10–30, sodium bicarbonate gr. 20, potassium citrate gr. 20, may be given in a mixture, well diluted, four-hourly. For the pains give aspirin. If the respiratory muscles are involved, and in the spreading types of the disease, the patient should be well propped up and atropine gr. 1/100 given four-hourly, to check the accumulation of bronchial secretion. The nose and nasopharynx should be sprayed with hydrogen peroxide (10 volumes) and an equal part of normal saline. *Isolation.*—This should be as strict as for other specific fevers. The infectivity probably continues for two or three weeks from the onset.

Rest and Posture.—The patient should be kept in bed, even in the mildest cases, for at least three weeks. The patient should be nursed on a water-bed or air-bed during the painful stages of the malady, which may last some weeks. When the erector spinæ muscles are involved, the patient should usually be nursed in the recumbent position, but in all other cases should be propped up. The affected muscles should not be allowed to over-stretch, otherwise appalling deformities from *contracture* will ensue. The relaxation of the affected muscles may be secured by sand-bags, bed boots, celluloid or poroplastic splints and pillows. *All apparatus should be of the lightest possible kind.* Plaster splints, which should be removable, are sometimes used. In a very few cases where the respiratory muscles are severely involved, life may be preserved by Drinker's or the Paul-Bragg Artificial Respiration Apparatus for weeks or months until recovery starts. Many cases will show no recovery, and the horrible situation may arise where life is possible only in the apparatus.

Re-education is of paramount importance in the treatment of the paralytic stage. Any voluntary movement performed by the patient is of much greater value in recovery than the same movement performed passively by a masseuse. Massage must never be regarded as an adequate substitute for active exercises. Faradism certainly cannot hasten recovery; it should never be used in the painful stage, nor can it replace re-educative exercises. Re-educative exercises may be assisted by immersing the limbs in a brine bath or may be undertaken in a salt-water swimming-bath, the water affording additional support to the affected limbs. A walking-machine on wheels is often necessary in re-education of the lower limbs. Contracture and deformity should never occur. When they have appeared they should be dealt with by passive manipulation and stretching, and by tenotomy or other orthopædic measures. In old paralysed limbs, cyanosis, œdema and chilblains may be relieved by lumbar sympathectomy.

Prophylaxis.—In the absence of precise knowledge regarding infection it is difficult to lay down dogmatic rules. When more than one case occurs in a boarding school, the prevailing custom at present is to quarantine the school for three weeks. Parents are given the option of leaving their child at school, or removal home: but as healthy children have to be isolated

for three weeks from other children and adolescents, parents often prefer to leave the child in the quarantined school. Cases which develop the disease should of course be rigidly isolated, treated as contagious and nursed with precautions as for typhoid fever. Nurses should wear gauze masks. The patient's stools and handkerchiefs are infectious. Contacts should not be allowed to handle food for four weeks. Human convalescent serum is not now believed to be of any value in treatment and it is of very doubtful value given prophylactically to contacts.

§ 733. II. **Acute Infective Radiculitis** ("Neuralgic amyotrophy").—After an initial febrile phase usually with pain, there appears flaccid paralysis and sensory loss of root distribution. The disease affects chiefly the roots of the cervical and upper dorsal segments, but sometimes individual muscles may be picked out and it is difficult to explain the findings in terms of affected roots. Paralysis of serratus anterior, rhomboids or trapezius is observed analogous to that seen in Bell's palsy. The disease may attack the lumbar or sacral roots. It is distinguished from poliomyelitis by the associated sensory findings and by the absence of spinal fluid changes.

III. In HERPES ZOSTER (§§ 635, 826) the muscles near the affected area may be paralysed; ocular palsies, facial palsies and upper and lower brachial palsies may occur in herpes of the face and neck. Recovery is usually very slow; paralysis may be permanent. Treatment is by splinting and re-education.

D. *A patient shows* PYREXIA *accompanied by rapidly* ASCENDING *or* DESCENDING PARALYSIS. *The disease may be :* I. ASCENDING *or* DESCENDING POLIOMYELITIS ; II. ACUTE FEBRILE POLYNEURITIS ; III. LANDRY'S PARALYSIS ; IV. ACUTE MYELITIS.

I. The Ascending and Descending Types of POLIOMYELITIS are described above.

§ 734. II. **Acute Febrile Polyneuritis** is a rare condition. Following a short febrile attack, spreading flaccid paralysis of muscles appears, with tenderness of muscles to deep pressure, pains, paræsthesiæ, numbness, tingling, and pins and needles in the chest and limbs, or diminution of deep reflexes. Occasionally fever is absent. Remissions and exacerbations occur. The paralysis differs from other forms of polyneuritis in that (1) it affects chiefly the proximal limb muscles, and (2) cranial nerve palsies, especially peripheral paralysis of the facial, are frequent. Sensory disturbances are of the "glove and stocking" type, or are absent, the plantar responses are flexor, there is no sphincter disturbance, no incontinence and no bed-sores. The affected muscles waste only slightly and retain some excitability to faradism throughout. The *cerebro-spinal fluid* contains excess of albumen and may be yellowish or brown, and may clot spontaneously, but is otherwise normal. (1) Toxic myocarditis may be present. (2) Rapid extension of the paralysis may occur as late as the sixth week and may prove fatal. (3) Some cases recover, but death may occur from respiratory failure.

The *diagnosis* from *Poliomyelitis* is made by (1) the presence of paræsthesiæ, (2) the slower progress of the paralysis, and (3) the spinal fluid findings. Other forms of polyneuritis affect the distal segments, but *diphtheria* and *diabetes* should be excluded.

§ 735. III. **Landry's Paralysis.**—This is probably the same disease as Acute Febrile Polyneuritis. Sensory disturbances are completely absent. The spinal fluid is hyperalbuminous but shows no other changes. In these two conditions there is no consistent pathological lesion present in the spinal cord or peripheral nerves.

§ 736. IV. **Acute Myelitis.**—In this disease a paraplegia develops acutely with a

febrile reaction, due to an unknown infective inflammatory process in the spinal cord. The patient is usually a young adult.

Symptoms.—(1) Marked paraplegia develops in association with fever, (2) pains in the back, and (3) numbness, tingling or a sense of *girdle constriction* round the body at the level of the lesion. (4) At the onset there is flaccid paraplegia with absence of tendon reflexes and retention of urine. (5) Sensory loss is present up to the level of the lesion. (6) Later, the paralysis becomes spastic, with reflex incontinence, the tendon reflexes are exaggerated, with clonus, and the plantars are extensor. Bed-sores develop. The paralysis and anæsthesia may ascend.

The *spinal fluid* may show the syndrome of Froin (*i.e.*, yellow coloration with hyperalbuminosis, formation of clot and relatively little pleocytosis), due to the formation of a zone of arachnoidal thickening and adhesion round the inflamed portion of the cord. If the spinal block persists, the syndrome of Froin may continue indefinitely in the spinal fluid.

Diagnosis.—The sensory loss and definite sensory level distinguishes this disease from other forms of spreading paralysis with fever, described above. This malady must be differentiated from other causes of acute paraplegia. (1) In *disseminated sclerosis*: evidence of disseminated lesions may be present clinically or ascertained by a careful history of previous illness. For other points, see § 755. (2) In *acute syphilitic meningomyelitis*: the Wassermann reaction is usually positive in blood and spinal fluid. (3) In the aged, sudden thrombosis of diseased arteries and veins, *myelo-malacia*, occurs. The presence of arterial disease, the age of the patient and the normal spinal fluid findings serve to differentiate these cases. (4) In *spinal cord compression*, particularly that associated with extension of tuberculous or pyogenic osteomyelitis of the vertebræ, the diagnosis may be very difficult, but is usually made by the slower onset of the symptoms.

Prognosis.—In the spreading and high cervical types of the lesion, death occurs in a few days from respiratory paralysis. There is a tendency in other cases for the disease to become arrested, usually with great residual disability.

Treatment.—See § 761.

E. *A patient with a* SUPPURATIVE FOCUS *in the Middle Ear, Mastoid, Accessory Air-sinuses, Thorax or elsewhere, develops* PYREXIA *with* LOCALIZING NERVOUS SYMPTOMS. The condition suspected is INTRACRANIAL ABSCESS.

§ **737. Intracranial Abscess.**—The majority of cases arise from a chronic infection in the middle ear or mastoid antrum usually with cholesteatoma. Infection spreads directly through the carious tegmen tympani to the under surface of the temporal lobe, or posteriorly into the lateral lobe of the cerebellum. Frontal lobe abscess arises in association with pansinusitis or chronic suppuration in the frontal or ethmoidal air-cells especially in children. Metastatic abscesses arise from chronic suppurative infections in the pleura, lungs or long bones.

All degrees of infectivity exist. The most virulent infections cause *acute suppurative encephalitis* with extensive diffluent cerebral softening and rapid death. Milder and more chronic infections from contiguous foci in the air-sinuses cause *pachymeningitis* with formation of sub-dural or extra-dural abscess. Metastatic intracerebral abscesses are often multiple. Otitic abscesses tend to be loculated and if chronic, they become enclosed in a fibrous capsule, in which state they are more amenable to surgical drainage or excision.

Symptoms.—Very rarely the formation of a pyæmic brain abscess

causes a rigor with meningeal or focal neurological signs. In almost all cases of otitic and pyæmic abscess the signs are latent for several weeks. The earliest changes are an alteration in the patient's mental and general condition. Apathy, defective attention, difficulty in expressing ideas and defect of memory are found. The patient begins to look ill, to lose flesh and the tongue becomes coated. There may or may not be elevation of temperature. In intracerebral abscess the pulse is slow; in extra-dural abscess the pulse rate is raised. In all cases the blood usually shows a polymorph leucocytosis. Papillitis is a late sign and headache is rarely marked. *Local Symptoms* are never marked in *Temporal Lobe Abscess.* Slight paresis of the lower face, diminished or absent abdominal reflexes and extensor plantar responses on the contra-lateral side may be found. Abscesses extending backwards in the temporal lobe involve the optic radiations with production of upper quadrantic hemianopic field defects. If the abscess is in the left temporal lobe and the patient is right-handed, there will be dysphasia, sometimes "jargon aphasia." Jacksonian fits may occur from cortical irritation. Occasionally signs of mid-brain compression are present, squint with diplopia or paralytic dilation of the ipsilateral pupil. In *Cerebellar Abscess* coarse horizontal nystagmus may be present on looking to the side of the lesion. Flaccidity of the ipsilateral upper limb with undue extensibility of the muscles may be present. There is no alteration in the tendon reflexes and the upper and lower abdominal reflexes remain brisk; a point which may be valuable in distinction from temporal abscess. The progressive apathy and drowsiness of the patient soon render clinical examination almost impossible. *Spinal fluid.*—Cells are perhaps 5–30 per cu. mm. and are mostly lymphocytes with a few polymorphs. This is the characteristic finding, but if the abscess is thick-walled, the cytology of the fluid may be normal. Protein may be increased. Glucose and chlorides are normal unless the abscess leaks, when signs of recurrent meningitis become apparent clinically, with fall in the cerebro-spinal fluid chlorides and rise in cell count.

Diagnosis is often extremely difficult. In selected cases air ventriculogram may be of great assistance in diagnosis, revealing abnormal dilation, displacement, or filling defects of the ventricles.

Treatment.—The treatment of intracranial abscess is surgical and the results have been very materially improved (1) by the use of penicillin and sulphonamides before, during and after operation, and (2) by the technique of aspiration and excision of the abscess with its capsule intact. Medical treatment pending surgical removal or drainage is as for purulent meningitis.

§ 738. **Pyogenic Sinus Thrombosis** causes (1) severe headache, vomiting and high fever of a pyogenic type, accompanied by rigors and sweats (see chart in § 515), (2) optic neuritis supervening in a day or two, and often photophobia, (3) drowsiness, deepening into coma and, if operative measures are not prompt, ending in death.

In *lateral sinus thrombosis* there are pain and tenderness in the mastoid region, together with other signs of suppurative otitis media; the inflammation spreads down the jugular vein on the same side, and backwards behind the mastoid; con-

sequently there is generally some hard brawny swelling in these positions. If there has previously been a discharge from the ear it usually ceases. When the *longitudinal sinus* is thrombosed the localising signs consist of œdema of the scalp, distension of the veins over the forehead and sometimes strabismus, associated with convulsions at the onset. There may be a spastic paraplegia or bilateral hemiplegia. With pyogenic thrombosis, the cause is usually some septic lesion of the face or scalp. When the *cavernous sinus* is affected the localising signs are œdema of the eyelids and root of the nose, sometimes also of the pharynx, exophthalmos and paralysis of the third, fourth, ophthalmic division of the fifth, and sixth nerves. There may be blindness from infarction of the retina, with retinal hæmorrhages and thromboses. Pyogenic thrombosis of this sinus may arise from some septic lesion of the orbit, nose, pharynx or face.

The *Diagnosis* of pyogenic sinus thrombosis is difficult unless the local signs are pronounced. In uncomplicated lateral sinus thrombosis there may be papillœdema, and this is not necessarily a sign of co-existing cerebral abscess. It may clear up when the sinus has been opened and drained and the jugular vein ligatured. The *Treatment* of lateral sinus thrombosis is a matter for an expert aural surgeon. In cavernous sinus thrombosis mortality is lowest when conservative treatment is employed. In septic cases, penicillin and sulphonamides are of great value.

§ 739. Cortical Thrombo-phlebitis. Although pathological confirmation is scanty, a spreading non-suppurative thrombo-phlebitis of the cortical veins is believed to exist. It is usually met in association with suppurative otitis, when it may cause foci of superficial encephalitis. The physical signs and spinal fluid findings are indistinguishable from those of abscess formation. In most cases, however, the condition resolves and the patient recovers.

Some cases of hemiplegia and other cerebral symptoms in women during the puerperium or after abortion are attributed to venous occlusions in the superior longitudinal sinus and cortical veins. Pelvic and femoral thromboses are often present. The hemiplegia comes on with convulsions or coma. In the sinus and cortical venous thromboses of puerperal women, the C.S.F. is under increased pressure but is normal in composition.

OTITIC HYDROCEPHALUS is met with in children and gives rise to papilloedema with signs of increasing intracranial pressure and a pleocytosis in the C.S.F. There are no signs of any focal brain lesion. It occurs in association with chronic otitis and is believed to be due to partial thrombosis of the superior longitudinal sinus with defective re-absorption of cerebro-spinal fluid.

F. *Following upon* VACCINATION *or an* ACUTE SPECIFIC FEVER *a patient develops* PYREXIA AND NERVOUS SYMPTOMS. The disease is ACUTE ENCEPHALITIS (Synonym: ACUTE ENCEPHALOMYELITIS).

§ 740. Post-Vaccinal Encephalomyelitis.—Six to fourteen days after vaccination with glycerinated calf-lymph the patient becomes acutely ill with fever, coma and delirium (§ 480). Meningeal signs may be present, with convulsions, trismus, papillœdema and various squints and pupillary abnormalities. The disease may end fatally, or the patient recovers with or without mental or physical sequelæ. A *spinal type* with sudden paraplegia and retention of urine also occurs but more rarely. Post-vaccinal encephalomyelitis has to be distinguished from severe constitutional reactions to vaccination seen in normal individuals and in the debilitated and mentally unstable. Pathologically, the brain and cord show in the white matter *perivascular zones of*

demyelination, with relatively little cellular infiltration. The grey matter suffers even more, and shows numerous punctate hæmorrhages. The disease has a definite incubation period after vaccination against small-pox, and has been produced experimentally in rabbits. It is probably due to the virus of vaccinia. Two members of a family, vaccinated on the same day, may develop the disease simultaneously.

§ 741. **Encephalomyelitis following Acute Specific Fevers.**—During the course of various acute specific fevers there may be a recrudescence of the fever with development of clinical signs, similar to those of Post-Vaccinial Encephalomyelitis. Cases are described in association with Measles, Chicken-pox, Small-pox, Mumps and Whooping-Cough.

Symptoms.—The onset is sudden during the first or second weeks of the illness. There is an exacerbation of fever and headache; delirium, diplopia and head retraction occur. Cranial nerve palsies, hemiplegia, paraplegia, with incontinence, or involuntary movements, may be observed. Papillœdema is common. After some days, during which the symptoms advance, recovery sets in and may be almost complete. In the *spinal form*, paraplegia, with retention of urine and incontinence of fæces develops. Sensory symptoms, ascending paralysis or root palsy may occur. If death does not take place within the first week, recovery ensues. The *spinal fluid* shows a lymphocytic reaction, with normal chloride and glucose content. The residual symptoms of the encephalomyelitis may be hemiplegia, spastic weakness of the legs, with or without sensory loss, involuntary movements or ataxia, fits, deafness or optic atrophy, and various cranial nerve palsies and pupillary abnormalities.

Treatment.—The appropriate convalescent serum may be given intrathecally, after withdrawing an equal amount of cerebro-spinal fluid.

§ 742. **Acute Disseminated Encephalomyelitis** is described (Brain and Hunter) as a clinical entity, probably distinct from disseminated sclerosis. These cases may be due to polioencephalitis. In all cases the spinal fluid shows excess of lymphocytes, but the characteristic clinical changes of acute meningitis are not found.

G. *A patient develops mild* PYREXIA *with* DOUBLE VISION, OCULAR PALSIES *and lethargy.* The disease is ENCEPHALITIS LETHARGICA.

ENCEPHALITIS LETHARGICA is fully described in § 698. The acute phases may be characterised by a febrile illness of some days' duration, with headache, pains in the limbs, malaise and constipation. Later supervene the striking drowsiness by day, with muttering occupational delirium by night and diplopia, ptosis, squint, pupillary abnormalities and nystagmus.

Any of the foregoing illnesses may be attended by **Febrile Delirium** (see § 469.)

GROUP IV. DEFECTS OF SPEECH AND ARTICULATION

I. *The patient has difficulty in exteriorising his ideas in speech or writing, or you are unable to communicate with the patient because he cannot recognise words spoken to or written for him.* The condition is APHASIA.

§ 743. **Aphasia** is the term applied to severe speech defects of cortical origin. *Dysphasia* is a milder degree of aphasia. Aphasia is of two kinds —motor and sensory. In *Motor Aphasia* there is loss of power to exteriorise thought in spoken or written words—the patient knows what he wishes to say but cannot say it, although the peripheral neuromuscular mechanism of articulation is intact. In *Sensory Aphasia* there is defect of comprehension of spoken speech (word-deafness) or written speech (word-blindness) in the absence of deafness or blindness; the words are heard or

seen by the patient but convey imperfect ideas or nothing at all, to him.

The symbols of speech are vocal words or written words. The movements which govern the production of vocal or written speech are under the controlling influence of sensory impressions. When we speak we use our auditory memories of words to control the correctness of our speech movements, and when we write we use our visual memories to control the correctness of our writing movements. Hence, it is that aphasias are rarely purely motor or purely sensory, but more often a combined defect.

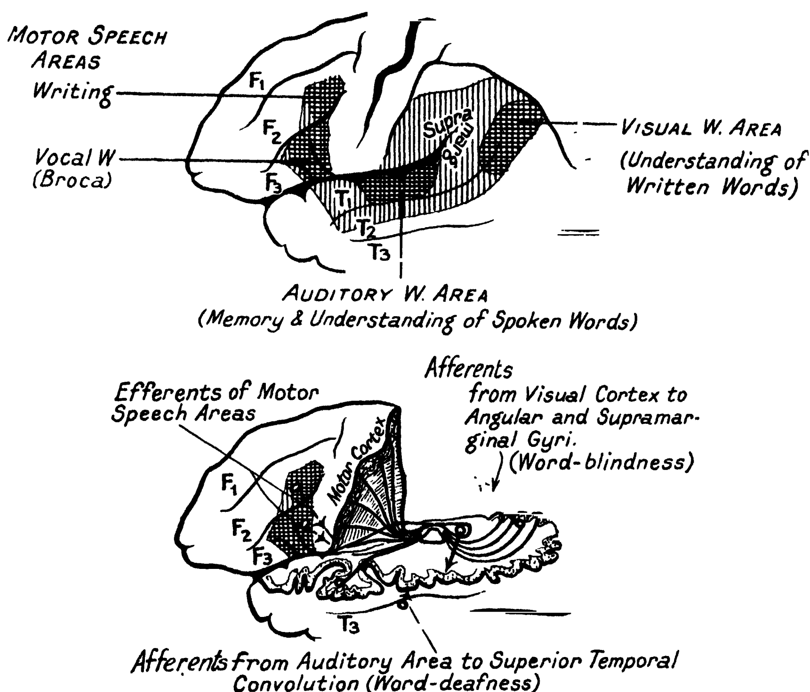


FIG. 175.—The Speech areas and their chief connections.

Applied Physiology of Aphasia.

The acquisition of speech in infancy is as follows: Like all motor processes speech is originated by afferent impressions and is sense-guided and governed. About the thirteenth month, gesture, in the form of shaking the head from side to side as a sign of negation, appears. (In conditions which damage the speech functions in later life this symbol is the last to be destroyed.) Then certain sounds are recognised by their association with objects handled (*word-hearing*): thus an area for the memory of words begins to be developed in the superior temporal convolution of the left side, in right-handed children. By eighteen months the child has a small vocabulary of thirty to forty words. These words are produced by the use of motor tracts proceeding from the cortex of the insula and posterior part of the third left frontal convolution. Between the second and third years the child can understand what is said and make himself understood by others. The acquisition of *reading* and

writing comes later. First the letters are recognised by educating the word-seeing area in the left angular gyrus, and afterwards they can be reproduced by education of the area of cortex concerned in writing in the posterior part of the second left frontal convolution. Later, further communicating pathways between these cortical areas are opened up and the child begins to read aloud, write from dictation, copy writing, etc. In right-handed children the speech area is laid down in the cortex of the left cerebral hemisphere, but in left-handed individuals the speech area may be in the cortex of the right cerebral hemisphere.

The *speech area of the brain,* after infancy, comprises a U-shaped area of the cortex of the left hemisphere (see Fig. 175) including (1) the superior temporal convolution (word-hearing), (2) the angular and supramarginal gyri (word-seeing), (3) the posterior end of the third frontal convolution (vocal speech, Broca's area), and insula, and (4) the posterior end of the second frontal convolution (writing). Although different functions have been ascribed to different " centres " in this area, these " centres " are so intimately connected with each other by association fibres in the subcortical white matter that one rarely finds pure types of aphasia in practice. In lesions involving the anterior part of the speech area the resultant aphasia will be predominantly *motor,* in those involving the posterior part of the speech area it will be mainly *sensory.*

Three main sets of *Afferents* reach the speech area (Fig. 175) :

(1) Afferents from the calcarine visual cortex to the angular and supra-marginal gyri. Lesions of this pathway produce " word-blindness " or inability to appreciate written speech.

(2) Afferents from the temporal projection of auditory fibres to the superior temporal convolution. Lesions of this pathway produce " word-deafness " or inability to understand spoken speech.

(3) Afferents from the thalamus to the superior temporal convolution concerned with the sensations produced in the movements of articulation. Lesions of this pathway, and the preceding one, produce " jargon aphasia " from the patient's inability to control the correctness of his speech movements or the sound of his words. We must be very careful not to mistake for insanity the voluble jabbering of such a case.

The *Efferents* from the speech area proceed from the posterior part of the third frontal convolution (vocal speech) and from the posterior part of the second frontal convolution (written speech) to the left pyramidal tract. The fibres are connected through the corpus callosum with the contralateral pyramidal tract and cortex.

The Speech area is supplied by the *left middle cerebral artery,* the anterior branch of which supplies the posterior ends of the second and third frontal gyri and the insula. The posterior branch running in the Sylvian fissure supplies the remainder of the speech area, those parts concerned with the production of word-blindness and word-deafness. Lesions of the calcarine branch of the left *posterior cerebral artery* may cause word-blindness, either alone, if the lesion is confined to the cortex, or in combination with right homonymous hemianopia, if the lesion extends into the optic radiation.

Aphasia can result only from a lesion of the cortex and the immediate

subcortical white matter, never from lesions of the deeper parts of the corona radiata or the internal capsule.

Symptoms.—MOTOR APHASIA. The patient's vocabulary is greatly reduced and the speech may be "telegraphic," *e.g.*, "Bring—spectacles—table," or the vocabulary may consist of a few recurrent utterances. "Oh my!" "Thank you!" In NOMINAL APHASIA the patient can recognise an object held before him and describe or demonstrate its use, but he cannot remember what it is called. Shown a penny he will say, "That is what you buy a stamp with," but he cannot name it. Suggest to him that it is a haddock and he will shake his head or become angry. Write on paper, or say "Penny," and he will nod and smile. The vocabulary of other languages known to the patient may be affected similarly in greater or less degree. Under the stress of great emotion a patient with a motor aphasia may become capable of coherent utterances impossible in his normal condition.

TABLE XLIX.—MOTOR APHASIA, *i.e.*, defect in the cortical motor mechanism of the areas for speech and writing.

	Tests.	*Nature of Defect.*	*Position of Lesion in Cortex Cerebri.*
Speech loss (Nominal aphasia).	Great defects in vocabulary, and cannot name objects.	Loss of educated recollection of the movements required for spoken words.	Posterior end of third left frontal (Broca's) convolution and lower end of ascending frontal.
Writing loss (Agraphia).	Cannot convey thoughts in writing, or write names of objects.	Loss of the educated recollection of the movements required for writing.	Posterior end of second left frontal convolution.

Together with these defects there is often associated a similar difficulty in writing—AGRAPHIA—proportional to the defect of speech, when all other movements of the hand and fingers are intact. Isolated agraphia does not occur. Patients with motor aphasia may stammer or perseverate, *i.e.*, tend to repeat a word or series of words already pronounced. Perseveration may be seen in aphasic writing, the same word being written over and over again.

Sensory Aphasia.—In WORD-DEAFNESS the patient is not deaf in the ordinary sense; he hears ordinary sounds and noises, but spoken words are not understood and sound to him like an unknown language. Not only this, but the activation of the executive mechanism is disordered, and, when he speaks, his words are mutilated and his word-deafness renders him unaware of his own errors. He uses wrong words, mixes up words in a sentence, or may jabber an unintelligible jargon—this is known as "Jargon Aphasia" or Syntactical aphasia, and results from lesions of the cortex of the superior temporal convolution. A partially word-deaf patient may use one word when he means another, *e.g.*, he may ask for the sugar when he means the salt (paraphasia).

TABLE L.—SENSORY APHASIA, *i.e.*, defect in the cortical receptive areas for word-vision or word-hearing.

	Tests.	*Nature of Defect.*	*Position of Lesion in Cortex Cerebri.*
Word-blindness (Visual Aphasia).	Can see but cannot read or recognise *printed* or *written* characters. Usually with right homonymous hemianopia.	Loss of the educated visual memory for written or printed signs.	Left angular and supramarginal gyri, or just beneath these.
Word-deafness (Auditory Aphasia).	Can hear but cannot understand or recognise *spoken* words.	Loss of the educated auditory memory for speech.	Left superior temporal convolution or just beneath this.

In WORD-BLINDNESS (Alexia) the patient can see, but the printed characters convey nothing to him. The lesion, in such cases, is in the neighbourhood of the angular gyrus, and, from its situation, the fibres of the optic radiation (see Fig. 175) are necessarily interrupted in some degree. A pure word-blindness is but rarely met with ; usually the lesion extends deeper than the cortex and there is an associated right homonymous hemianopia.

In Children word blindness is rare, but may be found in one or more members of a family. The *symptoms* are not unlike those of damage to the angular gyrus in later life. *Treat* by the cultivation of analytic and later synthetic vision.

INTELLECTUAL IMPAIRMENT.—Severe aphasia always produces some degree of mental defect. It is usually associated with widespread death of the cortex and severe right hemiplegia. The degree of intellectual impairment depends on the type of the aphasia, the greatest defects being encountered in cases of word-deafness, when there is confusion of speech intelligence (Semantic aphasia). The attention, in such cases, is difficult to hold and the patient becomes quickly tired and bored.

CLINICAL INVESTIGATION.

Confronted with a case which we recognise as aphasic, we must make some analysis to determine whether the aphasia is mainly motor or sensory. We determine what pathways remain open to the patient to express or receive speech. This is done by means of the following clinical tests. In applying them we should remember that most of these patients retain the capacity for comprehending gesture, and by no movement or glance should we indicate to the patient what is required of him. *We must be certain that we know accurately the condition of the patient's vision and hearing. We should know whether the patient is right-handed or left-handed :*

A. *Tests for Motor Aphasia.*
(1) Encourage the patient to talk spontaneously.
(2) Show him objects, *e.g.*, knife, key, penny, and ask him to name them.
(3) Ask the patient to write spontaneously.

B. *Tests for Sensory Aphasia.*
(4) Give a verbal command—*e.g.*, " Hold up your hand ! " " Shut your eyes ! " Can he understand what he hears ? (Word-deafness.)
(5) Give a written command—*e.g.*, Print on a card " Put out your tongue ! " Show it to the patient and await the response. Can he understand what he sees on the card ? (Word-blindness.)

C. *Further Tests.*

(6) Ask the patient to repeat complicated words after you—*e.g.*, "Ophthalmoscope."

Aud. ⟶ Motor.

(7) Ask him to copy a phrase which you write on a card.

Vis. ⟶ Writing.

(8) Ask him to write to your dictation.

Aud. ⟶ Vis. ⟶ Writing.

(9) Ask him to read aloud a passage and tell you the sense of it.

Vis. ⟶ Aud. ⟶ Motor.

Head's Terminology.[1]—(1) Verbal Aphasia—difficulty in word formation. (2) Nominal Aphasia—incorrect use of names and want of comprehension of nominal value of words. (3) Syntactical Aphasia—"Jargon Aphasia." (4) Semantic Aphasia —lack of appreciation of the deeper significance of words and phrases.

Etiology.—Lesions of any nature, involving the speech area, will produce these defects. (1) The commonest cause when the aphasia is of *sudden onset* is thrombosis of the cortical branches of an atheromatous left middle cerebral artery. (2) Embolism is a less frequent sudden cause. (3) Cerebral tumour, and (4) cerebral abscess produce aphasia of *gradual onset*, and it is in these cases, where the lesion undercuts the temporal cortex, that we meet with "word-deafness" and "jargon aphasia." (5) Subdural hæmatoma will produce an aphasia of gradual onset. (6) Head injuries, even simple cerebral contusions, may produce aphasia, usually transient. *Transient aphasia* may also occur in (1) migraine, (2) epilepsy, (3) cerebral arterio-sclerosis, (4) "congestive attacks" of General Paralysis of the Insane, and (5) uræmia.

Prognosis.—The prognosis largely depends on the cause of the aphasia. In vascular cases never make any definite statement until some time has elapsed after the occurrence of the lesion. Although recovery may occur there is danger of future recurrence. In children below the age of six years it is possible for speech formation to be transferred to the opposite hemisphere, provided sufficient intelligence remains from the effects of the lesion producing the aphasia.

Treatment.—When the aphasia is due to pressure on the cortical area by tumour, subdural hæmatoma or abscess, or other lesion of a removable character, it may be possible, by surgical operation, to afford the patient relief. In other cases, treatment should be directed to re-education of the patient on the lines used for defective children. The best results are obtained with children under the age of six years.

§ **744. Congenital Aphasia.**—Of the two main varieties described, *Congenital Word-Blindness* (*Dyslexia*) is commoner. The defect may run in families and is more frequently found in male children. It is first noticed when an attempt is made to teach the child to read. Although he can speak intelligently and fluently and repeat sentences by heart, the child has difficulty in learning the alphabet and cannot be taught to read at sight. In *Congenital Word-Deafness* the child may, owing to the difficulty in understanding spoken words, be regarded as imbecile. Talk, if attempted, is simply babbling. Signs of hearing are present, and the child learns the use of common

[1] *Brain*, Vol. XLIII, p. ii, 1920.

objects by visual perception, has normal habits and may be abie to draw objects accurately but cannot name them. With suitable education, word-deaf children may grow up to be mentally normal.

II. *The patient is unable to carry out some purposive movement when requested to do so, although there is no actual paralysis.* The condition is APRAXIA. Apraxia may be *Motor* or *Sensory.*

§745. **Motor Apraxia.**—By motor apraxia we mean that the patient is unable to carry out some purposive movement, when requested to do so, although he understands the significance of the movement and is not paralysed. He knows what he wants to do but cannot do it. We test for apraxia by asking the patient to make a military salute, first with one hand and then with the other. Although the affected hand is not paralysed, and the patient understands and can explain what is wanted of him, only aimless wandering movements are made. Other useful tests are—asking the patient to strike a match, cut a piece of paper with scissors, or use a pencil, etc.

Sensory Apraxia (Agnosia) is due, for the most part, to imperception, *i.e.*, failure to appreciate the nature of objects and their uses. If shown a key and asked to use it, he may put it in his mouth and try to smoke it like a cigarette.

Motor Apraxia results from a lesion of the posterior part of the left pre-frontal area or the genu of the corpus callosum: *Sensory Apraxia* results from bilateral lesions in the parietal regions.

A patient with apraxia and agnosia sometimes shows *perseveration.* Asked to salute, he may continue to respond to all other requests by saluting during the next few minutes.

In right-handed people the left hemisphere is the dominant one, and through the commissural fibres of the *corpus callosum* exercise a controlling influence over the contralateral hemisphere. Clinically, apraxia is commonly left-sided, even with lesions of the left hemisphere. It is met with in thrombotic lesions of the anterior cerebral artery, in cerebral tumours and abscesses and in bilaterally situated chronic subdural hæmatomata. It may be observed, also, in diffuse diseases of the cortex, General Paralysis of the Insane and cerebral arterio-sclerosis.

III. *There is defective movement of the lips, tongue and larynx, etc., so that the patient pronounces his words badly.* The condition is DYSARTHRIA.

§ 746. **Dysarthria** is a defect of the peripheral neuromuscular mechanisms of the larynx, lips, tongue, palate or pharynx. The patient "cannot get his tongue round words." You test for it by observing the patient's conversation or by asking him to repeat "test sentences." The lesion, as in other motor defects, may be in the upper motor neurone, cerebellum, striatal pathways, in the bulbar nuclei, the lower motor neurone, or the muscles themselves. Disorders of movement, exactly similar to those seen in the limbs, are observed in the muscles of the speech organs. One meets with spastic paralysis, tremor, rigidity, involuntary movements, inco-ordination and flaccid paralysis. According to the type of motor disorder present, the dysarthria is described as spastic, flaccid, ataxic, and so forth.

1. *Slow slurring and indistinct articulation* (Spastic Dysarthria) is seen in **Pseudo-Bulbar Paralysis,** where there is spasticity and weakness of the muscles concerned in articulation. There is usually a history of repeated slight "strokes" and the patient presents an expressionless, mask-like facies, with dribbling of saliva which the spastic lips cannot retain in the mouth, difficulty in swallowing, coughing, etc., but without the atrophy and fibrillation of the tongue seen in Chronic Bulbar Paralysis. The pseudo-bulbar patient weeps uncontrollably, with a curious spastic wail when he attempts to talk, or, less frequently, he laughs uncontrollably when he talks. It is caused by double hemiplegia or *thromboses* affecting both pyramidal tracts. The pyramidal signs are usually slight and, in most cases, considerable mental deterioration is present. The lesions are found bilaterally in the internal capsule, near the thalamus, but in almost all cases cortical softenings are found.

2. In *tremulous articulation* are seen coarse tremors of the lips, lower facial muscles and tongue, and the articulation is slurred and syllables jumbled. It is due to cortical disease and is met with in *Alcoholic Intoxication* or poisoning of the cortex from narcotic drugs, *e.g.*, after large doses of bromide. Its presence is often the clue to the diagnosis of *General Paralysis of the Insane*; when a patient presents himself in your consulting-room with a shaking voice and tremor of the lips and tongue, you should always carefully examine the pupils and question the relatives for a history of undue irritability, mistakes made at business, loss of interest in outside affairs, or other character changes. In this type of dysarthria, from the disease of the cortex which causes it, syllables are often jumbled or misplaced, or wrong words are used. The paretic makes the same mistake in spelling as he does in articulation. In *Psychoneurotic Anxiety States* and *Hyperthyroidism*, the articulation is occasionally tremulous, but syllables are normally placed and the patient's writing shows no mistakes in spelling, or omissions.

3. A *thin, monotonous voice,* which tails away at the end of sentences, is produced by the rigidity of striatal disease, either *Parkinsonism* following *Encephalitis Lethargica* or *Paralysis Agitans.* The characteristic facies, and bodily attitude and gait, and "cog-wheel" rigidity of the wrist-joints will be present.

4. *Jerking or gasping* articulation occurs in severe *Chorea* from involuntary movements of the respiratory, tongue and facial muscles. In *Bilateral Congenital Chorea* or *Athetosis,* the grimaces of the face and the rolling movements of the tongue cause the patients to "chew their words" so that articulation may be almost unintelligible.

5. "*Scanning Speech*" (Ataxic Dysarthria) in which syllables are separated staccato-fashion or elcided (slurred), occurs in lesions of the cerebellum or its medullary connections. It is found in *Disseminated Sclerosis* and is a very characteristic finding in *Friedreich's Ataxia* combined with absence of tendon reflexes, extensor plantar responses, impairment of sense of position and pes cavus. It is found in tumours and abscesses, vascular lesions and degenerations of the cerebellum, especially when these involve the vermis.

6. *Indistinct articulation* may result from flaccid paralysis of the lips, palate or tongue (Flaccid dysarthria). In *Bell's Palsy* articulation may be indistinct. After diphtheria the *palate* may be paralysed, so that B sounds are produced like M, and there may be nasal regurgitation of fluids. *Myasthenia gravis* may affect the lips, tongue or palate, producing a flaccid palsy, varying in degree from time to time and accentuated by fatigue. In certain types of *Myopathy* the facial muscles are affected, with clumsiness of articulation. If the *hypoglossal nerve* is divided during operations on the neck or throat, a clumsiness of articulation develops, which the patient, by practice, soon learns to overcome.

The most characteristic type of flaccid dysarthria, however, is found in—

§ **747. Chronic Bulbar Paralysis,** a variety of Motor Neurone Disease (§ 788). The speech is indistinct at first, and, on examination, the tongue is seen to be shrunken, wrinkled and fibrillating. Later, the laryngeal muscles, the muscles of the floor of the mouth, the palate and the lips are affected, saliva drips from the mouth, fluids regurgitate from the nose and, with involvement of the pharyngeal muscles, swallowing

becomes difficult. The atrophy and fibrillation of the tongue may be combined with atrophy and fibrillation of the intrinsic hand muscles, exaggeration of tendon reflexes and extensor plantar responses. The disease is usually fatal in three to four years, and death occurs from respiratory failure or aspiration pneumonia. The *cause* is a gradual degeneration of the motor cranial nerve nuclei, coming on, mostly in males, in adult life, frequently after the age of forty years. Occasionally, this disease is caused by *syphilis* and a Wassermann reaction should always be done to exclude this; even the syphilitic cases, however, do not improve and may advance in spite of treatment.

Treatment consists of careful feeding of the patient with semi-solid foods such as gruels, porridge, mashed vegetables and pounded meats and fish, made into semi-solid consistency. Salivation may be helped by giving tincture of belladonna, ♏ 10–15, thrice daily by the mouth, or hyoscine hydrobromide gr. 1/100 once or twice a day by mouth. These patients should not be kept in bed until this is absolutely necessary.

§ **748. Disorders of Articulation** are very common, especially in children. Perfectly normal children may make no attempt to talk until the third year of life. When the child uses baby talk until the age of five years or later, the cause may be sought in the parents who wish the child to preserve attractive baby-like ways. A severe illness in childhood may retard speaking.

Congenital aphasia is described in § 744.

Stammering and **Tics of Articulation.**—There are various spasmodic defects of articulation leading to a sudden check in the utterance; of these stammering is the commonest. The affection is not congenital; it appears during the self-conscious years of childhood, usually in boys. Often a family history of the complaint is ascertained. The patient sticks over the *consonants,* particularly explosives and labials, B, D, P, T, K, or G. The consonant is repeated, " P—P-P-Please ! " In a rarer minority of cases the patient sticks over the *vowels*, the mouth remaining open and the face contorted. After the difficulty is overcome the words may appear with a rush. The trouble is accentuated in conditions of excitement. Stammerers can sing normally and whisper normally, and the defect disappears when the patient is talking to himself. Stammering is due to want of co-ordination between the respiratory, laryngeal and labio-lingual components of articulation. It is common in left-handed children and may apparently be provoked by teaching left-handed children to write with the right hand.

Treatment is by (*a*) psychotherapy and (*b*) daily speaking exercises. It is most important to endeavour to correct errors in the environment. The child's relationship to the parents, brothers and sisters and school-teachers should be ascertained, and the co-operation of all these individuals enlisted. Speech training classes are now run in connection with most neurological clinics. The patient is directed to fill his chest with air, speak slowly with a resonant voice, and when he comes to a word on which he sticks, he is to sing it, accentuating the second part of the word with a raised voice. These daily exercises should first be

performed alone and, later, with an audience. Breathing and singing exercises and gymnastics or dancing may be used in treatment.

In **Palilalia**, a rare speech disorder, a phrase is reiterated with increasing rapidity, It is met with in Pseudo-Bulbar Palsy, § 746, and Post-Encephalitic Parkinsonism, § 767.

Lalling, or infantile speech, is that in which the letters difficult to pronounce —*e.g.*, R, L, C, Sh—are avoided; British is pronounced Bitty. It persists only when the child is mentally retarded.

In **Lisping** there is defective enunciation of certain consonants, *e.g.*, S becomes Th, Th becomes V. It is due to faulty articulation and occurs normally in infants learning to speak. In adult life it is due to bad habit of articulation or defective conformation of the mouth. Speech training, with an expert, can remove it in most cases.

In **Idioglossia**, the child has a speech of its own, which is unintelligible except to those accustomed to the child. It is due to an inability to reproduce the sounds of words said to him. It may be a variety of *Congenital Aphasia* (see § 744). Proper speech training may improve the condition where the intelligence of the child is normal.

IV. *Articulation is absent* (MUTISM) *or reduced to a whisper* (APHONIA).

Pure Mutism is invariably an hysterical affair. The aphasic patient is never completely wordless.

§ **749. Deaf-Mutism** is due to congenital peripheral stone-deafness which prevents the child learning to talk. No sound is heard, and induced vertigo is absent on galvanic labyrinthine stimulation (passing a current of 1 to 2 milliampères through the two mastoids). Deaf-mutes are often bright and intelligent and have a wonderful command of gesture.

§ **750. Aphonia** is concerned with the laryngeal muscles; the voice becomes a whisper but articulation is preserved. It is met with in laryngitis, in recurrent laryngeal paralysis from *thoracic aneurysm, mediastinal new growth*, or in *tabes dorsalis*. In *hysterical aphonia* the patient speaks in a scarcely audible whisper. The onset is usually sudden, after an emotional shock, and, when the patient is asked to cough, phonation occurs in a startlingly paradoxical fashion. Laryngoscopic examination shows that the cords are not sufficiently approximated to produce a sound on attempted phonation (see Figs. 60, 61, §§ 176, 888).

THE SYMPTOMS BELONG TO THE MOTOR SYSTEM

GROUP V. SPASTIC PARALYSIS

Persistent increase of muscular tonus may result from (1) Disease of the Pyramidal Tracts ("Clasp-knife" rigidity), (2) Disease of the Extra-Pyramidal Motor Tracts ("Cog-wheel" or "Lead-pipe" rigidity), or (3) Hysteria (Hysterical rigidity).

(1) "*Clasp-knife*" (*Pyramidal*) *Rigidity* is seen in *Residual Hemiplegia*. When you attempt to extend the patient's arm at the elbow, the resistance at first encountered increases up to a point and suddenly gives way, like the feeling met with in shutting or opening the blade of a pen-knife.

(2) "*Cog-wheel*" or "*Lead-pipe*" (*Extra-Pyramidal*) *Rigidity* is seen in *Parkinsonism*.—When you attempt to flex and extend an affected limb (say the hand at the wrist-joint) the resulting resistance is jerky but uniform, with none of the spring noted in Pyramidal Rigidity. The feeling is like that felt in moving a cog-wheel. Sometimes the "cog-wheel" element is absent and the resistance is so uniform that attempts to bend the limb passively feel like bending a piece of lead-piping.

(3) *Hysterical Rigidity* is seen in *Hysterical Monoplegia*.—When you attempt to move the patient's rigid limb, the resistance encountered increases with the amount of force used.

Lesions of the Pyramidal and Extra-Pyramidal Motor System are further differentiated as follows:

TABLE LI.

Pyramidal Disease.	*Extra-Pyramidal Disease.*
1. Motor Paralysis.	1. No true motor paralysis apart from stiffness.
2. "Clasp-knife" rigidity.	2. "Cog-wheel" or "lead-pipe" rigidity.
3. Tendon reflexes increased with clonus.	3. Tendon reflexes normal. No clonus.
4. Abdominal reflexes diminished or absent.	4. Abdominal reflexes normal.
5. Extensor plantar response.	5. Flexor plantar response.

Muscular Contractures.—In the later stages of pyramidal disease, an organic rigidity with degenerative changes followed by fibrosis, takes place in the muscles themselves causing diminution in the normal extensibility of muscles and fixation of the joints. Contractures may also occur in lower motor neurone lesions, *e.g.*, in the facial muscles after severe Bell's palsy (§ 859) and in muscles after an attack of acute poliomyelitis (§ 732).

§ **751. Spastic Paralysis** is the clinical term used for motor paralysis with "clasp-knife" rigidity. It is pathognomonic of lesions of the upper motor neurone. Lesser degrees of paralysis are termed paresis. The distribution of the paralysis or paresis varies.

(I) Spastic paralysis of one side of the body, including the face, trunk and limbs, is termed SPASTIC HEMIPLEGIA.

(II) Spastic paralysis of one limb is termed SPASTIC MONOPLEGIA.

(III) Spastic paralysis, if bilateral and affecting the limbs alone, is termed SPASTIC PARAPLEGIA. Usually both legs are affected, but the arms may be also involved.

(IV) Spastic paralysis of all four limbs, including the face, is termed SPASTIC DIPLEGIA.

Group V (I). *There is spastic paralysis of one side of the body, including the face, trunk, arm and leg. The condition is* SPASTIC HEMIPLEGIA, *resulting from a unilateral brain lesion.*

§ **752. Spastic Hemiplegia.**—*Symptoms.*—(*a*) *Acute Stage.*—If the cerebral lesion responsible be *acute*, the paralysis of the contralateral lower face, arm and leg will at first be *flaccid*. The plantar response, however, is extensor from the first, and the abdominal reflexes, upper and lower, are absent on the paralysed side. In the slightest cases there is weakness of dorsiflexion of the wrist, paralysis of fine finger movements and of the extensors of the fingers and elbow joint. In the lower limb there is weakness of flexion of the knee and inability to dorsiflex the foot. Within

a day or two the tendon reflexes become increased in the affected limbs, and in a week or two, with recovery of power, the characteristic *spasticity* appears. (*b*) *Stage of Recovery.*—In *chronic* progressive lesions and in the stage of recovery of acute lesions there is : (1) Spasticity of the affected limbs, of the " clasp-knife " variety. (2) The upper limb is held with the arm adducted at the shoulder, and flexed at the elbow, wrist and fingers, with the forearm slightly pronated. (3) The lower limb is extended at the hip and knee-joints, and (4) the gait is characteristic, the paralysed leg being dragged round in a semicircle, the toes scraping the floor. (5) There is weakness of the affected side of the face in smiling, and food collects round the teeth on the paralysed side, but the patient can screw up both eyes normally. (6) The tongue is protruded to the affected side, owing to the unbalanced action of the genio-hyoglossus. (7) There may be also certain involuntary " associated movements " in the affected limbs. Thus, if the patient yawns, he may extend his paralysed wrist and fingers and raise his hand in front of his face, performing, involuntarily, movements which he cannot voluntarily achieve.

Power returns first, with hypertonus, to the *flexor* muscles of the upper limb, in accordance with the physiological principle that primitive function (in this case prehension) is the last to be destroyed and the first to recover. Thus the patient may soon be able to elevate and abduct the affected upper limb at the shoulder, and to flex the elbow, fingers and wrist. In the lower limb, power returns first to the *extensors* of the hip and knee, and to the adductors (maintenance of erect posture). Power may return almost completely, with the exception of ability to dorsiflex the foot and perform fine finger movements. The upper abdominal reflex returns before the lower, the plantar reflex may become flexor. The recovery of aphasia, if present, precedes recovery of paralysis.

In addition, other than purely motor symptoms may be present. 1. *Aphasia*, in right-handed people, if the lesion is in the left hemisphere. 2. *Hemianæsthesia* may be present if the posterior part of the internal capsule is affected. 3. *Hemianopia* occurs if the lesion is still farther back in the internal capsule. 4. *Mental impairment*, apart from aphasia, often results in slight degree from a "stroke." 5. *Involuntary movements* are apt to appear in hemiplegia occurring in childhood; they take the form of athetosis or chorea. 6. *Pain* may occur in the hemiplegic limbs if the lesion involves the thalamus—" hemiplegia dolorosa." This pain is almost impossible to relieve. 7. *Crossed paralysis*, with an ipsilateral cranial nerve palsy and crossed hemiplegia, occurs in lesions of the crus, pons or medulla (see § 670).

(*c*) *Residual Hemiplegia.*—In some cases the recovery is only partial; the affected limbs remain spastic and weak, and the deep reflexes increased. The paralysed limbs are blue and cold, and fibrosis occurs in the muscles, especially of the upper limb, with contractures at the shoulder and elbow, and arthritic changes. The muscles show no wasting, apart from that due to disuse.

§ 753. **Hysterical Hemiplegia** is rare and is distinguished by: (1) sudden onset after emotional shock; (2) the rigidity, if present, is of the *hysterical*, not the " clasp-knife " variety (§ 750); (3) there is no alteration in the tendon reflexes or abdominal reflexes; (4) the plantar response is absent or of the flexor type. (5) Spurious ankle-clonus may occur in hysterical hemiplegia and is rarely sustained, as in organic hemiplegia. (6) In walking, the toes do not scrape the ground in hysteria, but are dragged behind the patient. Early slight hemiplegic weakness in *Disseminated Sclerosis* is often diagnosed as hysteria, but, in these cases, the tendon reflexes are increased on the side affected and other early signs of disseminated lesions may be present (§ 755). In all cases the optic discs should be examined for early temporal pallor, nystagmus should be looked for, the abdominal reflexes tested for diminution or ready fatigue on the affected side, and vibration tested over both tibial malleoli.

The *Causes of Hemiplegia* can be divided into two groups according to the mode of onset.

(A) HEMIPLEGIA OF SUDDEN ONSET.

I. Cerebral Thrombosis.
II. Syphilitic Thrombosis.
III. Cerebral Hæmorrhage (apoplexy). } Onset instantaneous.
IV. Cerebral Embolism. }
V. Congestive Attacks in General Paralysis of the Insane.
VI. Post-Epileptic Hemiplegia.
VII. Middle Meningeal Hæmorrhage.

In III and IV the onset is *instantaneous*, in the others it occurs over a period of minutes or hours.

(B) HEMIPLEGIA OF GRADUAL ONSET.

VIII. Cerebral Tumour. }
IX. Chronic Subdural Hæmatoma. } Onset over a period of weeks or months.
X. Cerebral Abscess. }
XI. Meningitis, Acute or Chronic. } Onset over a period of days.
XII. Acute Encephalitis. }

(A) **Sudden Hemiplegia.**—*The hemiplegia comes on within a few seconds, minutes, or hours.*

By far the commonest cause of *sudden* hemiplegia is CEREBRAL THROMBOSIS from atheroma or syphilitic endarteritis of the middle cerebral artery. SYPHILITIC hemiplegia occurs, commonly, before the age of forty years; ATHEROMATOUS lesions are common after that age.

I. CEREBRAL THROMBOSIS (§ 714).—(1) The onset occurs over minutes or hours with relatively slight disturbance of consciousness. (2) Thrombosis develops in conditions of enfeebled circulation, *e.g.*, during sleep, after exhaustion, whether the patient has high blood pressure or not. (3) Blood is absent from the spinal fluid. (4) The patient is middle-aged or advanced in years, except in syphilitic cases (see § 94).

II. SYPHILITIC THROMBOSIS.—(1) Due to syphilitic endarteritis, is a common cause of sudden hemiplegia in persons under the age of forty years. (2) In about half the cases the Wassermann reaction is positive

in the spinal fluid, in the other half it is negative in the spinal fluid and positive in the blood.

III. CEREBRAL HÆMORRHAGE (APOPLEXY, § 712) occurs from rupture of a vessel (commonly the lenticulo-striate branch of the middle cerebral) into the external or internal capsule. It may take place into a malignant and vascular glioma, proving rapidly fatal; and it may be a terminal incident in acute leukæmia and other severe anæmias. (1) The onset is instantaneous, with coma or convulsions, but apoplexy never causes sudden death. (2) Death occurs some hours after the onset of the symptoms. (3) Hæmorrhage is a likely cause of apoplexy in alcoholic subjects during severe physical strain or a bout of rage, or in the subjects of chronic interstitial nephritis with high blood pressure and retinal hæmorrhages. (4) The finding of blood in the cerebro-spinal fluid on lumbar puncture gives proof of cerebral hæmorrhage. (5) The patient is advanced in years, often alcoholic.

IV. CEREBRAL EMBOLISM (§ 713).—(1) The onset is instantaneous with coma or convulsions. (2) Embolism should be diagnosed in the presence of mitral stenosis, auricular fibrillation, aortic regurgitation, aneurysm, malignant endocarditis, puerperal sepsis, or in the presence of a recently fractured long bone (fat embolism). (3) Embolism occurs at any age, but the patient is commonly young.

V. CONGESTIVE ATTACKS IN G.P.I.—(1) The onset is sudden with convulsions or coma. (2) The hemiplegia recovers rapidly and perhaps completely; much more rapidly than in any other comparable cerebral lesion. (3) The characteristic spinal fluid findings of the disease (§ 902) are present in all cases.

VI. POST-EPILEPTIC HEMIPLEGIA.—The history of recurring attacks, the complete rapid recovery of the patient are characteristic.

VII. DELAYED TRAUMATIC APOPLEXY (MIDDLE MENINGEAL HÆMORRHAGE) is described in § 715. III. *Intra-cerebral Hæmorrhage* may also follow a head injury, the symptoms being instantaneous in onset. *Subacute Subdural Hæmatoma* produces increasing stupor, focal convulsions and monoplegia or hemiplegia, with blood in the spinal fluid.

(B) **Hemiplegia of Gradual Onset**.—*The hemiplegia comes on over a period of days, weeks or months.* Owing to the frequency with which it occurs, INTRACRANIAL TUMOUR should, in all cases, be suspected.

VIII. INTRACRANIAL TUMOUR is described in §§ 828 *et seq.*—(1) The *progressive* character of the hemiplegia, coming on insidiously over weeks or months, suggests tumour. The hemiplegia is sudden in onset only when a hæmorrhage into the growth or sudden cerebral œdema occurs, but symptoms have usually been present in the affected limbs before this appears. (2) The occurrence of *Jacksonian fits* during the slow progress of the hemiplegia is strong evidence of neoplasm. These symptoms are sufficient to warrant the diagnosis. Usually, however, general symptoms of increasing intracranial pressure are present, viz.: (3) *Headache* and *vomiting*, with or without giddiness or progressive apathy. In the absence of arterio-sclerosis these symptoms point to tumour. (4) *Papillœdema* is very frequent in cerebellar and temporal lobe tumours, but may be absent in tumours of the pons or subcortical tumours.

It rarely causes failure of vision even when marked; its presence has to be looked for. Intense papillœdema is not met with in any other condition, but lesser degrees with hæmorrhages and exudate may be seen in arterio-sclerosis and chronic nephritis with high blood pressure. These latter conditions, however, rarely cause a slowly *progressive* hemiplegia.

IX. Chronic Subdural Hæmatoma (see § 827).

X. Cerebral Abscess (see § 737) may produce hemiplegia. A septic focus is present in the middle ear, nasal accessory sinuses, lungs or elsewhere.

XI. Meningitis, Acute or Chronic (§ 726).—Tuberculous or other varieties of meningitis cause hemiplegia, usually of slight degree. Signs of meningitis in the form of head-retraction, intense headache and photophobia and Kernig's Sign, will be present and the spinal fluid will show pleocytosis. In *subarachnoid hæmorrhage* the signs are those of meningitis but the spinal fluid is pink from laked blood. *Syphilitic leptomeningitis* is a rare cause of hemiplegia. Usually hemiplegia in syphilitic subjects is due to thrombosis of a cerebral vessel affected by syphilitic endarteritis. Localised *gumma* of the brain is excessively rare: amongst hundreds of intracranial tumours the writer has seen only two proved cases.

XII. Acute Encephalitis (§ 740)—In children, after a specific fever or vaccination, this disease rarely causes hemiplegia. A cerebral form of poliomyelitis is described. Hemiplegia occurs occasionally in acute encephalitis lethargica.

Treatment of Hemiplegia.—See §§ 714, 716.

§ **754. Infantile Hemiplegia** may be pre-natal or post-natal; the onset is usually acute.

Symptoms.—(1) The onset, in post-natal cases, is with generalised or focal convulsions and coma, lasting perhaps twenty-four hours, and (2) fever, followed by (3) residual hemiparesis of varying degree. (4) The residual hemiparesis and aphasia, if present, clear up either completely or improve up to a point. (5) Athetosis (see § 771) in the hemiplegic limbs is a fairly common sequel; and (6) arrest of growth on the affected side, (7) epilepsy or (8) mental defect, may remain. (9) In pre-natal cases the skull may be asymmetrical.

Causes.—The pre-natal cases are due to atrophic sclerosis of the cerebral convolutions. Post-natal cases are much commoner and result from compression or tentorial tears at birth. Occurring *in the early years of childhood*, hemiplegia is commonly due to a localised focus of encephalitis following poliomyelitis or the acute specific fevers, *e.g.*, measles, mumps, chicken-pox or whooping-cough, or to any of the causes of adult hemiplegia.

Treatment is as for adult hemiplegia. In most of the cases the children are intelligent and may be educated at a school for physical defectives, or a small private school. It is unwise to force the child into competing with normal children.

Group V (II). *There is spastic paralysis of one limb. The condition is* Spastic Monoplegia *and it results from a focal* (1) *Cortical Lesion,* (2) *Spinal Lesion or, occasionally, from* (3) *Hysteria. In organic cases affecting the arm, the lower face is sometimes also affected*—Facio-brachial Monoplegia. *If the lower limb is the one affected, the condition is termed* Crural Monoplegia.

Spastic Monoplegia may be of (A) Gradual or (B) Sudden onset, or it may be due to (C) Hysteria.

(A). **Spastic Monoplegia of Gradual Onset** occurs in CEREBRAL TUMOUR, ABSCESS, or CHRONIC SUBDURAL HÆMATOMA, affecting the cortical motor areas, and producing crural, brachial or facio-brachial monoplegia. Such cortical motor paralysis is commonly associated with Jacksonian fits (§ 723), because the disease may irritate as well as paralyse the cortex. The monoplegic limb frequently shows cortical anæsthesia, astereognosis, atopognosis and loss of two-point discrimination (see § 674). Similar monoplegia, with cortical blindness, may be observed in children of eight to ten years with Schilder's ENCEPHALITIS PERIAXIALIS DIFFUSA.

(B). **Monoplegia of Sudden Onset** may be due to a vascular cortical lesion, *e.g.*, thrombosis or embolism of the anterior cerebral artery. The monoplegia produced is in the leg and is associated with left-sided motor apraxia (see §§ 685, 745). *Spinal* monoplegia due to a focal lesion of the pyramidal tract, producing spastic weakness of an upper or lower limb, is differentiated from cortical monoplegia by (1) the absence of Jacksonian attacks, (2) the associated anæsthesia is of the spinal-cord type, affecting the affective forms of sensibility (pain, temperature, touch) rather than the cortical discriminative forms. Monoplegia of sudden onset, either spinal or cortical, is particularly common in *Disseminated Sclerosis.*

(C). **Hysterical Monoplegia** may develop suddenly or gradually. It is distinguished by (1) the variability of the paralysis, (2) the stocking or glove anæsthesia, (3) the abnormal position of the limb, and (4) the absence of increased tendon reflexes or pyramidal signs. The early monoplegic weakness of *Disseminated Sclerosis* is frequently mistaken for hysteria (§ 888).

A YOUNG ADULT *comes to you saying: "My* LEG *has suddenly* BEGUN TO DRAG," *or "My* ARM *has suddenly* BECOME USELESS"*; there may have been* DOUBLE VISION *or* PRECIPITANCY OF MICTURITION. *The disease is probably* DISSEMINATED SCLEROSIS.

§ **755. Disseminated Sclerosis.**—Other initial complaints are common: (1) sudden onset of double vision, "I can see two of everything, doctor"; (2) sudden transient unilateral blindness (retrobulbar neuritis); (3) numbness, tingling, or pins and needles in a limb, and (4) difficulty in holding the water, precipitate micturition. As the essential characteristics of the lesions in this disease are (i.) their *acute development at irregular intervals* and (ii.) their *scattered distribution,* it will be realised that any conceivable type of neurological symptom or sign can occur in this disease, *e.g.*, (5) scanning speech, (6) intention tremor, (7) sensory ataxia, (8) cerebellar ataxia, (9) slight cranial nerve palsies, (10) fits, and even hemianopia. The speech is often characterised by slow separation of the syllables, like the scanning of prosody, with slurring of the articulation. The *intention tremor* merits special description; it appears on voluntary movement, not during the active phase of the movement, but when the object is about to be attained. Thus in the finger-nose test the finger is brought accurately to the neighbourhood of the nose, then violent oscillations appear before the nose is eventually touched. The ataxia is not increased by closing of the eyes. When the complaint is of "uselessness" in the limbs, sensory ataxia is often found to be present, with impairment of sense of position. Slight monoplegic weakness is often entirely missed through insufficient examination; "neuritis," writer's cramp, "rheumatism" or hysteria is diagnosed erroneously. But if you will look at such cases for one or other of the following signs you will not miss many cases

of disseminated sclerosis. (1) Examine the optic discs for pallor of their temporal halves. (2) Look for nystagmus. (3) Test vibration sensibility over the tibial malleoli; it is frequently diminished or absent. (4) Rapid tiring or diminution of abdominal reflexes, especially the lower, should always be looked for. The epigastric and upper abdominal reflexes tend to remain. (5) One or other plantar response may be extensor in type. Nystagmus, as an isolated sign, is of little use in diagnosis, but combined with any of the above signs it affords valuable evidence. The difficulties of diagnosis are often increased by the patient making light of the symptoms. In some cases the symptoms last for a few days or weeks and then clear up more or less completely with recurrences of further symptoms at later periods. In other cases the symptoms are slowly progressive from the start. Thus, two main types of the malady are recognised: (i.) The *Remitting Type*, and (ii.) the *Chronic Progressive Type*.

In early *remitting* cases the initial symptom is often acute in onset and severe in degree. It lasts, however, only a few weeks, then slowly clears up, perhaps with no residual physical signs. The recovery may be complete in early days, but subsequent lesions leave behind them residual weakness and physical signs.

In the *later stage of the disease* the gait becomes spastic and ataxic, and the patient holds on to the furniture in walking, or walks with one or two sticks, or is bedridden. Flexor spasms are added to ankle and rectus-clonus in the lower limbs and interfere with sleep. Precipitancy, even incontinence, of urine occur. Ataxia, either sensory or cerebellar, is manifested, with intention tremor, nystagmus, titubation (oscillation of the head) and scanning speech. Remissions cease to occur; finally, contractures of the flexor muscles set in with great loss of power, incontinence of urine and fæces, and bed-sores. The patient retains to the end the characteristic cheerfulness, lack of insight, and emotional unrestraint so characteristic of even the early stages of the disease. Occasionally psychoses with delusions or hallucinations occur. This lack of insight into the seriousness of the symptoms, combined with a certain jocularity or hilariousness, is a characteristic of the disease and is referred to as *Euphoria*. The patient laughs in unrestrained fashion when asked to touch the nose with the forefinger. Similar euphoria may exist in other chronic diseases, such as phthisis.

Diagnosis.—The clinical diagnosis is based on: (1) The acute development of symptoms at irregular intervals. (2) The presence of symptoms and signs of multiple lesions in the central nervous system. Multiple lesions also occur in *cerebro-spinal syphilis*, but this is excluded by serological examination of the blood and spinal fluid. Difficulty is likely to occur in early mono-symptomatic cases of four types:

(*a*) The patient, a young adult, complains of sudden unilateral, transient loss of vision and the signs of retrobulbar neuritis (see § 851) may be found if the lesion is near the nerve head. Retrobulbar neuritis rarely

occurs from sphenoidal or other nasal accessory sinus infection, but is extremely frequent in early disseminated sclerosis.

(*b*) The signs are those of a pure pyramidal lesion, either monoplegic or paraplegic in distribution. The disease may be *Motor Neurone Disease* or the *syphilitic spinal paralysis of Erb.* Examination of the spinal fluid may show the slight increase of globulin with paretic type of Lange curve and a negative Wassermann reaction, which is characteristic of disseminated sclerosis. The finding of muscular wasting or fibrillation, or bulbar paralysis, favours *Motor Neurone Disease*, while the serological reactions of blood or spinal fluid may indicate *syphilis.*

(*c*) There is a spastic paraplegia, with sensory loss, up to a segmental level. Yellow cerebro-spinal fluid or increased protein always indicates *spinal compression* in such cases.

(*d*) Diplopia, cerebellar symptoms and pyramidal signs, indicate a *focal lesion in the pons.* In pontine neoplasm, papillœdema is late and in disseminated sclerosis papillœdema is excessively rare and slight. The diagnosis in such cases can often only be settled by the ultimate progress of the case.

However protean the manifestations of the disease, certain symptoms are so rare that their presence in a case of supposed disseminated sclerosis warrants revision of your diagnosis. These are (1) *Pain*, which is rare in disseminated sclerosis apart from the muscular cramps and backache of the early stiffness and the paroxysmal pain associated with flexor spasms in the later stages of the disease. Pain suggests spinal compression. (2) *Muscular wasting* is excessively rare in disseminated sclerosis, but occurs in syringomyelia, motor neurone disease, and some spinal forms of syphilis. (3) *Absence of tendon reflexes* is excessively rare in disseminated sclerosis, whereas the knee and ankle-jerks are lost early in Friedreich's ataxia. Exaggeration of the tendon reflexes on one side is a highly important sign characteristic of disseminated sclerosis.

Cerebro-spinal Fluid.—In 50 per cent. of cases or more the spinal fluid shows some alteration in the Lange colloidal gold curve, which is usually of the " paretic " type. In many cases the globulin is slightly increased. Acute lesions, especially those near the ventricles, in the optic nerves, or on the surface of nervous structures in contact with the subarachnoid space, are accompanied by a slight mononuclear leucocytosis in the spinal fluid. The finding of a positive Wassermann reaction indicates that the disease is *cerebro-spinal syphilis,* not *disseminated sclerosis.* Increase of protein in the spinal fluid is never found in disseminated sclerosis but occurs in spinal cord compression.

Course and Prognosis.—(1) The outlook is better in the remitting type of the disease. Sometimes a remission lasts for years and raises hopes of cure, but, in all except the rarest cases, the symptoms recur. Death occurs in from five to thirty years from the onset, from urinary infection, bed-sores, or hypostatic pneumonia. The optic atrophy of disseminated

sclerosis never progresses to complete blindness. (2) The chronic progressive type of the disease is more rapidly fatal. Pregnancy and infections have an adverse influence on the course of the disease. The disease is not transmitted to children and therefore marriage may be permitted if the contracting parties understand the nature and probable course of the disease.

Etiology.—The disease occurs between the ages of sixteen and forty-five years. It is five times commoner in females than in males. *Infectivity.*– Familial cases are occasionally recorded, but the disease is not known to be contagious. *Frequency.*– It is the commonest organic nervous disease met with in neurological practice in the British Isles and appears to be on the increase. The disease is essentially due to scattered plaques of demyelination, mostly the size of lentils, but perhaps larger or smaller, which appear sown throughout the brain and spinal cord at varying depths from the surface and occasionally in the peripheral nerves. The cause is unknown.

Treatment.—The patient should spend twelve hours of the twenty-four in bed, and should rest as much as possible at the week-ends as long as the occupation can be continued. In other cases one day a week in bed should be prescribed. The patient should continue with the usual occupation until forced to give up, for prolonged rest in bed only increases spasticity. Short periods of rest in bed, a week or fortnight, may be advised when the patient is overtired. Liquor arsenicalis in ♏3 or 4 doses, well diluted, thrice daily after meals, may be prescribed in combination with tincture of belladonna ♏6, when there is precipitancy of micturition. To avoid a cumulative effect drop the medicine for one week in each month. In acute cases, intravenous arsenic in the form of neoarsphenamine B.P. (N.A.B.) or intramuscular sulpharsphenamine may be prescribed in weekly doses of 0·45 G. Quinine bihydrochloride grains 5, twice daily, has been recently advocated. General tonics, such as liver and iron, help debilitated patients. There is no indication for therapeutic abortion after the third month. Massage improves the spasticity in the early stages; in later stages, with flexor rigidity, only passive movements should be prescribed. Unsteadiness in walking is helped by Frenkel's exercises. The treatment of the late stages with paraplegia is described in § 761.

Group V. (III). *There is spastic paralysis of both legs. The condition is* Spastic Paraplegia *and it results from a spinal cord lesion. If the arms are also affected, the lesion is in the cervical region and the condition is termed* Brachial Paraplegia.

The Causes of Spastic Paraplegia may be divided into two groups according to the mode of onset:

(A) Spastic Paraplegia of Gradual Onset, and (B) Spastic Paraplegia of Acute Onset.

§ **756.** (A) **Spastic Paraplegia of Gradual Onset.**—The diagnosis of the cause of a paraplegia of gradual onset may present considerable difficulty.

The presence of *persistent localised root pain*, either unilateral or bilateral, should at once suggest the possibility of SPINAL COMPRESSION. The SYSTEM DISEASES of the spinal cord causing progressive paraplegia *can, in almost all cases, be excluded at once by a manometric examination of the spinal fluid.* The *loculation syndrome* of Froin (an increase in the total albumen, absence of, or slight increase in, the number of cells, and yellow coloration, xanthochromia) is pathognomonic of spinal cord compression. If you neglect these simple examinations you will fall into errors of diagnosis of the most serious import for your patient and yourself.

THE SYSTEM DISEASES OF THE SPINAL CORD producing **spastic paraplegia of gradual onset** are :

I. Disseminated Sclerosis.
II. Motor Neurone Disease, including Syphilitic Amyotrophy.
III. Spinal Syphilis.
IV. Syringomyelia.
V. Subacute Combined Degeneration of the Cord.
VI. Friedreich's Ataxia.

I. DISSEMINATED SCLEROSIS may present the picture of an incomplete transverse lesion of the cord, but such cases show other symptoms (§ 755).

II. MOTOR NEURONE DISEASE may be present with a paraplegia. Here the symptoms are purely motor. Wasting of the intrinsic hand or bulbar muscles or fibrillation may be present, and, except in *Syphilitic Amyotrophy*, the spinal fluid is normal (see § 788).

III. SPINAL SYPHILIS.—(i.) *Syphilitic amyotrophy* is described in § 789. (ii.) *Syphilitic meningomyelitis* develops slowly with root pains or girdle sensations round the trunk or in the limbs, numbness in the extremities and gradually oncoming spastic paraplegia. Objective sensory impairment is found and temperature and vibration are early affected. The tendon reflexes are exaggerated or diminished, the abdominal reflexes absent and the plantar reflexes extensor. Hesitancy and overflow incontinence occur as bladder symptoms. The root pains and slow progress of the malady may suggest spinal compression, but the spinal fluid shows a lymphocytosis as well as increased protein and the Wassermann reaction is positive. (iii.) *Erb's syphilitic spinal paralysis* occurs as a slowly developing spastic paraplegia, without pain but with early bladder involvement and little sensory loss. The absence of pain and lymphocytosis in the spinal fluid are due to the absence of meningeal reaction in this disease. The blood Wassermann reaction is always positive. In these syphilitic lesions Argyll-Robertson pupils may be present.

IV. SYRINGOMYELIA may simulate a spinal compression in the cervical region but is recognised by its characteristic sensory changes ("dissociated anæsthesia"), the presence of scoliosis, painless whitlows and upper limb arthropathies. The spinal fluid is usually normal or may show slight protein increase (§ 818).

V. SUBACUTE COMBINED DEGENERATION of the cord is a cause of spastic paraplegia, recognised by the tingling and numbness in the fingers and feet, the absence of a segmental level of anæsthesia and characteristic megalocytic anæmia and achylia. The spinal fluid is normal (§ 811).

VI. FRIEDREICH'S ATAXIA is recognised by the ataxic dysarthria, nystagmus and titubation, absent tendon reflexes, pes cavus and scoliosis (§ 813).

Flaccid Paraplegia of gradual onset may result from lower motor neurone lesions, *e.g.*, polyneuritis (see Group IX).

Treatment of Paraplegia due to any of these causes. See § 761.

§ 757. Spinal Cord Compression.

CAUSES—*Vertebral*

(1) Tuberculous, and other forms of Osteomyelitis.
(2) Chondroma, Sarcoma and Secondary Malignant Tumours.
(3) Osteitis deformans.
(4) Erosion by Thoracic Aneurysm or Lymphadenomatous deposits.

Meningeal

(5) Hypertrophic Cervical Pachymeningitis (Syphilitic).
(6) Circumscribed Serous Meningitis.
(7) Endothelioma.

Nerve-Roots

(8) Benign, neurofibromata.

Intramedullary

(9) Glioma, Angioma.

Of these causes Pott's Disease and Secondary Malignant Deposits are the commonest. Tuberculous caries compresses the cord either by the formation of (1) a tuberculous extra-thecal cold abscess, or (2) tuberculous granulations invading the theca. Malignant disease of the vertebræ (§ 693) is secondary to removal of a breast for carcinoma, to gastric carcinoma, to prostatic carcinoma, or hypernephroma. The lumbar vertebræ are commonly affected, with symptoms of bilateral "sciatica." Primary periosteal sarcoma of the vertebræ also occurs.

The VERTEBRAL *causes* are recognised by (1) Local tenderness or deformity of the spine, (2) the root pains are bilateral, (3) the X-ray findings may be characteristic. *The* MENINGEAL *causes* are recognised by the widespread distribution of the root-pains (several cutaneous segments) and by their severity. *The* NERVE-ROOT *cause* is recognised by the *unilateral* character of the root-pain or root-paralysis and its narrow distribution.

Symptoms of Spinal Cord Compression :

1. *Local Effects.*—These are early to appear and are due to involvement of the nerve-roots, posterior or anterior, by the cause of the compression. These root symptoms are of great help in localising the site of compression. When the posterior root is involved, pain of root distribution occurs, worse on coughing or sneezing, usually persistent and localised. Such root-pains are often accompanied by *cutaneous hyperæsthesia* to dragged pin, of segmental distribution (Fig. 176). When the anterior root is involved, wasting of the muscles supplied by that root ensues, with loss of power, flaccidity and diminished or absent tendon reflexes. The root symptoms will indicate whether the cause of the compression is acting posteriorly or anteriorly, according to whether sensory roots or motor roots are involved. Similar symptoms occur from damage to the segments of the cord itself and its anterior and posterior horns at the level of the lesion.

2. *Remote Effects.*—Cord symptoms next occur due to involvement of the long sensory and motor tracts, with accompanying motor, sensory

TABLE LII.

SYMPTOMS OF SPINAL CORD COMPRESSION AT VARIOUS SEGMENTAL LEVELS.

(For corresponding Cutaneous Root Areas, see Fig. 176.)

Cord Segment.	*Clinical Picture.*	*Muscles chiefly Paralysed.*	*Reflexes Abolished.*	*Other Features.*	*Level of Spinous Process.*
C. 3–4.	Brachial Paraplegia.	Lower part of Trapezius. Supraspinatus. Infraspinatus.		Relative analgesia and thermanæsthesia of face. Diaphragmatic palsy.	CIII
C. 5.	Brachial Paraplegia.	Biceps, Deltoid, Brachialis anticus. Supinator longus.	Biceps jerk (C. 5–6)	Triceps jerk + +	CIV
C. 7.	Paraplegia.	Triceps and Extensors of Wrist and Fingers.	T.J. and S.J. (C. 6–7).		CVI
C. 8–Th. 1.	Paraplegia.	Flexors of Wrist and Fingers. Small muscles of Hand.		Paralysis of ocular sympathetic. Arm jerks. +	CVII
Th. 6.	Paraplegia.	Intercostals. Upper Rectus abdominis.	Epigastric (Th. 6–8).	Spastic paralysis of trunk muscles and lower limbs.	Th. IV
Th. 9–10.	Paraplegia.	Lower Rectus abdominis. Obliquus abdominis.	Upper Abdominal (Th. 8–10).	Umbilicus deviates upwards.	Th. VIII
Th. 12–L. 1.	Paraplegia.	Ileopsoas.	Lower abdominal (Th. 11–12). Cremasteric (L. 1–2).		Th. X
L. 3–4.	Paraplegia.	Adductors of thigh. Quadriceps extensor.	Knee jerk (L. 2–4).	Flexion of wasted hip preserved.	Th. XI
L. 5.	Paraplegia.	Hamstrings.			
S. 1.	Paraplegia.	Glutei. Calf muscles.	Ankle jerk (S. 1–2).		
S. 2.	Paraplegia.	Anterior Tibial muscles. Peronei. Small muscles of foot.	Ankle jerk (S. 1–2).	Anal and Bulbocavernosus reflexes preserved.	Th. XII
S. 3–4.	No motor symptoms in legs.	Perineal muscles. Levator ani.	Bulbocavernosus (S. 3–4). Superficial anal. (S. 4–5).	Retention of urine and fæces. Motility of lower limbs and deep reflexes normal.	L. 1

and reflex changes below the level of the lesion. Spastic paralysis may be noticed first in the lower limbs on the side of the lesion, from pressure on the homolateral pyramidal tract. It spreads, and a spastic paraplegia develops with increased tendon reflexes, rectus- and ankle-clonus and extensor plantar responses. At first the lower limbs show *paraplegia in*

extension from interruption of local spinal reflex arcs. Later, when the long mid-brain reflex arcs subserving tonus in the extensor muscles are involved, *paraplegia in flexion* occurs, with involuntary flexor spasms, and, later, the thighs become flexed on the abdomen and the legs on the thighs. Sensory changes also develop in one of two ways. (1) Sensory loss may begin in the lowest sacral segments with anæsthesia over the "saddle-area," or (2) Sensory loss commences in the periphery of the limbs. Whatever the mode of onset, it gradually spreads up to the segmental level of the lesion, involving all forms of sensibility. In testing cutaneous sensibility at the level of the lesion we find the level highest for touch, lowest for pain (pin-prick) with temperature intermediate, owing to the oblique crossing of the fibres going to the spino-thalamic tracts. Not uncommonly, an incomplete *Brown-Séquard Phenomenon* is observed. The pyramidal weakness and loss of deep sensibility (joint sense and vibration) is greater on the side of the lesion, with loss of sensation to pain, heat and cold

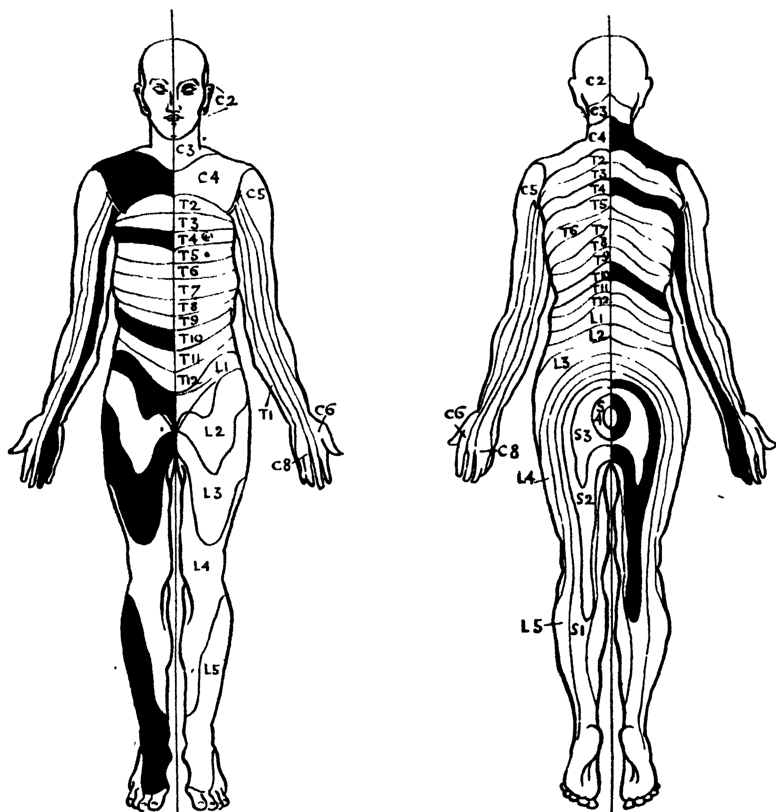


FIG. 176.—DIAGRAM SHOWING THE CUTANEOUS AREAS SUPPLIED BY THE SPINAL NERVE ROOTS. (Modified from Head.)

The student should memorise the blackened segmental areas.

in the contralateral lower limb. At first, there is hesitancy of micturition; later, reflex incontinence or overflow with dribbling occurs and incontinence of fæces. The abdominal reflexes disappear if the lesion is above the thoracic region. The tendon reflexes, which lie in the compressed segment or segments, are abolished. Below the level of the lesion they are increased, with clonus. Cord lesions involving C 8 and Th 1 segments, or above this, may be associated with oculo-pupillary sympathetic paralysis (miosis, enophthalmos and narrowed ocular fissure) on the side of the lesion. Lesions of the Cauda Equina are described in § 798. Unless the paraplegia is relieved, irreparable ascending and descending degenerations occur in the spinal cord tracts. Such degenerations occur after a year of compression, and, when the stage of paraplegia in flexion is reached, they are well established. Death, in such cases, takes place from infection, bed-sores or ascending pyelo-nephritis.

3. *Loculation Syndrome of Froin.*—This is due to a block in the subarachnoid space produced by the cause of the compression. The spinal fluid stagnates below the level of the lesion and the congested anterior and posterior spinal veins leak protein and hæmoglobin into it. The effects produced are: (1) Increase of protein above the normal (0·025 to 0·05 mgm. per cent.). Readings above 1·0 per cent. are common in this syndrome. (2) Yellow coloration of the fluid (this may have to be looked for) with massive coagulation (xanthochromia with massive coagulation), and (3) an absence or slight increase in the normal number (5 to 7 per cu. mm.) of cells. The fluid, above the level of the block obtained by cisternal puncture, may be normal. When the patient is in the lateral position with a lumbar puncture needle in the theca, the normal respiratory oscillations are absent if the block is complete, jugular compression (Queckenstedt's Phenomenon) produces little or no elevation in pressure and, after withdrawal of 5 c.c. of fluid, the pressure may fall to zero and not rise again. Above the block the cisternal needle connected with a cisternal manometer will show normal respiratory oscillations, normal rise on jugular compression, and normal fall after withdrawal of 5 c.c. of fluid.

Diagnosis of the Level of the Lesion.

The guide is the upper limit of the signs. The *root symptoms* are of paramount importance in this respect (Fig. 176). The root area of anæsthesia at the level of compression must be carefully mapped out and its upper limit defined with a skin pencil, testing each form of cutaneous sensibility in turn, working from the anæsthetic to the normal skin and carefully charting the observations in each case. In this connection, root areas of hyperæsthesia at the upper limit of sensory loss are as important as anæsthesia, and may indicate the necessity for a localisation above the upper limit of sensory loss. Any local loss of power, due to pressure on anterior roots, is of the greatest use in localisation of the level of the lesion. Wasting of an intercostal muscle may be observed. Lesions of the 9th Thoracic segment produce paralysis of the lower rectus with deviation of the umbilicus upwards and to the contralateral side when the patient attempts to sit up. Lesions of the 11th Thoracic segment spare the rectus but paralyse the obliquus abdominis, with bulging in the affected flank when the patient attempts to sit up.

Abolition of the tendon reflexes occurs in those reflexes seated in the affected cord segments. The upper limits of the *tract anæsthesia* often merges with the root anæsthesia but should always be carefully charted.

It is seldom possible to make the diagnosis between *Intra-medullary* and *Extra-medullary* lesions. In Intra-medullary lesions, however, (1) root pains are slight, (2) the lowest sacral segments tend to escape sensory loss, and (3) "dissociated anæsthesia" is more common than in extra-medullary compression.

Radiological Localisation.—Simple radiograms of the spine should not be omitted. Growths within the vertebral canal may erode adjacent pedicles, or may cause an apparent separation of the pedicles. Oblique radiograms may show enlargement of an intervertebral foramen where a "dumb-bell" neurofibroma exists on the nerve root.

Radio-opaque oils (*e.g.* lipiodol, myodil, pantopaque) are sometimes injected intracisternally, or by the lumbar route, in order to localise the precise upper or lower level of a spinal block. The injection is made with the patient in the sitting position. A few c.cs. of spinal fluid are withdrawn and kept for analysis and ½ to 1 c.c. of lipiodol injected. The lipiodol, owing to its density, should fall normally to the bottom of the theca. If the lipiodol is injected by the lumbar route, the patient is subsequently X-rayed on a tilting table so that the lower limit of the compression can be demarcated. These oils may cause local irritation at the site of their intrathecal arrest. Their use, therefore, should be reserved for cases where the localisation is especially difficult, or for those in which laminectomy is pending. They may cause increase in bladder symptoms in compression lesions of the cauda equina.

The level of the lesion having been determined clinically or radiographically, it must be noted that the segmental level of the cord segments differs from the level of the bony spinous processes. (See Table LII.) The tendency is to localise too low.

Hypertrophic Cervical Pachymeningitis (§ 820) is a syphilitic pachymeningitis localised to the cervical region, with wasting of the hand or shoulder-girdle muscles, sensory impairment, persistent root pains in the neck and upper limbs; and in the later stages, a spastic paraplegia from cord compression.

Circumscribed Serous Meningitis is a circumscribed cystic condition of the leptomeninges, probably inflammatory, sometimes discovered at operation for a supposed spinal tumour. The condition comes on months or years after an injury to the spine.

Prognosis of Compression Paraplegia.—The prognosis of compression in *tuberculous caries* is wonderfully favourable, provided that compression has not existed longer than eighteen months, when ascending and descending degenerations are likely to have occurred. No case is hopeless, but the existence of paraplegia in flexion, urinary infection and severe bed-sores are of grave import. *Vertebral tumours*, either primary or secondary, cause death in weeks or months. *Meningeal tumours* and *neurofibromata* growing from the nerve-roots are relatively benign and recovery may be looked for in cases where the symptoms have not been present longer than a year. Few *intramedullary* tumours can be removed without severe damage to the spinal cord. *Syphilitic* cases, if of recent onset, may recover with appropriate medicinal treatment.

Treatment, see § 761.

(B) **Spastic Paraplegia of Acute Onset.**

Paraplegia, coming on in a few hours, may be due to:

I. Sudden Compression from Fracture Dislocation of the vertebræ (*e.g.*, injury, gunshot wounds).
II. Acute Myelitis.
III. Hæmatomyelia.
IV. Caisson Disease.
V. Hysterical Paraplegia.

§ 758. I. **Sudden Compression from Fracture Dislocation.**—The paralysis of the lower limbs is at first flaccid from shock effect, with total anæsthesia below the level of the lesion. There is retention of urine and priapism, and bed-sores rapidly develop. About the third week after the onset of the lesion, spasticity develops in the paralysed extremities, with reflex incontinence and pyramidal signs. Flexor spasms may occur in the legs with associated involuntary micturition and defæcation, and sweating over the paralysed limbs—" mass reflex." If a useful degree of recovery of function is to occur, some recovery is usually manifest by the end of the first week. The majority of cases die in the phase of spinal shock, the remainder survive to a life of invalidism. Fracture dislocation and hæmatomyelia may both occur from diving into shallow water, or from a fall from a height on to the buttocks.

II. Acute Myelitis is described in § 736.

§ 759. III. **Hæmatomyelia,** or hæmorrhage into the substance of the spinal cord, produces symptoms like those of syringomyelia (see § 818) of rapid onset. The disease occurs in young adults before the age of thirty years. Root pains and paræsthesiæ are followed in a few minutes by loss of power in the arms and weakness in the legs. Muscular atrophy occurs in the intrinsic hand muscles, the paraplegia becomes spastic and characteristic " dissociated anæsthesia " is present. The site of the hæmorrhage is usually in the lower cervical region. The condition follows gunshot wounds of the spine, obstetrical injuries, or the hæmorrhage may occur into a congenital cleft in the spinal cord. The *diagnosis* has to be made from fracture-dislocation of the 5th and 6th cervical vertebræ, which may produce identical symptoms. The *treatment* consists in arresting hæmorrhage with morphine and quiet, and the nursing of the associated paraplegia.

§ 760. IV. **Caisson Disease** (Synonyms : Diver's Paralysis, Compressed Air Illness) is a form of acute paraplegia occurring in divers, workers in diving bells and in underground tunnellers, working at high atmospheric pressure. Too rapid decompression causes the excess of nitrogen in solution in the tissues and body fluids to be released as bubbles of gas in the tissues, especially in the central nervous system. If decompression is gradual, the nitrogen is liberated into the blood and excreted by the lungs, and symptoms do not occur.

Symptoms.—These depend on the localisation of the bubbles of nitrogen and their size. They occur thirty minutes to several hours after decompression is complete. Severe root-pains, " the bends," are common at the onset, then a paraplegia, transient or permanent, results. The paraplegia is never complete and the arms are rarely affected as much as the legs. Anæsthesia and sphincter paralysis only occur in the graver cases. Auditory vertigo, tinnitus, hæmorrhage of the nose, lungs and other parts, sometimes occur, with abdominal distension, pain and vomiting. The prognosis is favourable in most cases ; pain and paresis pass off in a few days to six weeks. A few cases have died.

Treatment.—The patient should be immediately put in a compressed air chamber and exposed to the same pressure as that under which he was working. This results in reabsorption of the nitrogen, with relief of symptoms, and decompression can then take place very gradually.

Prevention consists in adopting precautions for gradual decompression. Snell recommends ten minutes decompression for each atmosphere of pressure. The obese and alcoholic are more liable to the disease.

V. **Hysterical Paraplegia** may be of sudden or gradual onset (§ 888). The early paraplegia of disseminated sclerosis is frequently confounded with hysteria. In hysterical paraplegia the tendon reflexes and abdominal reflexes are unaltered, while the plantar responses are usually absent at the toes.

§ 761. **Treatment of Paraplegia :** (1) The *cause* should be treated. Where *spinal tumour* exists, a laminectomy should be undertaken, otherwise the cause of the disease leads to complete paralysis and death. The mortality rate of operations for spinal tumour, in expert hands, is less than 10 per cent. Improvement may continue for a

year or more after operation. *Secondary malignant disease* in the vertebræ should be treated symptomatically, relief of pain being achieved by such combinations as aspirin gr. 10, pyramidon gr. 5, heroin gr. $\frac{1}{12}$–$\frac{1}{8}$, given by mouth night and morning. Intrathecal injections of 2 cu. cm. of 25 per cent. magnesium sulphate solution may be tried at weekly intervals, or X-ray irradiation of the affected regions may give relief. In the case of *hypertrophic cervical pachymeningitis* and *syphilitic cases*, anti-syphilitic treatment should be commenced forthwith (§ 552). *Pott's Disease* is treated by fixation of the spine and extension, for periods not less than six to twelve months. Abscesses may have to be aspirated. Laminectomy in such cases is commonly fatal from tuberculous meningitis, whereas the results of conservative treatment are often brilliant even in cases with severe paraplegia. Natural sunshine treatment as practised by Gauvain and Rollier has much success (§ 557). In *Subacute Combined Degeneration* anti-anæmic measures should be instituted (see § 811).

(2) *Nursing.*—The patient should be nursed on a sponge rubber bed if possible, and the posture changed every three hours. The greatest care should be taken to avoid wrinkles in the sheets under the patient, and the patient must always be lifted, never dragged along the bed. Deep breathing exercises should be encouraged in the early stages to avoid hypostatic pneumonia, and the patient should be nursed propped up with pillows. A bed-cradle, sufficiently high to clear the lower limbs even when in flexor-spasm, will be necessary, with a rubber air-ring, and rings for the heels, and pads, bandages, and pillows to protect pressure points. Light splinting or sandbags will keep the affected limbs in good posture. Hot water bottles should have thick covers, free from holes, and should never be placed in direct contact with the patient's skin. A hot water bottle may burn an insensitive or unconscious patient through two layers of blankets. The skin should be washed twice daily with soap and water and carefully dried, rubbed with spirit or eau de Cologne, and powdered with zinc oxide and starch.

(3) The *bowels* should be kept constipated and opened by soap and water enemata (1–1½ pints) on alternate days. Strong purgatives may lead to rectal sloughing. As leakage may occur from the rectum after even mild aperients, the patient may be left on an air-cushioned rubber bed-pan after a bowel action, until leakage has ceased, when an absorbent pad should be applied. Abdominal distension from flatus is helped by intramuscular posterior pituitary extract ½ c.c.

(4) The *care of the bladder* is of greatest importance. An attempt should be made by suprapubic pressure to get the patient to empty his bladder. The use of carbachol injections is not advised. In most cases eight-hourly catheterisation is required, and for this the operator must scrub up and use rubber gloves as for a surgical operation. Before the catheter is passed, the meatus must be cleaned and the anterior urethra irrigated with 1 in 4,000 oxycyanide of mercury solution. Between catheterisations, if leakages occur, the penis may be inserted in the neck of a sterile swan-necked urinal. In hospital and where the patient has a nurse attendant, the method of automatic vesical lavage known as "tidal drainage" is likely to lessen the risk of infection.[1] In chronic cases, a catheter may be inserted into the bladder through a suprapubic incision and connected to a tidal drainage apparatus. The urine should be kept acid with acid sodium phosphate (30 to 60 gr.), ammonium chloride (5 to 60 gr.), or ammonium mandelate (50 gr.). Sulphamezathine may be given orally to combat infection. Suitable lavage solutions are 1 in 10,000 potassium permanganate, or 1 in 1,000 flavasol at 105° F.

(5) *Back blisters* should be aspirated and the skin left intact. Once an area of necrosis forms, a slough will separate. Deep sloughs should be curetted. The separation of more superficial sloughs may be helped by compresses of hydrogen peroxide applied for half an hour twice daily. Saline or sterile paraffin dressings well covered with elastoplast should be changed daily. Systemic penicillin and daily local applica-

[1] Munro, D., and Hahn, J., *New Engl. J. Med.*, 1935, **212**, 229.

tions of penicillin (20,000 units in 10 c.cs. normal saline) may help to sterilise a sore. Zinc oxide castor oil paste is useful in treating clean bedsores (see also § 617).

General measures to replace protein loss and dehydration are indicated, *e.g.*, blood transfusions, high protein diet, drinks of glucose and fruit juices.

(6) *Flexor and adductor spasms* in the lower limbs are most difficult to treat. Contact of the limbs with the bed clothes must be reduced to a minimum by cradles. Tinct. gelsemii ♏ 20, codein gr. ¼, aspirin gr. 10, or phenobarbitone gr. ½, may be tried separately or in combination, thrice daily. Excessive sweating is controlled by adding tinct. belladonnæ ♏ 10 to a medicine.

(7) *Physiotherapy.*—Voluntary movements should be encouraged. Daily bed-exercises, chair and walking exercises, and exercises on a couch, with the affected limbs suspended in slings, should be undertaken. Many apparently helpless cases can be taught to walk, at first between parallel bars and later with elbow crutches. As soon as possible in the convalescence the patient should be got out of doors on a couch or wheeled chair.

Group V. (IV). *There is spastic paralysis of all four limbs, including the face. The condition is* SPASTIC DIPLEGIA, *and it results from a bilateral cerebral lesion or double hemiplegia.*

In **Brachial Paraplegia** the upper and lower limbs show spastic paresis, but the face escapes. The lesion in such cases is in the medulla or cervical spinal cord above the C 5 segment (see §§ 756, 757).

Spastic Diplegia occurs from bilateral lesions in the internal capsules in CEREBRAL ARTERIO-SCLEROSIS; in lesions of the pons and medulla, *e.g.*, NEOPLASMS, and from bilateral lesions involving the upper parts of the Rolandic areas, *e.g.*, LONGITUDINAL SINUS THROMBOSIS, CHRONIC SUBDURAL HÆMATOMA. In the latter cases the lower limbs are chiefly affected by the weakness and spasticity.

The condition is seen, characteristically, in children, as the result of pre-natal or post-natal atrophic sclerosis of the cerebral cortex: CEREBRAL DIPLEGIA.

§ **762. Cerebral Diplegia** (Little's Disease).—The *symptoms* are noticed at birth (pre-natal) or within the first three years of life (post-natal). The infant never learns to walk correctly. The extensor plantar response of infancy is perpetuated, and the legs are spastic and adducted, the arms flexed across the chest and adducted at the shoulders. When an attempt is made to put the child to the ground, stepping may be attempted, but the legs tend to cross in front of one another (scissor-gait). The degree of intelligence varies but is commonly subnormal. The post-natal cases tend to progress, the pre-natal ones to improve. Microcephaly, internal squint and contractures, are frequently present. The head is symmetrical, the distribution of the spasticity is rarely perfectly symmetrical. The following types occur:

(1) *Congenital Paraplegia* (Cortical Diplegia).—The spasticity affects the legs only. The mentality is usually bright and the outlook fairly good.

(2) *Cortico-Striate Diplegia.*—Athetosis or choreiform movements are added to the spasticity on one or both sides, rarely symmetrically. The prognosis is bad and the disease tends to progress.

(3) *Striate Diplegia* (Congenital Chorea, Congenital Athetosis), see § 771.—These children are often mentally normal, but writing and reading are impossible owing to the perverse movements.

(4) *Cerebellar Diplegia and Cerebro-cerebellar Diplegia.*—The signs are those of cerebellar disease and the hypotonia resembles that of Amyotonia Congenita, but is never so great. Nystagmus may be present.

Etiology.—Syphilis is not a cause. The lesion found in the brains of such children

is a scattered atrophic condition of the convolutions with, occasionally, porencephalic cystic cavities, which may or may not communicate with the ventricles.

Prognosis.—In the absence of fits, severe sphincter disturbance or gross mental defect, education should be undertaken. Intelligence tests will reveal the mentality, and careful clinical examination the physiological residuum of voluntary movement.

Treatment.—Orthopædic treatment, combined with massage and remedial exercises, patiently persisted in over months or years, has wonderful effects in non-progressive cases. Petit mal attacks occurring in such children should be treated as in ordinary epilepsy.

§ 763. **Cerebro-Macular Degeneration** (Amaurotic Family Idiocy) is a familial disease, particularly common in Jewish families. The onset is at three to six months of age (Infantile Type) or in the first three years of life (Juvenile Type). The *symptoms* are those of a progressive diplegia, with rapid mental deterioration and repeated slight epileptiform convulsions. The *diagnosis* is made from other forms of diplegia by the finding of a cherry-red spot at the macular region of both fundi in the infantile type, and a "pepper and salt" appearance of the macula in the juvenile type. Primary optic atrophy occurs in both types. Death occurs rapidly in six to twelve months. The disease is a degeneration of the nerve-cells throughout the whole nervous system, including the spinal cord. Treatment is of no avail (§ 907 C).

§ 764. **Decerebrate Rigidity in Man.**—*In animals*, when the controlling influence of the cerebrum is removed by section through the red nucleus, the full effect of the influence of Deiter's nucleus on muscle tone is shown by the condition of "decerebrate rigidity" which results. In man, however, lesions of the pyramidal tract alone are sufficient to produce the attitude of decerebrate rigidity. The position of the leg is extension at the knee and plantar-flexion ("extension") at the ankle, while the head is extended in both animals and man. *In man*, the position of the arm differs from the extension seen in animals; it lies semi-flexed at the elbow across the chest, the forearm slightly pronated and the fingers flexed. The explanation of the different attitude of the upper limb in man is due to the fact that man uses his upper limb in a very different way from that in which an animal uses its forelimbs—for prehension, not for progression. Tonus is increased in the extensor (anti-gravity muscles) connected with the maintenance of the erect posture. In man the condition is sometimes seen in its fully developed form, in tumour, hæmorrhage, meningitis, causing interruption of the brain-stem above the level of the red nucleus, and it is seen also in cerebral diplegia, anencephaly and hydrocephaly. In an incomplete form, it is seen in hemiplegia and other injuries to the pyramidal tracts. In irritative lesions, the rigidity may take the form of *Tonic Fits*. The influence of the vestibular mechanisms can sometimes be demonstrated in these cases by altering the position of the head. Passive rotation of the head will cause increased extensor tone in the leg, and increased flexion in the arm towards which the chin is turned, with corresponding diminution in tonus on the contralateral side (tonic neck reflexes of Magnus and de Kleijn, § 707).

GROUP VI. PARKINSONISM

The patient shows continuous stiffness and slowness of movement, and a fixed mask-like expression. The condition is PARKINSONISM, *and it is encountered in the following diseases:*

I. Paralysis Agitans.
II. Post-Encephalitic Parkinsonism.
III. Arterio-sclerotic Rigidity.
IV. Hepato-Lenticular Degeneration.

§ 765. *The Clinical Picture of* **Striatal Rigidity**, or Parkinsonism.—The onset is insidious, and (1) *Muscular Rigidity* (poverty of movement) is

the earliest symptom. The face assumes a fixed, unblinking, mask-like, staring expression (Parkinsonian mask). In the limbs, the rigidity is at first hemiplegic in distribution, the arm being usually affected before the leg, though not invariably so. Later, the rigidity is bilaterally distributed. The natural swing of the arm in walking is first lost and the limb lies adducted and slightly flexed at the side of the trunk, while the gait assumes a monotonous gliding character. The attitude becomes one of moderate flexion, head bent forwards, arms adducted and flexed at the elbows, forearms held between pronation and supination, fingers slightly flexed in the position of interosseous extension (interosseal attitude) and the legs always flexed at the knees in sitting, walking or standing (Fig. 4). On walking, the patient moves like a statue and turns round rigidly. He tends to walk faster and faster (festination) and will fall forwards unless pulled up against a table or other object. If pushed backwards, he continues to walk backwards, unable to stop until he meets an obstacle (retropulsion). But in spite of his difficulty in walking, he may display agility in running, jumping or catching a ball. The handwriting becomes progressively smaller. (2) Usually within a few months of onset the arm first affected becomes the seat of rhythmic *Tremor*, which may be present when the limb is at rest and fully supported, or may appear only when it is in action. It can often be controlled by an effort of will or by voluntary supination of the forearm, but only for a short time. It disappears during sleep and in rapid movements, and is intensified by fatigue or emotion. It may begin in the fingers and thumb, which are approximated in "pill-rolling" movements, and, later, spreads to the muscles of the forearm. Coarse rhythmic shaking of the semi-flexed fingers may be observed as the patient walks, indicating the limb or side of the body most affected. As the seated patient confronts you on his chair, you may notice rhythmic shaking of one or both lower limbs even when his feet are supported on the ground. Tremor may be seen in the lower jaw as a slow rhythmical oscillation, or the head may be tremulous. The eyelids flicker tremulously when the lids are lightly closed. (3) Gradual *Weakness*, largely due to rigidity, is seen, especially in the small muscles of the hand, so that delicate manipulations and especially those needing repetition become difficult. (4) All movements become *Slow* and restricted in range, speech becomes monotonous, mastication is slow and saliva dribbles from the mouth. This combination of weakness of the small muscles of the hands and slowness of performance causes progressive difficulty in dressing, *e.g.*, fastening buttons and fixing collar-studs. The toes curl downwards on the soles of the feet (Purves-Stewart) and numerous aches and discomforts ensue from the immobility manifested, resulting in restlessness and a constant desire to be moved in bed.

Some weakness of ocular convergence is almost always present; the conjugate movements of the eyes are jerky (*saccadé* movements). Ultimately, the patient becomes bed-ridden and helpless. Periods of stupor occur, muscular rigidity is so intense as to make swallowing difficult and

speech becomes almost unintelligible. Progressive mental deterioration sets in, in most cases, and the last months of life are spent in stupor and anarthria, with a terminal coma.

Causes of Parkinsonism.

§ 766. I. **Paralysis Agitans** (Parkinson's Disease).—This disease rarely occurs before the age of fifty, and 65 per cent. of the cases are males. It is due to a senile degeneration of the nerve-cells and -fibres in the corpus striatum and sub-thalamic region. The course is slowly progressive over a period of ten years, and spontaneous arrest may be seen. Rigidity may be seen alone, or tremor alone, or a combination of the two.

§ 767. II. **Post-Encephalitic Parkinsonism.**—As a result of infection with the virus of Encephalitis Lethargica, Parkinsonism frequently appears. There may be no history of an "acute attack," the onset of the Parkinsonism being the only sign of the progressive activity of the virus in the mid-brain extra-pyramidal nuclei (substantia nigra and sub-thalamic region). The rigidity may be the residuum of an acute attack, or may follow a complete recovery from such an attack after a period of as long as nine years. The condition may remit, but rarely improves. The course is as in Paralysis Agitans, but sudden exacerbations may occur. Post-Encephalitic Parkinsonism is distinguished by the following features:

(1) The patient may be of any age from childhood onwards.

(2) Owing to the incidence of the disease on the mid-brain, involvement of the ocular mechanisms occurs early. The ocular signs persist, and are valuable evidence of the encephalitic origin of the Parkinsonism. Any known abnormality of pupillary reaction may be met with, including the Argyll-Robertson phenomenon and its converse. Protracted attacks of upward or lateral conjugate deviation of the eyes, lasting minutes or hours (*oculo-gyric crises*), are highly characteristic of this malady.

(3) Bizarre and complicated involuntary movements of the voluntary muscles may be present, and, when these involve the respiratory mechanisms, "respiratory antics" occur, paroxysms of tachypnœa, faint breathing, yawning, etc.

(4) An extensor plantar response may be present in rare cases.

(5) The skin of the face may be excessively greasy, and sialorrhœa is common.

§ 768. III. **Arterio-sclerotic Rigidity.**—In subjects of Cerebral Arterio-sclerosis, with high blood-pressure and evidence of arterial disease in the retinal or systemic arteries, Parkinsonism may result. The plantar responses, in such cases, are often extensor, a rare finding in other forms of Parkinsonism. The gait becomes shuffling, with short steps, and the rigidity is never as marked as in fully-developed Parkinsonism from other causes. It may be part of the picture of "Pseudo-Bulbar paralysis" with spastic involuntary weeping or laughter, and gross dysarthria (see § 746).

Diagnosis.—The rigidity of Parkinsonism should not be confused with *Arthritic Muscular Rigidity,* met with characteristically in *Spondylitis Deformans.* In the latter condition there may be poverty of movement and a general attitude of flexion, but the facies is never mask-like, tremor is absent and root-pains occur. X-ray plates show lipping of the vertebral bodies in the affected regions of the spinal column.

Treatment.—In the great majority of cases the cause of Parkinsonism is either unknown or cannot be influenced directly by treatment. The condition inevitably progresses, and treatment therefore is symptomatic. The patient should be advised to continue at work for as long as he possibly

can. Daily walking exercise or daily car or bus rides should be taken as long as the patient is able to do so. Rigid cases will require help with cutting-up of food, bathing, dressing and with turning over in bed at night. The question of the patient going to a hospital or home arises in the later stages: provided they have the attention, most elderly patients are happiest in their own homes. The illness, however, imposes a severe burden on the relatives.

The *rigidity* may be influenced in considerable degree by the Atropine series of drugs. Not all patients will submit to taking hyoscine, belladonna or stramonium continuously: some prefer to take the drug only before making a special effort. Hyoscine hydrobromide 1/200th to 1/100th, tinct. belladonnæ ♏ 5 to 15, or tinct. stramonii ♏ 10 to 60 twice or thrice daily may be given by mouth. ♏ 1 of a 0·5 per cent. solution of atropine sulphate may be given thrice daily increasing the dose gradually until a maintenance dose is reached. The appropriate medicine and its dose can often only be found after trial. These drugs diminish the salivation and sweating, but they also cause blurring of vision from paralysis of accommodation, flatulence and dryness of the mouth. During treatment by increasing doses of these drugs the pulse rate should not be allowed to rise above 120/minute. Toxic symptoms apart from tachycardia include skin rashes, vomiting, headache and delirium. The blurred vision may be helped by instilling into each eye one drop of 0·5 per cent. eserine solution every second day. The *tremor* is very little influenced by medication. It may be temporarily diminished by a car or bus ride. Some cases do best on a sedative mixture such as the following: Soluble phenobarbitone gr. $\frac{1}{4}$ to $\frac{1}{2}$, tinct. stramonii ♏ 15 to 20, thrice daily. Pilocarpine nitrate gr. $\frac{1}{12}$ to $\frac{1}{6}$ is sometimes added to diminish dry mouth and blurred vision. Benadryl (50 mgm. three or four times daily) has been tried with some good effect. It may be given alone or with the Atropine group. *Depression* may be helped by alcohol given alone or with food. Alcohol acts too as a digestive stimulant. Dexedrine 5 to 10 mgm. given after breakfast and before lunch helps depression but it should not be given continuously for long periods. It is said also to diminish the tendency to oculogyric crises in post-encephalitic Parkinsonism. Hypnotics may be required and should be in the charge of a responsible relative to be kept locked up and given to the patient as necessary. Helpless patients are better nursed in an arm-chair by day and in bed at night. Prolonged rest in bed is bad.

§ **769.** IV. **Hepato-Lenticular Degeneration** (Synonym: Progressive Lenticular Degeneration).—This is a rare and fatal malady occurring in adolescents, often in more than one member of a family. It is not hereditary. There is widespread and slowly developing striatal rigidity of the voluntary muscles, with tremor. Spasmodic involuntary weeping and laughter occur and there may be mild dementia. Articulation and deglutition are defective. The reflexes are qualitatively unaltered, there is no true paralysis of muscles. The average duration of life is four years from the onset. The diagnosis may be made by demonstrating (1) the associated portal cirrhosis of the liver, (2) a deposit of pigment in granular form at the periphery of the cornea, seen with a slit-lamp, (3) the similar affection of other members of the family. Post-mortem reveals a bilateral degeneration of the putamen of the lenticular

nucleus and, to a less extent, of the globus pallidus, and hepatic cirrhosis of the multilobular ("hobnail") type.

GROUP VII. INVOLUNTARY MOVEMENTS

The three main types of involuntary movements met with clinically, are TREMOR, CHOREA and ATHETOSIS. For a description of these and the physiological principles underlying their production, see § 672. Other types of involuntary movements are TIC (§ 772), MYOCLONUS (§ 775), FIBRILLATION (§ 776), and HICCOUGH (§ 273).

(1) *The patient comes to you with rhythmical* SHAKING *of some part of the body. The condition is* TREMOR.

§ **770. Tremor** is an involuntary rhythmical oscillation of one or more parts of the body, resulting from alternate contraction of muscle groups and of their antagonists (Purves-Stewart).

CAUSES OF TREMOR.

Toxic.
I. Hyperthyroidism.
II. Chronic Alcoholism.
III. Nicotine Poisoning.
IV. Mercurial Poisoning.

Organic Affections of the Brain.
V. Parkinsonism.
VI. Hepato-Lenticular Degeneration.
VII. General Paralysis of the Insane.
VIII. Disseminated Sclerosis.
IX. Friedreich's Ataxia.
X. Cerebral Tumour.
XI. Senile Tremor.

Functional.
XII. Anxiety Neurosis.
XIII. Hysteria.

Familial.
XIV. Familial Tremor.

In Infants.
XV. Spasmus Nutans.

CLINICAL INVESTIGATION OF TREMOR.—The (1) Amplitude, (2) Rate, (3) Distribution of the movements should be noted with (4) Factors increasing the tremor or diminishing it (voluntary movement, effort of will, sleep, etc.) and (5) whether the tremor is accompanied by "cog-wheel" rigidity, as in Parkinsonism. (6) The age of the patient is important. Tremor in children is familial, or due to Friedreich's Disease, or follows Encephalitis, sometimes syphilitic. In early adult life it is due to hyperthyroidism, encephalitis lethargica, disseminated sclerosis or psychoneurosis. After middle life it may be due to Paralysis Agitans, General Paralysis of the Insane, or it may be Senile. Tremor occurs in (*a*) Toxæmias, (*b*) Organic Affections of the Brain, (*c*) Functional Disease. Occasionally, tremor is (*d*) Familial. (*e*) In debilitated Infants, coarse tremor of the head (Spasmus Nutans) may occur.

(*a*) TREMOR *may be* **Toxic** *in origin.* Toxic tremors are best demonstrated by asking the patient to hold his hands horizontally in front of him, with his fingers widespread, when the tremor can be seen or felt by light palpation of the dorsum of his hands. Toxic tremor is characteristic of I. *Hyperthyroidism,* in association with tachycardia, thyroid enlargement, lid-retraction, exophthalmos and wasting. In II. *Chronic Alcoholism,* and in III. *Nicotine Poisoning* from excessive cigarette smoking,

and in poisoning by heavy metals, especially IV. *Mercurial Poisoning*, toxic tremors occur.

(*b*) Tremor may be due to **Organic affection of the brain.** It is seen in association with Striatal Rigidity in V. *Parkinsonism* (due either to *Paralysis Agitans*, *Encephalitis Lethargica*, or *Arterio-sclerosis*) and in VI. *Hepato-Lenticular Degeneration.* In VII. *General Paralysis of the Insane* spontaneous tremors of an irregular type occur in the lips and tongue, or can be demonstrated by asking the patient to write. They are often mistaken for simple nervousness, but the diagnosis can usually be made from the jumbling of syllables and slurred dysarthria, the irregular or unequal pupils, often of the Argyll-Robertson type, and the history of apathy, unreasonable irritability, or mistakes made at work. In doubtful cases, the spinal fluid examination will clear up the diagnosis. General *tremulousness* is seen in VIII. *Disseminated Sclerosis*, as well as the "intention tremor" met with in this disease. Intention tremor appears on voluntary movement only, as a coarse oscillation of the finger or limb just before the objective of the movement is reached. Titubation, a constant rapid oscillation of the head, may occur also in disseminated sclerosis. Tremor, intention tremor and titubation, are also met with in IX. *Friedreich's Ataxia*, accompanied by absence of the tendon reflexes, extensor plantar responses, scanning speech, and pes cavus, or scoliosis. In X. *Cerebral Tumours* of the frontal lobes, mid-brain or corpus callosum, tremor, often unilateral, may occur. XI. *Senile* tremor, or rhythmical head-nodding, occurs in the aged.

(*c*) XII. **Functional Tremors** occur in *Anxiety Neurosis*. XIII. *Hysterical* tremors are sometimes very difficult to differentiate from organic disease. They are, however, usually (1) sudden in onset, following emotional trauma, (2) they are variable and diminished or abolished by suggestion, (3) if rigidity is present, it is of the hysterical, not the "cog-wheel" type, *i.e.*, on passive movement, the greater the force used to overcome it, the greater is the resistance.

(*d*) XIV. **Familial Tremor** is rarely encountered. It may be of the "intention" type.

(*e*) XV. In debilitated infants **Spasmus Nutans,** a rhythmic head-nodding, with nystagmus, is met with. The prognosis is good with improvement in general health. Rickets is commonly blamed; cod-liver oil and sunshine, with suitable feeding, may be given with advantage.

(2) *Irregular and spasmodic involuntary movements of groups of muscles occur during rest and are increased by exertion. They are bilaterally distributed in the face, producing grimaces, and tend to be of hemiplegic distribution in the limbs. Voluntary movements are inco-ordinate. The condition is* CHOREA.

Chorea.—Choreic movements are irregular, jerking, wriggling and grimacing involuntary movements. Chorea occurs in association with other neurological symptoms and signs in FRIEDREICH'S DISEASE and

HEPATO-LENTICULAR DEGENERATION. As an isolated nervous phenomenon chorea occurs in six diseases of different etiology.

I. Rheumatic Chorea.
II. Huntington's Chorea.
III. Encephalitis Lethargica.
IV. Congenital Chorea.
V. Apoplectiform Chorea.
VI. Hysterical Chorea.

§ 771. I. **Rheumatic Chorea.**—This is a rheumatic encephalitis, with a tendency to recur and to cause cardiac damage, seen usually in children or young adults between the ages of five years and twenty years, and in girls three times more often than in boys.

Symptoms.—A history of rheumatic "growing pains," recurrent sore throat, rheumatic nodules or erythema, is elicited in most cases. The earliest symptom is (1) emotional instability, and the child becomes fretful, wakeful and easily tired. (2) General motor restlessness ensues, the child drops things or bumps clumsily against the furniture. (3) Spontaneous involuntary movements of groups of muscles appear, hemiplegic in distribution in the limbs, but bilaterally distributed in the face and motor cranial nerves. These jerky involuntary movements appear at rest, or they are superimposed upon voluntary movements, rendering them inco-ordinated. Thus, in picking up a pin, the choreic child will stretch out the hand, but just before the thumb and forefinger can close on the pin, the supinators of the wrist contract suddenly, jerking the hand away from the pin. After several attempts, success may be obtained. (4) In most cases, there is definite general muscular weakness. (5) Excitement and emotional instability persist through the illness, in varying degree. All types of spasmodic movement are seen in the facial, ocular, lingual and bulbar muscles, leading to grimacing, grotesque squinting, dysarthria and dysphagia. The respiratory rhythm is inco-ordinate, owing to affection of the respiratory muscles. The pupils are normal, or are rarely unequal and show hippus. The hand-grip is poorly sustained, and when the upper limbs are outstretched, the wrists become flexed and the fingers hyperextended at all joints (the "choreic hand"). The knee-jerks may become pendular, viz., during the eliciting of the knee-jerk, the quadriceps tendon suddenly contracts and holds the leg suspended in the air for a second before it flops back. Any known abnormality of the knee-jerks may be met with in chorea; they may be diminished, increased, or absent. The cutaneous and plantar reflexes are always normal. Tachycardia and slight cardiac dilatation are common in the first attack. Endocarditis may develop between attacks or during recurrent attacks, and occurs in at least a third of cases. Uncomplicated chorea is an apyrexial disease.

Flaccid Chorea (Chorea Mollis).—Here the movements are absent and the child is brought because "she has suddenly lost the use of her arm or leg," *i.e.*, muscular weakness predominates. Do not fall into the trap of diagnosing hemiplegia, even although the symptoms are hemiplegic in distribution. The plantars are flexor, the abdominals brisk, and careful physical examination will rarely fail to show slight choreic movements in the face or fingers. The paralysis, moreover, is never more than slight, although the flaccidity and lack of spontaneous movement are great.

Chorea Gravidarum.—This is the same disease as rheumatic chorea, met with in

young pregnant women during the first three months of their pregnancy. The disease may be serious, with great motor restlessness and delirium, and is said to be fatal in 30 per cent. of cases.

Etiology.—The disease is an encephalitis, caused by rheumatic infection, affecting the corpus striatum, cerebral cortex and pia-arachnoid.

Prognosis.—Most cases recover in from six weeks to six months. Other severe cases may last longer. Recurrences, with cardiac complications, are always to be feared.

Treatment.—This comprises the following essentials: (1) Absolute rest in bed, with interesting adult companionship and isolation from other children. (2) Protection from injury through the violence of the movements, attained by padding the limbs or arranging pillows round the patient. (3) Systematic over-feeding with milk and farinaceous foods, in addition to the ordinary diet. In the early stages, a rubber tube should be fitted round the tube of a china feeding-cup, which may, otherwise, be bitten off; an enamelled feeding-cup is safer. (4) Of drugs, the best is aspirin, in 10 gr. doses, thrice daily after meals for a child, and 15 gr. doses for an adult. Chloral hydrate, in 3 or 4 gr. doses, at similar intervals, may be used. Fowler's solution probably "cures" chorea by producing a mild arsenical polyneuritis. In maniacal cases, hyoscine hydrobromide gr. 1/100 hypodermically, may be used. Its effects are, however, uncertain. Neither morphia nor any preparation of opium should be given to children. If possible, the child should be rested in bed until all movements have ceased for several weeks. Thereafter, a restricted life should be led for at least six months, with a convalescent holiday and no school. Tonsillectomy should never be undertaken while movements are present. The recovery should be completely consolidated before this is thought of, even when the tonsils appear greatly diseased.

II. **Huntington's Chorea.**—This is a rare, heredo-familial disease, characterised by (1) Bilateral Chorea and (2) Progressive Dementia. It comes on gradually in the fourth, fifth, or sixth decades, and most of the cases finish their lives in mental hospitals. It is essentially a chronic, progressive degeneration, and has nothing to do with rheumatism. When no family history can be traced, the condition is spoken of as *Senile Chorea*. In Senile Chorea, mental changes are slight or absent. In these diseases, the degenerative process affects the cells of the cortex and basal ganglia. Treatment is of no avail.

III. Encephalitis Lethargica (§ 698).—Cases of lethargic encephalitis may manifest themselves first with symptoms of chorea and pains in the limbs. A careful watch should be kept for the advent of ptosis, ophthalmoplegia, or lethargy, which differentiate these cases from rheumatic chorea.

IV. **Congenital Chorea.**—The choreic movements date from birth, or shortly after, and are invariably slowly progressive. They are usually bilateral and more marked on one side than the other. The mentality is often normal. These children may reach adult life. The lesion is in the corpus striatum on the two sides, and may follow icterus gravis neonatorum.

V. **Apoplectiform Chorea.**—This is due to a thrombosis in the neighbourhood of the corpus Luysii (subthalamic body) or substantia nigra. When the patient recovers from the stroke, severe choreiform movements remain, which are strictly unilateral in distribution.

VI. **Hysterical "Chorea"** can usually be easily diagnosed by the absence of choreic movements in the face. These are constant in all other varieties.

(3) *Slow involuntary writhing and stereotyped movements occur in one or more limbs and sometimes produce bilateral grimacing of the face, and dysarthria. The condition is* ATHETOSIS, *and the onset is usually in childhood.*

The slow, writhing movements of **athetosis** are most commonly observed in (1) *Infantile Hemiplegia* (§ 754), due to trauma at birth, encephalitis following an acute specific fever, or polioencephalitis. The movements occur in the hemiplegic limbs, which retain some degree of power. They are spasmodic and initiated by emotion or attempts at voluntary movement. Athetosis is seen also in (2) *Cerebral Diplegia* (§ 762), where it may be unilateral or bilateral in its distribution. Commonly, the movements affect one side of the body more than the other. Cases of (3) **Congenital Bilateral Athetosis** occur without signs of pyramidal disease, and in these cases, there are found degenerative changes and a marbled appearance, to the naked eye, of the caudate nucleus and putamen of the lenticular nucleus. (4) Athetosis, coming on in adult life, is rare and is usually the result of vascular disease, encephalitis, syphilis, or neoplasm affecting the basal ganglia.

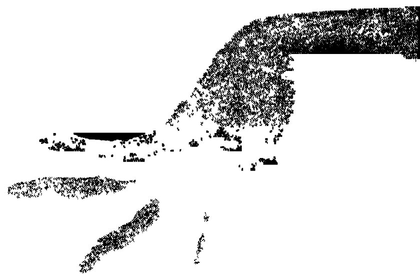


FIG. 177.—ATHETOSIS in the hand of a child suffering from infantile hemiplegia. This figure shows athetosis of the fingers and wrist.

(4) *There are recurrent, involuntary, localised and stereotyped tricks of movement. The condition is* TIC (*including Spasmodic Torticollis*) *or* FACIAL HEMISPASM.

§ **772.** A **Tic** is a spasmodic stereotyped trick of movement of psychical origin. The movements are often of a defensive character, *e.g.*, sudden blinking of the eyes, with or without retraction of the head, wriggling of the shoulder, shaking or tossing of the head. The movements are violent and irregular; they do not interfere with, but are rather improved by voluntary effort. A minor form of Tic is *Habit Spasm*, occurring usually in children between five and ten years of age. Although it may appear that the movements have originated as a habit from some peripheral source of irritation, *e.g.*, refractive errors, coryza, ill-fitting clothes, etc., it cannot be sufficiently emphasised that tics are psychical in origin and are not cured by removal of teeth or tonsils, nor by circumcision. The condition is probably allied to the Obsessive-Compulsive Neuroses (§ 890).

The following types of tic are encountered:

1. *Simple Tic.*—Sudden blinking (blepharospasm), tossing of the chin, grimacing, wriggling, jerky stereotyped movements of the arms or, less commonly, of the legs. Respiratory grunts, barks, sudden explosive laryngeal sounds also occur.

2. *Convulsive Tic.*—The movements are highly complex and purposeful, *e.g.*, "salaaming" or explosive and defensive movements of the whole body.

3. *Psychical Tic.*—There is no spasmodic movement, but the patient is compelled to make an explosive utterance of words or sentences, often obscene. The patient may be a child.

4. *Co-ordinated Tic.*—The patient feels impelled to execute some apparently meaningless, highly co-ordinated act, *e.g.*, twiddling a piece of string, in conditions of mental stress or boredom.

2, 3 and 4 are forms of Obsessive-Compulsive Neurosis. In 2 and 3 serious mental instability is usually present.

5. *Post-Encephalitic Tics.*—Following an attack of Encephalitis Lethargica (§ 698), various respiratory tics are met with, *e.g.*, polypnœa, yawning, even to the extent of jaw dislocation, grunting or barking laryngeal noises. Any form of tic may appear in a patient after this disease. The Post-Encephalitic Tics appear to have an organic origin; some of them, in the young, are possibly associated with the psychical disturbances which follow the malady (see § 907 C).

6. *Post-Choreic Tics.*—After an attack of rheumatic chorea, the patient may be left with a transient simple tic. The prognosis is good.

7. *Spasmodic Torticollis* (§ 773).

Diagnosis.—Tics should not be confused with *Chorea.* In tic (1) the movement is localised and stereotyped, not generalised like the irregular varied movements of chorea, (2) objects are not dropped, and (3) the hand-grip is not irregularly sustained as in chorea. Facial tics are often bilateral and must be clearly separated from Facial Hemispasm (see § 774).

Treatment.— (1) Attend to the general health and hygiene. (2) Psychotherapy may help, even if it is of a very simple kind. Often simple removal of the child from a faulty home or school environment is necessary. (3) Criticism of the habit is to be avoided. (4) Re-educative exercises in front of a mirror, breathing and gymnastic exercises, will help. (5) Aspirin, in gr. 10 doses, thrice daily after meals, is a useful sedative. (6) In severe exacerbations, rest in bed, with complete freedom from emotional strain, should be enjoined. In tics occurring after the age of forty, the outlook is almost hopeless as regards cure. Psychoses may develop in such cases.

§ **773. Spasmodic Torticollis.**—In this disease tonic and clonic contractions of the deep and superficial muscles of the neck occur, causing spasmodic turning of the head to one side and upwards, or there may be tonic extension. It occurs, like other tics, in neuropathic subjects or after encephalitis lethargica. It may result from emotional strain.

Spasm appears first in the sternomastoid and may be tonic or clonic, or both. The contralateral splenius capitis and trapezius are next involved, and, later, the other deep cervical muscles. Bilateral affection of the splenii causes tonic or clonic retraction of the head (retrocollic spasm). True hypertrophy of the affected muscles is frequently seen. The patient may make a "corrective gesture" touching the cheek with a finger or

pushing his chin forwards when the spasm occurs. Spasms are initiated by voluntary effort (*e.g.*, lifting a chair) or emotional causes. Writer's cramp may coexist. The disease, like other tics, is chronic, with spontaneous remissions. It should be distinguished from *congenital wry-neck*, due to birth injury of the sterno-mastoid muscle, which is contractured, with coexisting slight facial asymmetry. In *rheumatic torticollis*, there are no clonic spasms, nor do these occur in *stiff-neck* due to *cervical Pott's disease, retro-pharyngeal abscess,* or *cervical glands.*

Treatment is highly unsatisfactory. Surgical operations (section of the spinal accessory, greater and lesser occipital nerves) meet with temporary, rarely permanent success. After these operations the spasm tends to spread to other muscles. Massage, relaxation exercises and graduated suspension of the neck on spinal apparatus, are tried. In psychological cases, psychotherapy may help. Prolonged sleep, for a period of three weeks, induced by 10-gr. doses of chloral hydrate, six-hourly, has aided some cases.

§ 774. **Facial Hemispasm.**—There is unilateral clonic spasm of the facial muscles with slight facial paresis: although mainly intermittent, persistent tonic spasm may occur. This malady is not a tic and differs from facial tic in that (1) there is always *weakness* of the peripheral type in the facial muscles affected by the spasm, (2) it is always strictly *unilateral*. It is an incomplete form of peripheral facial paralysis and may be seen also in cases recovering from Bell's Palsy. The facial nerve is the only nerve of the body in which this manifestation of incomplete interruption of function occurs. In this way, tumours, such as sarcoma of the skull, or aneurysm of the vertebral artery, syphilitic or tuberculous disease or fracture of the petrous temporal bone, cholesteatomata, or chronic suppurative otitis media, may all cause clonic spasm, with weakness of one side of the face. All the structures, meninges, bone, middle ear, near which the nerve passes, should be suspect.

Treatment is removal of the cause if it can be found. Short of this, the injection of the facial nerve at the stylomastoid foramen with 50 per cent. alcohol, or partial nerve section, has been performed with success. It is best to cut down on the nerve in both operations. A facial palsy results, relieving the spasm, sometimes permanently.

The spasmodic facial movements of *Tic Douloureux* are referred to in § 822.

(5) *There are sudden shock-like contractions of an individual muscle or muscles. The condition is* MYOCLONUS.

§ 775. **Myoclonus** may be confined to a single muscle or, more commonly, the shock-like contractions occur in various muscles throughout the body, with the rapidity of ten to fifty shocks per minute. Myoclonus may be observed in the following conditions: (1) In *Acute Encephalitis Lethargica* (§ 698), the onset may be with myoclonic movements, root-pains and restlessness, or delirium, instead of lethargy. The lethargy, with ocular palsies, may follow. Persistent hiccough may occur, lasting many days. (2) In *Post-Encephalitis Lethargica*, particularly affecting the abdo-

minal or facial muscles. (3) In association with familial epilepsy and dementia, in adolescence—**Myoclonus-Epilepsy**, an excessively rare disease. (4) **Paramyoclonus Multiplex** is another excessively rare disease, in which simple, shock-like myoclonic movements, of proximal distribution, affect symmetrical muscles. It occurs between the ages of five to forty years. Many of the recorded cases are heredo-familial. The disease may be analogous to epilepsy and runs a similar course.

(6) *Flickering or quivering movements of individual muscle bundles are present. The condition is* FIBRILLATION.

§ **776. Fibrillation** may be seen as a transient phenomenon in normal muscles, as the result of (1) *Fatigue* or *Debility* and (2) *Exposure to cold.* When it is persistent and localised, and associated with *muscular wasting,* it *invariably indicates progressive disease of the anterior horn cells.* It is seen in all forms of (3) *Motor Neurone Disease* (see § 788) including *Chronic Bulbar Palsy* (where it appears in the wasted tongue). It may be seen also in (4) *Syringomyelia* (§ 818) and in (5) *Peroneal Muscular Atrophy* (§ 797).

GROUP VIII. TONIC SPASMS OR CRAMPS

When the resistance encountered on passively moving the rigid limb or limbs increases with the amount of force used, the condition is HYSTERICAL SPASM.

§ **777.** In **Hysterical Rigidity** (Hysterical Spasm) the prime-movers and antagonistic muscles contract simultaneously. The distribution of the spasm does not correspond to any anatomical or physiological muscular grouping, and the reflexes are all normal, although the plantars may be absent and an ill-sustained ankle-clonus elicited. Hysterical anæsthesia may coexist. The onset is usually sudden, and the past history may reveal previous hysterical episodes (§ 888). The spasm is often variable.

The patient shows intermittent rigidity in one or more muscles of a limb. The condition is TONIC SPASM *or* CRAMP.

Spasms or **Cramps** are intermittent tonic muscular contractions occurring in one or more muscles of a limb. The spasms may occur (*a*) spontaneously or (*b*) only when a specialised action, *e.g.*, writing is performed, (*c*) only on voluntary movement or (*d*) they may follow on a wound or abrasion. They are met with in the following clinical conditions:

(*a*)
- I. Tetany.
- II. Spastic Paraplegia in Flexion.
- III. Cramp.
- IV. Heat Cramp.

(*b*)
- V. Writer's and other Occupational Cramp.

(*c*)
- VI. Myotonia Atrophica.
- VII. Myotonia Congenita.

(*d*)
- VIII. Tetanus.
- IX. Hydrophobia.

(*a*) *The Spasms occur spontaneously.*

§ 778. I. **Tetany** is a paroxysmal stiffness of the hands, feet, and facial muscles, in association with organic disease, usually outside the nervous system. The chief *symptom* is a paroxysmal stiffness, affecting the forearms, hands and feet (carpo-pedal spasms). The attitude of the fingers, compressed into a cone (the accoucheur's hand), has been emphasised, but other attitudes occur. The paroxysms last from a few seconds to an hour or so, and in severe cases, there is no intervening relaxation. In severe cases, moreover, all the muscles of the body are affected and there may even be opisthotonos. Pressure on the vessels or nerves of a limb can bring on an attack. There is neuro-muscular irritability to percussion and to both faradism and galvanism. If the finger-nail be drawn down the face, a wave of muscular contractility follows it (Chvostek's sign). In children it may be associated with laryngismus stridulus. Many grades of severity are seen ; the disease may only last two days, or two or more months, recovery being the general but not invariable rule.

Etiology.—Tetany may result from disease or damage to the parathyroids, in disturbances of calcium metabolism and in conditions where there is alkalosis. It may be brought on, in the normal subject, by forced deep breathing over a period of some minutes. Clinically, it is met with :

(1) In infants, in association with rickets and cœliac disease.
(2) When the parathyroids are removed or damaged by operations on the thyroid gland. The attacks may last for months but, eventually, become less severe.
(3) In pyloric stenosis, with vomiting. Blood analysis, in these cases, shows a high plasma bicarbonate (alkalosis) and often a high blood urea, with symptoms of uræmia.
(4) In chronic diarrhœa (and especially with spruc).
(5) In chronic interstitial nephritis.
(6) During lactation.
(7) In association with attacks of hysterical or emotional hyperpnœa.

Treatment.—The treatment is that of the associated clinical condition. For cases following thyroidectomy, give up to 30 units of parathormone subcutaneously twice a day in adults. The injection should be controlled by serum calcium estimation. Calciferol by mouth is the most effective remedy in chronic cases. The serum calcium should not be allowed to rise over the normal 10 mg. per cent. The patient should be put on a milk diet and meat should be avoided.

II. **Spastic Paraplegia in Flexion,** due to any of the causes considered in §§ 756, 757, is commonly associated with painful reflex "flexor spasms," in which the legs are suddenly and involuntarily flexed. They occur in the late stages of paraplegia, particularly when the patient is in bed.

§ 779. III. **Cramp.**—Severe muscular cramp may seize one or all the limbs, and even the respiratory and trunk muscles of a swimmer, and prove fatal. Some persons are affected by cramp throughout life on slight provocation, such as lying with the limb in a strained position. Others only suffer from it when there is constipation, a septic focus, diabetes, or when the general health is out of order. In elderly people with arterio-sclerosis or claudication, cramp in the legs may occur at night ; the best remedy is to get out of bed and gently move and rub the limb. Cramp is often a premonitory sign in diabetes and in peripheral neuritis. Localised arterial disease may exist, even in the young, causing cramp.

§ 780. IV. **Heat Cramp** of miners and workers in stokeholds affects the muscles of the limbs and abdomen (§ 508). The temperature may be raised, the pulse rapid. There is loss of fluid and chloride by excessive perspiration, and the cramp appears to be precipitated by drinking a large quantity of water to relieve thirst. A small amount of salt added to the water acts as a prophylactic.

(*b*) *The Spasms appear only during the performance of a specialised action, e.g., writing.*

§ 781. V. **Writer's Cramp** is probably a malady of cerebral origin, unlike ordinary cramp, and consists essentially of (1) spasm, and (2) cramp-like pain in the affected muscles *when writing is attempted.* For all other movements of similar complexity, *e.g.*, shaving, the hand and fingers move normally. It is this specificity that stamps the disease. It affects adults, usually those who write with the intrinsic hand muscles only, not from the wrist, elbow or shoulder. The pen is at first grasped with undue firmness. The onset of spasm and cramp-like pain produces great ataxia in the writing; the pen suddenly flies from or is driven through the sheet. The writing becomes smaller and more undecipherable, and the lines slope upwards or downwards, not running horizontally across the sheet. In diagnosing the malady the physician should be careful not to fall into the pitfall of mistaking early Parkinsonism, a peripheral nerve lesion, or tenosynovitis, for this malady. In these diseases the symptoms are not solely determined by the act of writing.

Treatment.—Absolute rest from writing is essential for not less than six months. During this time the patient should learn to use a type-writer. If the left hand is educated to write, the cramp usually appears in it also. Re-education is attempted with a large pen, or the pen is thrust through a solid rubber ball, which is grasped in the patient's hand. He then learns to write from the wrist, elbow or shoulder, using fresh groups of muscles. In the majority of cases the disease tends to recur, but not invariably.

Occupational Cramps are met with among telegraphists, drapers (in using scissors), cigarette-rollers, musicians, gold-beaters—in short, amongst those following any occupation necessitating the constant repetition of one particular movement.

(*c*) *The Spasms appear only on Voluntary Movement.*

The phenomenon is either *tonic innervation* or *myotonia.* In these conditions there is inability to relax the muscles after a voluntary effort, with peculiar tonic spasm of the muscle. Myotonia can be distinguished from tonic innervation by the associated prolonged dimpling of the muscles on direct percussion, seen in the tongue and skeletal muscles (myotonia on percussion). It occurs in certain muscular diseases. Tonic innervation is met with in organic hemiparesis, due to acute lesions in the upper part of the frontal lobe cortex. It is analogous to the "grasp-reflex" (see § 707, 2).

§ 782. VI. **Myotonia Atrophica** (Synonym: Dystrophia Myotonica) is a rare heredo-familial disorder, developing in early adult life, usually in males. The inherited

factors are: (1) Muscular atrophy, especially of the sternomastoids, facial muscles, and peripheral limb muscles. (2) Muscular weakness, with myopathic facies and falling forwards of the head. (3) Myotonia on voluntary movement, percussion, or faradic stimulation. (4) Dystrophic symptoms, cataract, premature baldness, testicular atrophy, slight mental deterioration.

Healthy children may be begotten by those suffering from the malady, but any, or all, of the inherited characteristics may appear in the family. The disease, like other myopathies, runs a chronic course and may reduce affluent families to conditions of poverty, and at last exterminate the stock. Some cases develop pulmonary tuberculosis.

§ 783. VII. **Myotonia Congenita** (Thomsen's Disease) is probably a variant of Myotonia Atrophica. (1) Hypertrophy of muscles, (2) Muscular weakness, which may be profound, (3) Myotonia, and (4) Slight mental impairment, are the inherited factors. True or pseudo-hypertrophy of muscles may be met with, scattered or generalised, the large muscles contrasting with the patient's poor muscular strength. Myotonia is demonstrated on voluntary effort, not in slight but in more powerful movements. The patient has difficulty in relaxing the grip on shaking hands, cannot open his eyes after he has screwed them up, etc. Violent reflex actions, such as coughing or sneezing, produce transient tonic spasm in the muscles concerned. Myotonic dimpling is present on direct percussion of the tongue or affected skeletal muscles, and myotonic spasm is produced with mild faradic currents. Repetition of the movement tends to abolish the myotonia. The disease is slowly progressive, and death may occur from sudden asphyxia during coughing. Quinine sulphate or bihydrochloride in 5-gr. doses twice or thrice daily, is said to diminish the myotonia.

(d) *The Spasms follow upon a wound or abrasion*—TETANUS *and* HYDROPHOBIA.

§ 784. VIII. **Tetanus** (Lockjaw) is a severe disease characterised by paroxysms of tonic and sometimes clonic spasms, due to the inoculation into a scratch or wound of the tetanus bacillus, the chief habitat of which is highly manured earth and the excreta of herbivora.

Symptoms.—(1) Within 2–10 days after the injury the patient complains of stiffness of the jaw and back of the neck. The incubation period may, however, measure weeks or months in cases who have had a prophylactic anti-tetanic serum injection. (2) Very soon the jaw and neck muscles become rigid. The spasm of the jaw-muscles is known as trismus, or lock-jaw. A similar tonic spasm spreads to all the muscles of the trunk, and in a less degree of the extremities. The back is rigid, sometimes arched in the position of *opisthotonos*, in which only the head and buttocks rest on the bed. Or there may be flexion to one side—*pleurosthotonos*, or bending forward of the body—*emprosthotonos*. The angles of the mouth are drawn down and the eyebrows are elevated—*risus sardonicus*. (3) Reflex spasms supervene from time to time, in which the already rigid muscles become still more contracted, causing agonising pain. The slightest touch may excite these. In severe cases these spasms become more frequent, leading to death from involvement of the laryngeal or respiratory muscles. (4) The temperature may be normal or slightly raised throughout, and may rise to 108° F. just before death. There is often retention of urine. The mind is clear to the last. A local form due to head wounds is described, with paralysis of the facial muscles and difficulty in swallowing. A single case may show (i.) tonic spasms, (ii.) reflex spasms, (iii.) peripheral palsies.

After an injection of antitetanic serum to prevent the disease, temporary *localised spasm* or paralysis may result, *e.g.*, in one arm if the wound is in the hand.

Diagnosis.—In *strychnine poisoning* the muscles relax in the intervals between the spasms, and the spasms involve the extremities to a greater degree. In *meningitis* there is a temperature, and there is no trismus. *Tetany* is not likely to be mistaken for tetanus. In *hysterical opisthotonus* there are other evidences of hysteria.

Trismus is caused also by disease of the pons and *acute bulbar paralysis,* and in association with periostitis of the jaw, disease of the temporo-maxillary joint, or other *local irritation,* such as quinsy or the cutting of a tooth; but the course of the disease serves to differentiate these from tetanus.

Prognosis.—The earlier serum is given, the better the outlook. Fever or rapid onset of symptoms are unfavourable signs. The severity of the case is inversely proportional to the length of the incubation period. With incubation periods of under five days, almost every patient will die in spite of treatment.

Etiology.—The disease is due to the B. tetanus, a spore-forming anærobic bacillus. It may contaminate catgut. Introduced into the body through an abrasion or wound, it produces, locally, a toxin which travels up the axis cylinders of a contiguous nerve and affects the motor cells, spinal or cranial, at first causing local spasm, then general spasm, or paralysis.

Treatment.—(1) *Prophylactic.*—Two injections of tetanus toxoid (1 c.c. each) at a fortnightly interval give active immunity lasting for years. After cleaning suspicious wounds, give 3,000 units of anti-tetanic serum (A.T.S.) subcutaneously, and repeat in seven days. (2) *Curative.*—All dead tissue, blood clot and foreign bodies should be removed from the wound. This helps to prevent gas-gangrene as well as tetanus: some surgeons practice excision of the wound when possible. Keep the patient in a noiseless, darkened room. In view of the excessive muscular action it is essential to feed well: if necessary use the nasal tube. Chloroform or morphine may be used to control severe spasms. Other drugs which control the spasms are curarine (0·1 mgm. four-hourly, increased to 0·3 three-hourly p.r.n.), potassium bromide up to 300 grains a day; large doses of chloretone and avertin per rectum up to 9 times the normal dose. Myanesin (G.1 in 20 c.cs. sterile water) given intravenously or intramuscularly will produce muscular relaxation lasting as long as twelve hours. Details of serum treatment are given in § 521.

§ 785. IX. **Hydrophobia** (Syn.: Rabies) is a fatal disease, transferred to man by inoculation by the infected saliva of an animal (dog, vampire bat, wolves, or other warm-blooded animals) suffering from the disease. It is characterised by spasms and paralysis of the muscles, notably those of deglutition and respiration. The virus travels along the axons centripetally from the area of the bite.

Symptoms.—(1) After an incubation stage, which is generally about six weeks, never less than twelve days, and sometimes as long as twelve to eighteen months or more, there is an insidious onset of malaise, with perhaps slight fever and, sometimes, tingling in the wound. (2) With or without premonitory symptoms, paroxysms of painful spasms of the pharynx supervene, at first brought on by any attempt to swallow, or even the sight of fluid; hence the name hydrophobia. (3) These clonic spasms later become tonic, lasting a quarter to half an hour at a time, and spread to the muscles of respiration and of the neck. The attacks produce excruciating pain and agony of mind. The mind is quite clear, but in the intervals, there are prostration and general hyperæsthesia. (4) Paralysis ensues in three or four days' time, first of the muscles of the lower jaw, and death follows within a week from the onset. Once the disease is established, it is invariably fatal; serum does not cure.

Treatment is entirely prophylactic. Cauterisation of the wound, if performed immediately after the bite, prevents the disease. The immunisation treatment of Pasteur is dealt with in § 521.

GROUP IX. FLACCID PARALYSIS AND MUSCULAR WASTING

Flaccid Paralysis may result from (1) Lesions of the Lower Motor Neurone, (2) Lesions of the Muscles (Myopathy), (3) Lesions of the Upper Motor Neurone in the stage of shock, (4) Hysteria.

(1) **Lesions** of the **Lower Motor Neurone** produce the following CLINICAL SIGNS:

(i.) Flaccid Muscular Paresis.
(ii.) Muscular Atrophy.
(iii.) Diminution or Absence of Tendon Reflexes.
(iv.) Flexor Plantar Response.
(v.) Reaction of Degeneration.

(2) **Myopathic Weakness** produces similar clinical signs. In addition, there may be Pseudo-hypertrophy or Myotonia. The *distribution* of the weakness and wasting is characteristically proximal and bilateral in the shoulder and pelvic girdle musculature, and there is often a family history of the complaint.

(3) **Lesions** of the **Upper Motor Neurone** may produce flaccid paralysis as a transient Shock effect, if the onset has been sudden, *e.g.*, in the acute stages of vascular Hemiplegia or Monoplegia, or in acute spinal cord lesions, such as Acute Myelitis. When the Shock effect has worn off there appear (i.) the exaggeration of the tendon reflexes and (ii.) the spasticity characteristic of Upper Motor Neurone Lesions.

(4) **Hysterical** Flaccid Paralysis is diagnosed by (i.) the paradoxical posture of the limbs, (ii.) presence of normal reflexes, and (iii.) coexistent hysterical anæsthesia (§ 888).

§ 786. THE CLINICAL INVESTIGATION OF A CASE OF MUSCULAR WASTING is made on the following lines: (1) We should first exclude *disease outside the central nervous system*, which may be responsible for the wasting. See section on Causes of Emaciation, § 554. General muscular wasting occurs in cancer, tuberculosis, diabetes, hyperthyroidism, and as the result of starvation. (2) Local muscular wasting, in the neighbourhood of a diseased joint, occurs and is known as *Arthritic Muscular Wasting*, *e.g.*, it is seen in the hand muscles in rheumatoid arthritis. Atrophy of muscles also occurs from disuse, *e.g.*, in a hemiplegic limb—*Disuse Atrophy*. (3) Next we proceed to exclude muscular wasting, due to primary *disease of Muscles—Muscular Dystrophy* (Myopathy). Myopathic wasting is proximal and bilateral in distribution, affecting the shoulder and pelvic-girdle muscles, and sparing the distal muscles of the limbs. It may affect the facial muscles (myopathic facies). In myopathy there may be a family history of the complaint and certain characteristic features may be present—pseudo-hypertrophy or myotonia. (4) Having excluded Muscular Dystrophy, we know the lesion must be *in the Anterior Horn Cell, Anterior Root, or Peripheral Nerve*. In the diagnosis of the lesion the following points should then be considered:

(*a*) *Mode of Onset of the Symptoms*. A *rapid onset*, after a febrile attack, is characteristic of Acute Poliomyelitis (§ 732) and Acute Radiculitis (§ 733). A *more gradual onset* occurs after a febrile attack or sore throat, in Diphtheritic Neuritis and Acute Febrile Polyneuritis. In Motor Neurone Disease and other spinal cord lesions, the *atrophy precedes* the weakness, whereas in Polyneuritis of any causation (*e.g.*, lead) the reverse is true.

(*b*) *Distribution of the Paralysis*.—In muscular atrophy of *Spinal Cord origin*, *e.g.*, Motor Neurone Disease, the paralysis and wasting, affecting chiefly the peripheral limb muscles and the bulbar muscles, are *distal*, often symmetrical, in distribution. In *Root-lesions*, *e.g.*, Radiculitis, the weakness and wasting are of root distribution, *i.e.*, a radiculitis of the C6 root will cause paralysis of the triceps, the flexors, extensors and pronators of the wrist, some of the scapular muscles and the serratus magnus. The symptoms are commonly unilateral. In *Peripheral Nerve Lesions*, the

wasting and weakness will be of peripheral nerve distribution, *i.e.*, a paralysis of the long thoracic nerve (of Bell) will cause isolated paralysis of the serratus magnus, with winging of the scapula. A *unilateral* muscular wasting points to a peripheral cause, and in wasting of the intrinsic muscles of one hand which has persisted for over a year, a cervical rib should be suspected.

(*c*) *Presence of Fibrillation or Tenderness in the Affected Muscles or Palpable Abnormalities in the Superficial Nerves.*—Fibrillation is characteristic of progressive degeneration of the anterior horn cells. It occurs typically in Motor Neurone Disease, but may be seen also, but in slight degree, in other diseases affecting anterior horn cells, *e.g.*, Syringomyelia, Peroneal Muscular Atrophy. Tenderness of muscles to pressure is characteristic of Polyneuritis, due to alcohol, lead, diabetes, arsenic, etc. Myotonia occurs only in the myopathies, and, in combination with muscular wasting of myopathic distribution, in Myotonia Atrophica. *Peripheral Neuromata* may be associated with local wasting and paralysis. Thickening of the peripheral nerve trunks occurs in Hypertrophic Polyneuritis, in Leprosy, and in the exposed part of the ulnar nerve as the result of repeated trauma.

(*d*) *Presence of Extensor Plantar Responses.*—The presence of an extensor plantar response indicates that the disease is in the spinal cord. In combination with muscular atrophy, extensor responses occur in Motor Neurone Disease (including Syphilitic and Lead Amyotrophy) and in Syringomyelia and other central cord lesions, and in Extra-medullary neoplasm and Pachymeningitis.

(*e*) *Type of Sensory Change Present.*—If sensory loss is present, it will be one of these types: (1) In *Spinal Cord Disease*—Sensory loss is of the posterior column type, *i.e.*, defective loss of deep sensibility, sense of position, vibration. "Dissociated anæsthesia" will be present if the cord lesion is central. (2) In *Root-lesions*—Root-pains and sensory impairment of segmental distribution will be present, corresponding to the roots involved. (3) In *Peripheral Nerve Lesions*—The sensory loss will correspond to the cutaneous distribution of the nerve affected. In Polyneuritis it is of the "stocking and glove" type.

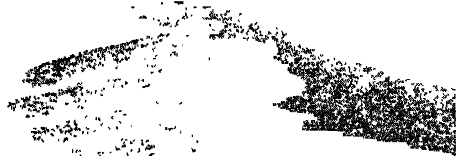


FIG. 178.—ATROPHY DUE TO RIB PRESSURE SYNDROME, showing flattening of the radial side of the thenar eminence, due to wasting of the abductor and the opponens pollicis muscles (C.7). There is slight general wasting of the intrinsic hand muscles. This patient complained of pain shooting down the middle finger of the hand, and numbness and tingling in the two ulnar fingers and ulnar border of the hand.

§ **787**. Let us apply these principles to a given case, taking a patient who presents himself with *a wasted hand.* This may be due to *rheumatoid arthritis.* When the muscular atrophy is of rapid onset and sensory changes are absent, *acute poliomyelitis* should be thought of. Where the muscular atrophy is gradual in onset and accompanied by fibrillation and an extensor plantar response, *motor neurone disease* is likely. When the distribution of the muscular wasting and sensory changes corresponds to the distribution of a peripheral nerve, the condition is *median* or *ulnar neuritis*, and when the distribution of symptoms is that of a nerve root, the condition is due to *infective radiculitis*,

spondylitis, new growth in the spine or brachial plexus, or a *rib pressure syndrome.* When a sensory level is present on the trunk, with root pains and extensor responses, spinal puncture should be performed to exclude *spinal cord syphilis* or *compression.* Dissociated anæsthesia, *i.e.*, impairment to temperature and pain, with touch preserved, is characteristic of *syringomyelia* or a central cord lesion. Where there is a history of *occupational pressure* on the palm of the hand, a neuritis of the deep palmar branch of the median nerve may be the cause of wasting of the thenar muscles. In certain *congenital nervous diseases, e.g.*, peroneal muscular atrophy, Friedreich's ataxia, and myotonia atrophica, wasted hands may be the presenting symptom.

The Causes of Flaccid Paralysis or Muscular Wasting come under these headings:

(1) The symptoms are **purely Motor**: weakness and wasting are present WITHOUT SENSORY CHANGES.
(2) The symptoms are BOTH MOTOR AND SENSORY. See § 793.
(3) The distribution of the weakness and wasting is proximal and bilateral, *i.e.*, MYOPATHIC. See § 804.
(4) The flaccid paralysis is RECURRENT. See § 808.

(1) *The Symptoms are* **purely Motor**: *weakness and* WASTING *are* PRESENT WITHOUT SENSORY CHANGES. The disease may be:

Acute Poliomyelitis	§ 732
Motor Neurone Disease (including Syphilitic and Lead Amyotrophy)	§§ 788, 789
Polyneuritis, due to (*a*) Lead, (*b*) Diphtheria	§§ 790, 791
Acute Radiculitis	§ 733
Post-Herpetic Paralysis	§ 733
Acute Febrile Polyneuritis and Landry's Paralysis	§§ 734, 735
Progressive Spinal Muscular Atrophy of Infants	§ 792

ACUTE POLIOMYELITIS.—In the *paralytic stage* one or more muscle groups are found to have been picked out by the disease. The affected muscles undergo rapid wasting, with partial or incomplete R.D.; the corresponding tendon reflexes are diminished or absent; sensory loss is absent. The wasted limb is often blue and cold and clammy, and in children, may subsequently show defective growth. PERIPHERAL NERVE palsies, similar to Bell's palsy, may occur in purely motor nerves or nerve-roots, *e.g.*, serratus paralysis from affection of the long thoracic nerve. Similar motor palsies, usually of root distribution, may be observed after HERPES ZOSTER.

§ **788. Motor Neurone Disease** (Synonym: Progressive Muscular Atrophy) is a chronic degeneration of the motor nerve-cells with the following characteristics: (1) It has a gradual and insidious onset, occurring in adults at any age, usually after the age of forty years. (2) It begins unilaterally, and later becomes bilateral. The small muscles of the hand begin to waste; more rarely it begins in the shoulder, leg, or bulbar muscles. The wasting precedes the weakness, which is not usually noticed

by the patient until later. (3) Fibrillation is present in the wasted muscles while the disease is active, and is described by the patient as "flickerings or flutterings under the skin." The electrical reactions are a mixture of R.D. and healthy reactions. (4) There is an associated degeneration of the Betz motor cells of the cortex and their axis cylinders; hence extensor plantar responses are present. If the lesion predominates in the upper motor neurone, the tendon reflexes are exaggerated, in spite of muscular wasting. If the lesion predominates in the lower motor neurone, the tendon reflexes are diminished or absent. (5) Aching pains and cramps may occur in the affected limbs, but objective *sensory changes are absent* and sphincter disturbance does not occur. The progress of the disease varies in different cases; where the fibrillation is extensive and marked occurring in muscles not yet wasted, the progress is likely to be rapid, with involvement of the respiratory and bulbar muscles, and death within two years from the apparent onset. The abdominal reflexes tend to be preserved until a late stage of the malady. Where there are wasted hands, with pyramidal signs in the lower limbs, the disease is called **Amyotrophic Lateral Sclerosis.** When it commences in the upper or lower motor neurones of the bulb, it is called **Chronic Bulbar Paralysis** (see § 747).

Diagnosis.—Spinal compression is diagnosed by the characteristic raising of pressure and increase of protein in the spinal fluid. In Motor Neurone Disease the spinal fluid is normal. *Syringomyelia* is distinguished by the characteristic dissociated anæsthesia. *Pseudo-bulbar paralysis* in cerebral arterio-sclerosis has a more gradual onset, with a history of "faints" or strokes, the patients are emotionally unstable, with spastic weeping or laughing: fibrillation of the tongue is absent as a rule, and retinal arterio-sclerosis may indicate disease of the cerebral arteries. In all cases of Amyotrophic Lateral Sclerosis the cervico-dorsal vertebræ should be X-rayed for evidence of subluxation when there is a history of trauma.

§ 789. **Syphilitic Amyotrophy** is due to a pachymeningitis of syphilitic origin, affecting the cervical region. The symptoms are the same as those of motor neurone disease except that: (1) Root-pains may be present, (2) Argyll-Robertson pupils may be found, and (3) the Wassermann reaction is positive in the spinal fluid in all untreated cases. It may coexist with tabes. CHRONIC LEAD POISONING may cause a similar amyotrophy, with extensor plantar responses. There is a history of exposure to lead or its compounds and other signs of lead poisoning (§ 553).

Prognosis.—Remissions, indicated by the cessation of fibrillation, occur, but never improvement. In most cases the disease is fatal in from three to five years.

Treatment.—As the cause of the malady is entirely obscure there is no specific treatment, but rest, warmth, and careful feeding are indicated. Where the bulbar muscles are involved, semi-solid foods are most easily swallowed. In syphilitic amyotrophy anti-specific treatment may arrest the progress of the wasting. Strychnine and massage do not influence the course of the disease; faradism does harm by exhausting already weakened muscles. Careful splinting of the hands and feet at night and daily passive movements will prevent contractures.

§ **790. Lead Neuritis.**—In chronic poisoning with lead a polyneuritis, purely motor, occurs. There is (1) Bilateral wrist-drop, with paralysis of the extensors of the fingers and thumb, *except* the extensor ossis metacarpi pollicis and the supinator longus, which escape. Occasionally, foot-drop occurs, from anterior tibial and peroneal paralysis. (2) Cramps, tremors and pains may be present in the limbs, but no sensory changes. (3) A history of lead colic or lead encephalopathy (headaches, irritability) may be obtained (§§ 246, 553). The disease may occur in infants who lick the paint from their cots. A "lead-line" can be shown at the epiphyses of the long bones radiographically, in such cases. (See also §§ 553 and 829. IV.)

§ **791. Diphtheritic Neuritis.**—Diphtheritic infection produces a symmetrical, flaccid paralysis with loss of tendon reflexes. The *local* paralysis (palate in the case of faucial diphtheria) occurs during the first or second week. The *specific* paralysis, paralysis of accommodation, also occurs early, and often can only be detected by asking the child to read. The *general* paralysis, affecting the periphery of the limbs, appears about the sixth week, when the ocular and palatal paralyses are clearing up. Dilatation of the heart may be present. It is the commonest form of polyneuritis met with in children and is described in § 494. It occurs in association with peripheral wounds and sores infected by C. diphtheriæ.

§ **792. Progressive Spinal Muscular Atrophy of Infants** (Werdnig-Hoffman) is a very rare condition, sometimes familial, in which progressive flaccid paralysis starts in children within the first year. It affects first the trunk and proximal muscles, with abolition of the tendon reflexes, and gradually spreads to the distal muscles. The sphincters are unaffected and there is no sensory disturbance or mental deterioration. The condition is usually fatal before the fifth year. The lesion is an atrophy of the ventral horn cells. Improvement has been reported with parathormone, ♏ 2 b.d. subcutaneously, and suitable doses by mouth of calcium salts and radiostoleum (Warner).

(2) *The* FLACCID PARALYSIS *and* WASTING *are accompanied by objective sensory changes: the symptoms are* **both Sensory** *and* **Motor.** The disease may be:

(*a*) In the upper limbs (wasted hand or wrist-drop).

Tumour of the Cord or Pachymeningitis	§ 757
Syringomyelia and Hæmatomyelia	§§ 759, 818
Acute Radiculitis (motor and sensory root)	§ 733
Brachial Plexus Lesions (including Rib pressure)	§§ 799, 800
Polyneuritis from alcohol, arsenic	§§ 793, 794
Peripheral Nerve Lesions	§ 803
Hysteria	§ 888

(*b*) In the lower limbs (wasted leg or foot-drop).

Peroneal Muscular Atrophy	§ 797
Progressive Hypertrophic Neuritis	§ 796
Lesions of the Cauda Equina	§§ 757, 798
Acute Radiculitis	§ 733
Sacral Plexus Lesions	§ 802
Polyneuritis from diabetes, alcohol, arsenic	§§ 793, 794
Peripheral Nerve Lesions	§ 803
Hysteria	§ 888

In SYRINGOMYELIA (§ 818) and other cord lesions involving the ventral horn cells, the case may present itself with wasting of muscles,

especially the intrinsic hand muscles. Sensibility to pain and to temperature should be carefully tested in order to avoid overlooking the malady in its early stages. In LEPROUS POLYNEURITIS (§ 647) "dissociated anæsthesia" may also occur. Lepra bacilli are present microscopically in large numbers, in the nasal mucosa or skin nodules.

ACUTE RADICULITIS (§ 733) may follow a slight pyrexial attack, unnoticed by the patient. Muscular paralysis and sensory impairment of root distribution, preceded by root-pains, ensue.

§ 793. **Polyneuritis** (Synonym: Multiple Symmetrical Peripheral Neuritis) is a parenchymatous or interstitial inflammation or degeneration of the peripheral nerves, affecting the peripheral muscles of the arms or legs, or sometimes all four limbs and the cranial nerves. The *Symptoms* are usually fairly symmetrical in distribution. (1) The onset is usually gradual, with tingling in the hands and feet, peripheral numbness or painful cramps in the calves. (2) The motor paralysis affects the extensors of the wrists and fingers, producing wrist-drop; or the anterior tibial and peroneal muscles, producing foot-drop. Atrophy follows the weakness and is generally slight. Peripheral diaphragmatic, ocular, facial, or bulbar palsies occur, usually symmetrical. (3) The muscles are often extremely *tender* on palpation. (4) The tendon reflexes are diminished or absent and the plantar responses flexor. (5) There is diminution of cutaneous sensibility to light touches over the periphery of the limbs, of glove and stocking distribution, with hyperæsthesia to pressure, especially on the soles of the feet. (6) Sensory ataxia may occur from impairment of deep sensibility. (7) The affected limbs may be œdematous, blue and clammy, and contractures of the paralysed muscles may occur in chronic cases. (8) Headache, mild mental confusion (even a confusional psychosis) may occur in very toxic patients. (9) The heart is commonly dilated. (10) The *Spinal Fluid* is unaltered in most cases of polyneuritis, but in others it may show the most profound changes. The inflammatory exudate may travel up the nerves into the subarachnoid space, producing increase in protein in the spinal fluid, even yellow coloration (xanthochromia) and massive coagulation. Increase in cells is rare. When the spinal fluid changes are marked, the polyneuritis tends to run an acute and favourable course. In the chronic polyneuritis of lead, mercury and arsenical poisoning, the spinal fluid commonly shows no change from the normal.



FIG. 179.—BILATERAL WRIST DROP, in polyneuritis due to lead poisoning.

Different *types* of Polyneuritis are produced by different *causes*. They may be classified as follows:

Types due to Metallic and Organic Chemical Substances.

I. Alcoholic Neuritis.
II. Lead Neuritis.
III. Arsenical Neuritis.
IV. Occupational Poisoning.

Types due to Infective Toxins or Viruses.

V. Diphtheritic Neuritis.
VI. Leprous Neuritis.
VII. Acute Infectious Fevers.
VIII. Acute Febrile Polyneuritis.
IX. Erythrœdema Polyneuritis.

Types due to Metabolic and Nutritional Diseases.

X. Diabetic Neuritis.
XI. Pernicious (Addisonian) Anæmia.
XII. Allergic Conditions.
XIII. Beri-beri.
XIV. Pellagra.
XV. Conditions of Starvation, *e.g.*, in prisoners of war, in cases of pyloric obstruction, anorexia nervosa, hyperemesis gravidarum, chronic tuberculosis and cancer.
XVI. Immersion in the sea.

§ 794. I. **Alcoholic Neuritis** is usually met with in women ("secret drinkers") and is associated with a peculiar mental state called Korsakoff's Psychosis (see § 899). The paralysis is most marked in the lower limbs; slight bilateral facial weakness may be present. Characteristically, there is anæsthesia of the skin with hyperæsthesia of the calf-muscles and soles of the feet. Œdema and trophic disturbances are marked in chronic cases.

II. LEAD NEURITIS (§ 790).

III. **Arsenical Neuritis** is similar to the alcoholic forms but mental changes are absent. Arsenical pigmentation may be present on the abdomen, with hyperkeratosis of the skin of the palms and soles. It results from therapeutic long-continued administration of Fowler's solution, occasionally from prolonged over-dosage with neoarsphenamine. It may also arise from the drinking of beer, contaminated with arsenic in the process of brewing, or from weed-killer, taken accidentally or otherwise.

IV. OCCUPATIONAL POISONING may cause polyneuritis from *Mercury*, in barometer-makers (mercurial tremor, sponginess of the gums), *Carbon bisulphide, Trinitrobenzol, Anilin*, etc.

V. DIPHTHERITIC NEURITIS (§ 494 and § 791). VI. LEPROUS NEURITIS (§ 647).

VII. ACUTE INFECTIOUS FEVERS.—Besides Diphtheria, Influenza, Pyogenic Infections, Typhoid, Malaria and Dysentery can cause polyneuritis.

VIII. ACUTE FEBRILE POLYNEURITIS (§ 734).—The pyrexia may be very slight and transient, or short bouts of fever accompany extension of the paralysis.

IX. ERYTHRŒDEMA POLYNEURITIS (Pink Disease, § 581) occurs in infants under three years of age. Polyneuritis and intense erythema, with photophobia, characterise the complaint. Later, the skin of the palms and soles desquamates.

X. DIABETIC NEURITIS is characterised by its sensory ataxia and pains in the legs (pseudo-tabes), and the finding of sugar in the urine. Diabetic retinitis may be present. It should not be forgotten that tabes may supervene in a person suffering from diabetes. In diabetes, three other types of neuritis occur. (1) Interstitial neuritis of one or both sciatic nerves (diabetic sciatica), (2) Sudden and painless paralysis of a large nerve trunk (usually the sciatic) from vascular thrombosis of the artery to the nerve. If the sciatic nerve is affected, the ensuing foot-drop does not usually recover. (3) Rarely, external ocular palsies, either unilateral or bilateral. The lătter two conditions occur in arterio-sclerotic diabetes only.

XI. PERNICIOUS ANÆMIA may cause polyneuritis, in addition to the spinal cord lesions (see Subacute Combined Degeneration, § 811).

XII. ALLERGIC CONDITIONS.—Polyneuritis rarely occurs in association with Serum Sickness and Urticaria (§§ 521, 609).

XIII. BERI-BERI, see § 795.

XIV. PELLAGRA may produce polyneuritis in addition to cutaneous and mental and spinal cord symptoms (§ 618).

XV. CONDITIONS OF STARVATION.—In Pyloric Obstruction, Anorexia Nervosa, Hyperemesis Gravidarum, Chronic Tuberculosis and Cancer, polyneuritis may be encountered. Some of these cases are associated with gastric achlorhydria and hypochromic anæmia. A generous protein diet, with injections of vitamin B_1 concentrate, is indicated. The anæmia responds to iron by mouth.

XVI. IMMERSION in the sea for three or four days or longer may give rise to a polyneuritis usually affecting the feet, but occasionally also the hands. The condition is distinct from frostbite and is usually transient and benign (and see § 578).

Diagnosis of Polyneuritis.—The ataxic cases have a superficial resemblance to tabes, but the (1) tenderness of muscles, (2) absence of Argyll-Robertson pupils, (3) absence of sphincter disturbance, and (4) negative spinal fluid findings, usually make the diagnosis from tabes easy (see Table LV, p. 1009). In diagnosing the *cause*, the history of infection or exposure to poisoning is important. In lead neuritis punctate basophilia is present; in arsenical neuritis arsenic may be discovered in abnormal quantities in the urine, or in the hair.

Prognosis.—Only in the infective cases and the polyneuritis of infants can complete recovery be expected.

Treatment.—The cause should be removed. It is believed that alcoholic polyneuritis may be due to deficiency of vitamin B_1, rather than to the toxic effect of the alcohol. Chronic gastritis prevents the requisite absorption of vitamin B_1. In these cases injections of vitamin B_1 extract may help recovery. The patient should be in bed and rest until (1) the heart is normal in size and the exercise tolerance normal, and until (2) recovery is sufficient to enable him to use his paralysed limbs. Light splints are essential to prevent contractures. Passive movements may be employed to prevent contractures; massage only when the muscles have ceased to be tender. Re-educative exercises are of great value during convalescence. Warm baths and aspirin help the pain. Strychnine tonics are indicated. Faradism should never be used.

§ 795. XIII. **Beri-beri** is a vitamin B_1 deficiency disease especially affecting rice eaters in the tropics. It shows dropsy in the legs, and polyneuritis, producing paraplegia.

The *Symptoms* are of two kinds: (*a*) those referable to the neuromuscular system associated with polyneuritis; (*b*) those referable to the cardio-vascular system associated with neuritis of the vagus and sympathetic nerves and œdema of the heart muscle. Either form may occur alone, but generally there is gastro-intestinal trouble and polyneuritis as well as œdema and other evidence of cardiac failure: many cases do not fall into either of these categories. The onset is gradual and the incubation period two to three months.

In (*a*), known as "dry beri-beri," there is no œdema, the dominant features being wasting and weakness of the muscles. The knee-jerks are at first exaggerated, then lost, the calf-muscles are tender and areas of hyperæsthesia and anæsthesia develop. High-steppage gait and wrist-drop occur and the patient cannot rise from a squatting position.

In (*b*), known as "wet beri-beri," various grades of œdema involve the feet, legs, and later the serous cavities. Dyspnœa, dilatation of the right heart associated

with systolic murmurs and perhaps tachycardia, and a rapid, low tension pulse develop. The urine is free from albumen and casts as a rule. Signs of polyneuritis are generally also present.

(c) Acute fulminating cardiac beri-beri (Shöshin) has been studied by Hawes in Malaya. The patient is extremely breathless, with vomiting, epigastric pain and great restlessness, and, unless appropriately treated by intravenous injections of pure vitamin B_1, dies in a few hours. The heart is greatly enlarged, the cervical veins engorged, but the pulse does not often exceed 120–130 per minute. The systolic pressure is well sustained until just before death; the diastolic always drops with the onset of severe symptoms.

(d) Infantile beri-beri is found in breast-fed infants of mothers with latent or clinical beri-beri. It occurs in acute and chronic forms in Japan and the Philippine Islands. Cardiac œdema and gastro-intestinal symptoms such as nausea, vomiting, diarrhœa or constipation are characteristic.

Diagnosis.—Wet beri-beri has to be diagnosed from severe ankylostomiasis, cardiac failure and nephritis, and the dry form from alcoholic and arsenical neuritis, tabes, etc. The dietetic history and the occurrence amongst Orientals of wide-spread polyneuritis should arouse suspicion.

Prognosis.—The fulminating cases of cardiac beri-beri were invariably fatal until recently, when dramatic cure was found to follow pure vitamin B_1 given intravenously. Ordinary cases recover with extra supplies of vitamin B_1 by mouth.

Etiology.—Beri-beri occurs in the rice-eating population of Oriental countries, and is also endemic in Newfoundland and Labrador where white wheaten flour is used. It is due to deficiency in the antineuritic vitamin B_1 which is removed from the rice grain by the polishing process and from flour when refined. The use of undermilled rice and whole wheat flour prevents its development. Recent work has shown that vitamin B_1 influences carbohydrate metabolism and controls the conversion of pyruvate into oxidation products like CO_2 and H_2O: in fulminating beri-beri pyruvic acid increases in the blood, cerebrospinal fluid and urine, especially in the presence of fever, muscular work and increased carbohydrate intake, all of which intensify metabolism; normal levels are restored following the administration of pure vitamin B_1. In some instances, however, disturbances appear to have reached the stage at which recovery cannot be effected merely by supplying adequate amounts of vitamin B_1, and in "dry atrophic" types of beri-beri pyruvic acid may not be demonstrably increased.

Treatment.—Cardiac cases should immediately receive pure vitamin B_1 intravenously in a dosage of 5–10 mg. As much as 50 mg. of the crystalline substance may be necessary. Dramatic cures follow even in profoundly ill patients: venesection may be advisable and the bowels should be kept open with salines. Absolute rest in bed is essential. Later on, and in less severely ill patients, pure vitamin B_1 can be injected intramuscularly in daily doses of 5 mg. or 3 mg. t.d.s. given *per os.* At first small feeds, including marmite, eggs and milk, are given two-hourly, and later a dry diet rich in vitamin and low in carbohydrate. Foods rich in vitamin B_1 are especially indicated; these include yeast, liver, sheep brains, heart muscle, haricot beans, lettuce, wholemeal or brown bread, undermilled rice and rice polishings (§ 928).

§ 796. **Progressive Hypertrophic Neuritis** is a rare heredo-familial disease, producing thickening of the peripheral palpable nerve-trunks, *e.g.*, superficial cervical nerves, with distal atrophy of muscles and sensory loss of posterior column type. It is probably allied to Neurofibromatosis (§§ 647, 803).

§ 797. **Peroneal Muscular Atrophy** is a symmetrical wasting of the distal limb muscles, which commences in childhood in the peroneal muscles and intrinsic muscles of the feet, producing dropping and clawing of the feet (pes cavus). The wasting progresses slowly, inch by inch, up the limbs until the junction of the middle and lower thirds of the thigh is reached, producing thighs like inverted champagne bottles. Similar atrophy occurs in the intrinsic hand-muscles, producing claw-hands, and spreads inch by inch to the upper part of the forearms. Fibrillation may occur in

the affected muscles, but is slight. The gait is high-stepping from dropped-foot, but a great degree of muscular power remains in the wasted muscles owing to the deposition of yellow elastic tissue which follows upon the atrophy. The patients are usually males and the disease may be heredo-familial. These patients survive to adult life and are able to follow useful occupations, and may even play games. All forms of sensation, especially deep sensation, may be impaired in slight degree in the wasted limbs. The ankle-jerks are diminished or lost, later the knee-jerks may diminish or disappear. The plantar responses are lost, owing to the atrophy of the toe muscles.

Flaccid Paralysis, with muscular wasting, occurs in lesions of the I. CAUDA EQUINA, II. PLEXUSES, *or* III. LOCAL PERIPHERAL NERVES.

§ **798.** I. **Lesions of the Cauda Equina** (for their causes see § 757).—The cauda equina consists of L2–S5 nerve-roots, motor and sensory. Sphincter disturbances, retention of urine, with overflow incontinence and dribbling of fæces, are commonly present. Severe root-pains often characterise pressure lesions. The motor symptoms are those of a flaccid paralysis of root distribution, with muscular wasting and diminution or absence of tendon reflexes, commonly bilateral but asymmetrical in distribution. The knee-jerks are commonly abolished. Sensory loss will be of root type (L2–S5), but frequently only the sacral roots are affected, and the anæsthesia is found over the "saddle-area" and over a strip (S2, 1) down the posterior aspect of the lower limbs. When the sacral roots alone are affected, the motor paralysis is confined to the muscles below the knee. The prognosis for recovery is usually poor if the condition is at all established.

II. **Plexus Lesions.**—*The motor and sensory symptoms are of* ROOT DISTRIBUTION.

§ **799.** (1) **Brachial Plexus** (C5–Th1).—Two clinical types occur (*a*) Upper plexus type (Erb-Duchenne) and (*b*) Lower plexus type (Klumpke). In (*a*) the muscles affected are those supplied by C5, 6 roots, viz., deltoid, biceps, brachialis anticus, supinator longus, supra- and infra-spinatus, serratus magnus, subscapularis, latissimus dorsi and the clavicular part of pectoralis major. The scapula is winged, the arm cannot be flexed at the elbow (flexors of elbow) nor raised and abducted (deltoid). The movements of the wrist, and the hand-grasp and finger movements are unimpaired. Sensory loss involves the outer aspect of the shoulder and runs along the outer border of the whole limb. In (*b*) the hand and fingers are paralysed, and the ulnar border of the forearm and hand is anæsthetic from involvement of the C8, Th1 root supply, viz., intrinsic muscles of the hand, and flexors of the wrist and fingers. From involvement of the first thoracic root, oculo-pupillary phenomena may occur (pupillary miosis, enophthalmos and narrowing of the ocular fissure) from damage to the cervical sympathetic fibres.

If the lesion is near the intervertebral foramina, the long (posterior) thoracic nerve (to the serratus magnus) will be involved, with winging of the scapula. This nerve leaves C5–7 roots immediately after their exit from the intervertebral foramina.

The *Causes of Brachial Plexus Lesions* are usually (1) Traumatic crushes or wrenches of the upper limbs, including obstetrical injuries. The roots may be avulsed from the cord. (2) Enlarged supraclavicular glands in malignant disease, tuberculosis, adenitis, lymphadenoma, etc. (3) Acute Infective Radiculitis (see § 733). (4) Cervical rib. (5) Vertebral disease, tuberculous caries, spondylitis or neoplasms. (6) Syphilitic pachymeningitis.

§ **800. Rib Pressure Syndrome.**—Various abnormalities are encountered. There may be (1) an enlarged transverse process of the seventh cervical vertebra, (2) a fibrous band uniting such a transverse process or an abnormal cervical rib to the first rib, or

(3) a cervical rib articulating posteriorly with the transverse process of the seventh cervical vertebra and anteriorly with the first rib. Symptoms are produced by the rib, process or band compressing the C.7 and 8 roots (in a "prefixed" brachial plexus), or by hypertrophy of the scalenus anticus muscle. Although the abnormalities are bilateral and congenital, symptoms are *unilateral* and do not show themselves until adult life. Women are most affected. Neuritic, vascular and cervical sympathetic symptoms are encountered.

Symptoms.—(1) Localised pains and tingling occur along the inner side of the forearm and hand, in the distribution of the first thoracic root. This pain is relieved by raising the arm above the head, thereby relaxing the compressed nerve trunk. (2) Localised wasting of the abductor and opponens pollicis occurs, so that the normal convexity of the thenar eminence is replaced by hollowing of the soft parts and exposure of the metacarpal of the thumb. Whenever localised pain or atrophy of this distribution has existed for over a year in one hand, cervical rib should be suspected. Symptoms occur when the muscular tonus is diminished by fatigue so that the shoulder girdle drops. (3) Coldness of the hand is frequently observed and (4) the radial pulse may be diminished. (5) There may be oculo-pupillary sympathetic phenomena on the affected side. (6) The abnormal rib may be visible or palpable as a bony swelling in the neck. (7) X-rays sometimes reveal the presence of an abnormal rib or transverse process.

Diagnosis.—Cervical ribs occur in syringomyelia, but in these cases the sensory impairment is dissociated and transgresses the limits of the C8 and Th 1 segments. The pain may at first be mistaken for neuritis or fibrositis (§ 824). Antero-posterior, lateral and oblique X-ray views of the lower cervical and upper dorsal vertebræ should be taken in all obscure cases. They may show erosion of the bony pedicles in cases of *spinal neuro-fibroma*; osteo-arthritic changes in the intervertebral foramina in *spondylitis*; a narrowing of the disc space in *prolapsed cervical disc.*

Treatment.—Surgical treatment offers relief, but should be undertaken early the best results are to be obtained. Advanced atrophy will not be cured by operation. The procedure consists of removal of the rib, process, or band, perhaps combined with section of the scalenus anticus muscle.

§ 801. **Obstetric Paralysis,** usually of the upper plexus type, occurs during delivery in head presentations where lateral traction is made on the head, separating it from the shoulder, or in breech presentations when similar lateral traction is made on the trunk, the head being fixed in the pelvis. The attitude of the limb is typical, the arm being rotated inwards at the shoulder (from paralysis of the supra- and infra-spinati), the forearm is extended and the hand pronated (from paralysis of the supinator longus and brevis). It is the attitude of a waiter "receiving a tip." Less commonly the lower type of brachial plexus injury occurs.

§ 802. (2) **Sacral Plexus** (L4–S5).—Lesions of the plexus are relatively rare, owing to its protected situation. The sciatic nerve L4–S3, owing to its size, is the trunk most affected in traumatic and pressure lesions, and its peroneal branch suffers most. The common symptoms are foot-drop, with paralysis of the peronei and dorsiflexors of the foot, and sensory loss over the outer aspect of the leg and dorsum of the foot. The hamstrings are rarely affected.

Causes of Sacral Plexus Lesions: (1) Traumatic, from fracture of the pelvis, with subperitoneal effusion of urine or blood. (2) Pressure Lesions. (*a*) Pressure of the fœtal head in transverse presentations. (*b*) Pressure of pelvic neoplasms or infiltration by malignant or lymphadenomatous glands, or rectal neoplasms. (3) Acute Radiculitis. (4) Sacro-iliac tuberculous caries, spondylitis or vertebral neoplasms.

Prognosis and Treatment of Plexus Lesions.—Unless the roots are partially or completely severed, recovery will occur. Most cases of obstetrical paralysis recover in a few weeks or months; rarely the paralysis is seen in adult life. In traumatic cases surgery is contra-indicated, owing to the fibrosis and distortion resulting from the injury. In no case should an amputation of a useless limb be undertaken until

three years have elapsed from the time of injury. The tendency, in the great majority of slighter cases, is to complete or partial recovery. Treatment should be directed to careful splinting to prevent contractures, re-educative movements and massage.

§ 803. III. Peripheral Nerve Lesions.

The **motor** *and* **sensory** *symptoms are of* PERIPHERAL NERVE DISTRIBUTION. For these sensory and motor symptoms, see Figs. 180 to 183 and Table LIII.

Radial (*Musculo-Spiral*) *Paralysis,* with resulting wrist-drop occurs, (1) from pressure of a crutch in the axilla ("crutch palsy"), (2) from pressure against a hard object in alcoholic sleep ("drunkard's palsy"), (3) after application of a tourniquet ("tourniquet paralysis"), (4) toxic neuritis following an acute infection, (5) chronic lead poisoning (usually bilateral, and the extensor ossis metacarpi pollicis and supinator longus, escape), (6) fracture of the shaft of the humerus or involvement by callus.

Circumflex Paralysis results from injury to the shoulder or toxic neuritis.

Long Thoracic.—Toxic neuritis, radicular brachial plexus injuries.

Median.—(1) traumatic, (2) toxic neuritis following infections. (3) Occupational in hand workers, from pressure of a file, screw-driver, iron, etc., on the palmar branch. Trauma of this nerve may be followed by the burning pain of *causalgia* (see § 824). (4) Thickening of the nerve in the carpal tunnel is described in manual workers.

Ulnar.—(1) Repeated trauma to the nerve in its superficial course behind the external condyle in draymen, etc.; the nerve is palpably thickened. (2) Fracture of the internal condyle. (3) Toxic neuritis following infection (and see § 824).

Femoral.—(1) Fracture of the pelvis. (2) Syphilis. (3) Pelvic neoplasms or glands.

Sciatic.—(1) Prolapsed intervertebral disc. (2) Spondylitis deformans. (3) Cancer of the rectum or pelvic or sacral neoplasm. (4) Fracture of the femur (especially from new growth). (5) Sudden thrombosis of the vessels of the nerve in diabetes. (6) Toxic neuritis following infection. (7) Syphilitic Radiculitis. (8) Compression during pregnancy or parturition. (9) Misplaced intramuscular injections of sulphonamide and certain other drugs in the close neighbourhood of any peripheral nerve trunk may result in complete and permanent palsy.

Phrenic Nerve.—Causes of paralysis are: (1) lesions of the cells of origin, C3, 4, 5 (poliomyelitis, syringomyelia, spinal compression, motor neurone disease); (2) lesions of the roots—pachymeningitis, vertebral caries or neoplasms; (3) lesions of the peripheral nerve-trunk in the neck or thorax (enlarged glands, thoracic aneurysm, mediastinal tumour, pericardial effusion), diphtheritic and other forms of polyneuritis.

Symptoms of phrenic paralysis: (1) Dyspnœa on exertion. (2) During deep inspiration the abdomen does not protrude, owing to paralysis of the diaphragm. (3) Paradoxical diaphragmatic movement is found on radiographic screening.

Prognosis and Treatment of Peripheral Nerve Lesions.—Unless the nerve is partially or totally interrupted by disease or injury, the prognosis is good. The median and ulnar nerves are slow to recover; the musculo-spiral recovers rapidly. If recovery is delayed beyond three months in traumatic cases, nerve-suture may be undertaken *in the absence of all sepsis.* Careful splinting is necessary in all cases, in the position of relaxation of the paralysed muscles, in order to prevent contracture. Re-educative exercises are of the utmost value and massage is useful.

Other peripheral nerve conditions met with are: (1) Simple neurofibromata. (2) Multiple neurofibromata (**Von Recklinghausen's Disease**), a heredo-familial affection associated with (*a*) *café au lait* cutaneous pigmentation, (*b*) sessile or pedunculated tumours, often purplish, on the skin, (*c*) bone cysts at the ends of long bones, (*d*) endocrine disturbance, *e.g.*, dyspituitarism, (*e*) abdominal mesenteric ganglioneuromata, and (*f*) intracranial or spinal tumours. See also § 647.

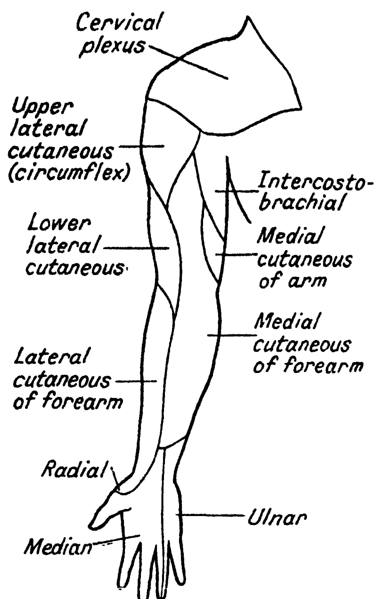


FIG. 180.—ANTERIOR ASPECT OF UPPER LIMB.

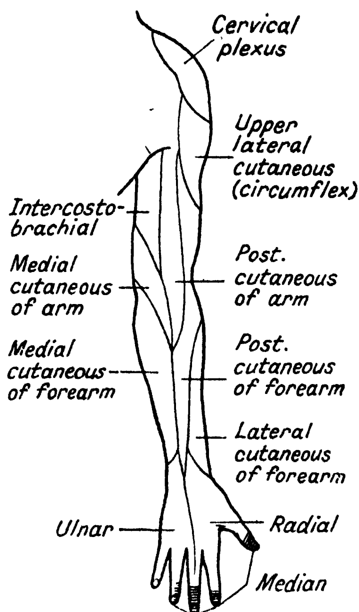


FIG. 181.—POSTERIOR ASPECT OF UPPER LIMB.

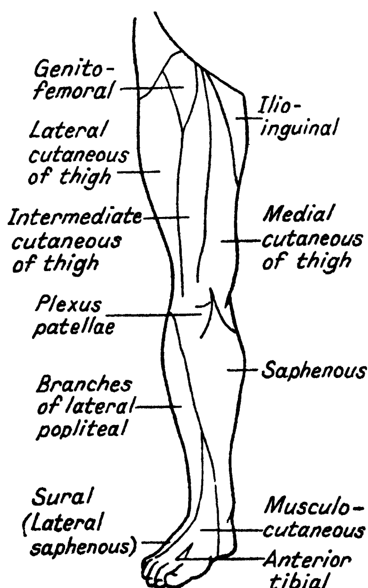


FIG. 182.—ANTERIOR ASPECT OF LOWER LIMB.

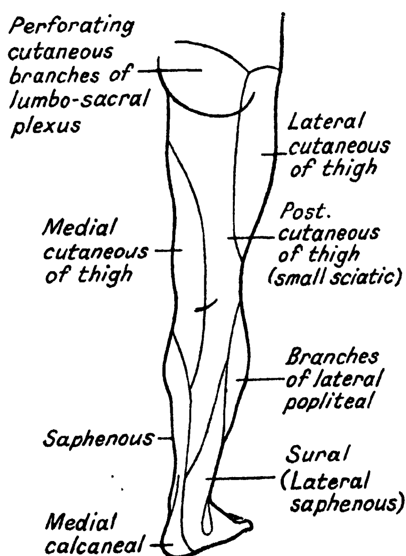


FIG. 183.—POSTERIOR ASPECT OF LOWER LIMB.

Approximate areas of CUTANEOUS SENSATION supplied by peripheral nerves.

TABLE LIII.—PERIPHERAL NERVE LESIONS.

Nerve.	*Muscles Supplied.*	*Defective Movement.*	*Deformity Produced.*
Accessory Nerve.	Trapezius.	Shoulder cannot be shrugged or braced back. Arm cannot be elevated above the head.	Dropped shoulder with "winging" of the vertebral border of the scapula.
Brachial Plexus.	Rhomboids. Serratus anterior. Pectoralis major and minor. Supraspinatus. Infraspinatus. Subscapularis. Teres major Latissimus dorsi.	Loss of adduction and of lateral rotation of arm at shoulder. Arm cannot be raised above horizontal position.	Atrophic shoulder girdle with winging of the vertebral border of the scapula.
Circumflex Nerve.	Deltoid. Teres minor.	Arm cannot be abducted nor elevated backwards or forwards.	Contours of shoulder become flattened; later, shoulder-joint relaxes.
Musculo-cutaneous Nerve.	Coraco-brachialis. Biceps. Brachialis.	Forearm flexed with difficulty in the supinated position.	Depression on outer surface of arm between insertion of deltoid and origin of supinator.
Radial and Posterior Interosseous Nerve of forearm.	Triceps. Brachio-radialis. Extensor carpi radialis longus. Supinator Extensor digitorum. Extensor carpi radialis brevis. Anconeus. Extensor digiti minimi. Extensor carpi ulnaris. Abductor pollicis longus. Extensor pollicis longus. Extensor pollicis brevis. Extensor indicis.	Elbow, wrist and basal phalanges of fingers cannot be extended; grip weakened; impaired flexion of forearm if supinator is involved.	"Wrist-drop," fingers flexed at metacarpo-phalangeal joints; thumb opposed to fingers and depressed somewhat.
Median Nerve.	Pronator teres. Flexor carpi radialis. Palmaris longus. Flexor digitorum sublimis Flexor pollicis longus. Flexor digitorum profundus. Flexor pollicis brevis. Abductor pollicis brevis. Opponens pollicis. Lumbricals I and II.	Power of flexion of hand defective. Ulnar deviation on flexion of hand. Fingers cannot be properly flexed at first inter-phalangeal joint, while flexion of terminal phalanges only practicable in 3 ulnar fingers. Thumb cannot be opposed and its terminal phalanx cannot be flexed.	Wasting of the thenar muscles and of the muscles of the palm of the hand.
Ulnar Nerve.	Flexor carpi ulnaris. Flexor digitorum profundus IV and V. Abductor minimi digiti. Lumbricals III and IV. Dorsal interosseous I. Dorsal and ventral interossei. Opponens digiti minimi. Flexor digiti minimi. Adductor pollicis.	Radial deviation on flexion of hand. Terminal phalanges of the ulnar fingers cannot be flexed nor thumb adducted. Basal phalanges of two ulnar fingers cannot be satisfactorily flexed nor their middle and distal phalanges be extended. Abduction and adduction of fingers impossible.	"Claw-hand" most pronounced in fourth and fifth fingers. First phalanges of fourth and fifth fingers in extreme extension and second and third phalanges flexed; atrophy of hypothenar eminence and interossei.
Femoral Nerve.	Ilio-psoas. Pectineus. Sartorius. Rectus femoris. Vastus lateralis. Vastus intermedius. Vastus medialis.	Inability to flex the hip or extend the knee. Absence of knee-jerk.	Gait disturbed: patient drags leg.

TABLE LIII.—PERIPHERAL NERVE LESIONS (*continued*)

Nerve.	*Muscles Supplied.*	*Defective Movement.*	*Deformity Produced.*
Obturator Nerve.	Adductor magnus. Adductor longus. Adductor brevis. Gracilis.	Adduction, and to slight extent rotation, at hip impaired.	—
Inferior Gluteal Nerve.	Gluteus maximus.	Extension at hip-joint impaired; also abduction.	Pelvis is tilted in walking to swing leg.
Superior Gluteal Nerve.	Gluteus medius and minimus. Tensor fasciæ latæ.	Loss of abduction and circumduction of thigh.	—
Sciatic, Medial Popliteal and Posterior Tibial Nerves.	Semitendinosus. Biceps (long head), and ½ Adductor magnus. Semimembranosus. Gastrocnemius. Soleus. Tibialis posterior. Flexor digitorum brevis. Lumbricals. Accessorius. Flexor digitorum longus. Flexor hallucis longus. Abductor hallucis. Abductor digiti mimimi. All interossei.	Loss of flexion of knee and plantar flexion of the foot and toes. Patient unable to walk on his toes.	Claw position of toes (pied en griffe), pes calcaneus or valgus.
Sciatic, Lateral Popliteal and Anterior Tibial Nerves.	Biceps (short head). Tibialis anterior. Extensor digitorum longus. Extensor hallucis longus. Extensor digitorum brevis.	Foot falls from its own weight, and cannot be raised nor can first phalanx of great toe be extended. Patient cannot walk on heels.	Foot-drop. Toes scrape the floor in walking.
Sciatic. Lateral Popliteal and Musculo-cutaneous Nerves.	Peroneus longus. Peroneus brevis.	Foot cannot be everted.	Foot in varus position.

HYSTERICAL FLACCID PARALYSIS may occur with normal tendon reflexes, "stocking and glove" anæsthesia of the hysterical type, and paradoxical posture of the affected limbs (see § 888).

(3) *The* DISTRIBUTION *of the* WEAKNESS *and* WASTING *is* **proximal** *and* **bilateral.** *A family history of the disease may be obtained and pseudo-hypertrophy or myotonia is present.* The disease is MYOPATHY.

§ **804. Myopathy** (Synonym: Muscular Dystrophy) has a gradual onset in infancy, childhood, or early adult life, and produces symmetrical bilateral wasting and weakness of the *proximal* muscles of the limbs, the muscles of the shoulder and pelvic girdles and the face. The ocular and bulbar muscles are never affected. Fibrillation does not occur. The tendon reflexes are qualitatively diminished in the affected muscles, according to the degree of disease present. The condition is a primary one of the muscles themselves, and no signs of central nervous disease can ever be demonstrated, clinically or pathologically. The malady is usually heredo-familial, and any of the following characters may be inherited: (1) Simple

Atrophy, (2) Pseudo-hypertrophy, (3) True hypertrophy, (4) Myotonia, (5) Slight mental impairment, (6) Dystrophic phenomena, cataract, premature baldness, testicular atrophy. Varying combinations of these characters produce bizarre clinical types. A *distal* type is described but is excessively rare.

There are various clinical types of myopathy which have received special names; much confusion arises in terminology. They may be classified as follows:

TABLE LIV.—CLASSIFICATION OF MYOPATHIC DISEASES (MYOPATHY).

	Synonyms.	*Chief Clinical Features.*
I. Simple Atrophic Myopathy.	(*a*) Juvenile type of Erb. (*b*) Facio-scapulo-humeral type of Landouzy-Déjérine.	Wasting of muscles of shoulder and pelvic girdles. In (*b*) the wasting affects also the face, and usually starts there.
II. Pseudo-Hypertrophic Myopathy.	Pseudo-Hypertrophic Paralysis.	Combination of Pseudo-Hypertrophy with Atrophy.
III. Myotonia Atrophica.	Dystrophia Myotonica.	Myotonia with Atrophy.
IV. Myotonia Congenita.	Thomsen's Disease.	Myotonia with Hypertrophy.
V. Amyotonia Congenita.	Myatonia Congenita. Oppenheim's Disease.	"India-rubber baby." Smallness and extreme flaccidity of muscles.

§ 805. I. **Simple Atrophic Myopathy.**—This is a slowly disabling type, which appears in childhood or early adult life, producing weakness of the shoulder and pelvic girdle muscles, winging of the scapula and lordosis. When the myopathic muscles contract, "cricket-ball hardenings" may be observed in the middle of the muscle, *e.g.*, hamstrings, quadriceps, due to the fact that the dystrophy is most marked at the muscular attachments. Pseudo-hypertrophy is occasionally seen. This type was first described by Erb. Sometimes the facial muscles are affected in addition (Facio-scapulo-humeral type of Landouzy-Déjérine) with weakness of the orbicularis oculi so that the eyes cannot be properly closed, and weakness of the orbicularis oris, producing eversion and thickening of the lips ("tapir-mouth").

§ 806. II. **Pseudo-hypertrophic Myopathy.**—This is a disease of early childhood (onset usually four or five years of age) affecting little boys. Small girls are sometimes affected, but rarely. The disease is heredo-familial, transmitted by the mother, who does not herself suffer from the malady (as in hæmophilia and congenital night-blindness). The patient usually dies before he can transmit the disease, and several brothers in a family, affected in varying degree, are usually seen.

A waddling gait and enlargement of the calves draw attention to the malady. In a few years the child is bedridden. The gait is wide-based and the body is lurched from side to side, with a rolling motion to clear the toes from the ground. There is great lordosis. When the patient is laid on his back on the floor, he rolls over on to his face, gets on to hands and knees and proceeds to climb up his legs by moving his hands alternately upwards on his thighs, to extend his hip-joints. These symptoms are due to weakness of the pelvic girdle muscles, the glutei, hamstrings, and other hip extensors. In the shoulder girdle, the muscles chiefly affected are the lower part of the trapezius and pectoralis major, serratus magnus, and biceps and triceps, producing winging of the scapula and "loose shoulders," the child tending to slip through one's hands when lifted by the axillæ. Pseudo-hypertrophy is most

often seen in the calf muscles and infraspinati, but may occur elsewhere (Fig. 5). Contractures may occur.

III. MYOTONIA ATROPHICA (see § 782).

IV. MYOTONIA CONGENITA (see § 783).

§ 807. V. **Amyotonia Congenita** is a familial and possibly hereditary condition of extreme flaccidity of the muscles, which are small and soft to touch. The onset is probably pre-natal, the mothers of children with this disease usually giving a history of absence of "quickening" at mid-term. The disease is, however, usually not noticed until an attempt is made to teach the child to walk. Some abnormal mode of progression is often adopted by these children, *e.g.*, sitting on the buttocks and "rowing" with the heels. The joints are flail-like and the limbs can be hyperextended into bizarre attitudes. The disease is heredo-familial, but differs from all other myopathies in its tendency to gradual but incomplete recovery. In adult life the muscular power is never normal.

The *diagnosis* from the hypotonia of *rickets* and *diphtheritic paralysis* is made by the history and associated findings. The diagnosis from *cerebellar diplegia* may be very difficult, but the hypotonia of this type of diplegia is not as marked, nystagmus may be present, there is no family history, and diplegia is progressive.

Etiology of Myopathy.—The cause is unknown. The muscular wasting may be an inborn developmental defect (abiotrophy). It is not, however, apparent at birth; the age of onset varies from two to sixty years.

Prognosis.—In nearly all cases, when the disease appears, it is slowly progressive over a number of years. In the Pseudo-hypertrophic variety death occurs in a few years, from broncho-pneumonia or gastro-enteritis, but other varieties are compatible with a much longer life. In Amyotonia Congenita alone the tendency is to improvement.

Treatment.—Massage and passive movements will prevent contractures. Fatigue should be avoided and walking practised daily as long as this is possible. Glycine in doses of 15 grammes twice daily by mouth is claimed by some to effect clinical improvement, but no drug is known which cures.

(4) *The* FLACCID PARALYSIS *is* **Recurrent.**

Certain forms of flaccid paralysis are transient, and, in the intervals between the attacks, the patient is able to execute all voluntary movements in a normal, or almost normal, fashion. Such paralyses are often wrongly diagnosed as hysterical.

§ 808. I. **Myasthenia Gravis** is characterised by rapid development of fatigue and transient paresis in voluntary muscles, without discoverable nervous system disease. Between the attacks, some of the paralyses, especially the external ocular and facial palsies, may persist for years.

Symptoms.—Clinically, the disease is characterised by two kinds of paralysis, "fatigue paralyses" and "permanent paralyses." The bulbar muscles seem to be selectively attacked. The *fatigue paralyses* disappear after a night's rest, but towards the end of the day the waitress may begin to drop trays from her weakening hands, the commercial traveller is aware of dragging in his legs as he climbs the stairs, the lecturer's voice may fall to a whisper, or his articulation becomes indistinct at the end of his discourse. After a short period of rest recovery ensues, but the paralysis recurs on slight exertion. The extrinsic ocular muscles are almost always affected in varying degree, and ptosis, variable squints and diplopia occur. Dropping of the jaw, feebleness of mastication, palatal paralysis, dysphagia and dysarthria, dropping forwards of the head, are all found. The *permanent paralyses* are found in the chronic cases and affect chiefly the ocular and facial movements. The ocular paralyses are

bilateral and complete external ophthalmoplegia may occur. Bilateral ptosis or bilateral lid-retraction and external opthalmoplegia may be found. Some cases of "ophthalmoplegia with exophthalmos" are, possibly, a "forme fruste" of this disease. The pupils, however, react normally. The weakness of the facial muscles is bilateral, and produces weakness of upper and lower facial muscles, with a peculiar "myasthenic smile," the angle of the mouth being drawn upwards and very feebly outwards in a kind of nasal snarl. Repeated faradic stimulation of voluntary muscles causes temporary exhaustion and the muscle ceases to react for some seconds (Jolly's myasthenic reaction). Increased carbohydrate tolerance may be present and creatine may be found in excess in the urine.

Diagnosis depends on the finding of variable paralysis, increasing on exertion, diminishing with rest. The absence of changes in the reflexes (which cannot be exhausted by repeated elicitation), absence of wasting and sensory loss, should be noted. *Any bilateral bulbar paralysis, without wasting, may be myasthenic.* Early cases are often mistaken for hysteria.

Prostigmin test.—4 c.cs. Prostigmin (2 mgm.) combined with atropin sulphate gr. 1/100 is injected intramuscularly. If the paralysis is myasthenic it should gradually commence to disappear, ten to twenty minutes later. The relief from paralysis lasts some hours after the injection.

Prognosis.—Death may occur in a few months from respiratory failure, but sometimes the patient lives for years, free from symptoms.

Etiology.—The disease is not hereditary ; it is commoner in women than in men, and usually shows between puberty and middle life. Occasionally it is found in association with exophthalmic goitre, tumours of the thymus gland, or mediastinal neoplasms. The muscles contain excess of glycogen and small foci of mononuclear cells (lymphorrhages) are found post-mortem in the striped muscles, myocardium and liver.

Treatment.—Ephedrine hydrochlor. (gr. ½ twice or thrice daily) helps mild cases. Many patients can live a reasonably active life whilst on a maintenance dose of prostigmin. The dose and method of administration is a matter for trial in each individual case and should be worked out whilst the patient is in bed at home or in hospital. Pills of prostigmin bromide (each 15 mgms.) are given by mouth—10 to 20 of these may be taken daily. An overdose causes epigastric ache, sweating and blurred vision, but atropin sulphate gr. 1/100 taken by mouth soon abolishes these unpleasant effects. Guanidine by mouth in doses of 50–100 mgms. twice or thrice daily is an additional help. Severe cases require prostigmin and atropin by injection.

When the thymus gland is enlarged benefit may follow from irradiation of the gland with X-rays : in selected cases remissions have been produced by thymectomy, a procedure to be advised with caution. The respiratory and other crises are best treated by prostigmin injection ; the ephedrine treatment may be given to tide over the periods when the patient is having no prostigmin.

§ **809.** II. **Family Periodic Paralysis** is a rare heredo-familial affection, developing in infancy or adolescence, characterised by attacks of paralysis affecting the thoracic and proximal limb muscles, lasting a few hours or days. The paralysis may come on during the night, or during a period of rest, and in the attack, the heart may become dilated, while in the paralysed limbs the tendon reflexes wane and the electrical excitability of the muscles disappears. The disease appears to be connected with the amount and form of potassium salts in the muscle fibres. *Treatment.*—Attacks of paralysis may be prevented by giving orally gr. 30–40 of potassium chloride dissolved in water or milk.

III. Polyneuritis may produce recurrent flaccid paralysis when exposure to the causal agent continues (see § 793). The tenderness of the muscles, sensory changes and diminution in tendon reflexes are diagnostic.

IV. Hysterical flaccid paralyses may recur.—The tendon reflexes are normal and wasting is never more than can be accounted for by disuse.

GROUP X. ATAXIA OR INCO-ORDINATION

The patient, in the absence of paralysis, shows clumsiness, unsteadiness or awkwardness in movements which he could previously execute normally. The GAIT *is* UNSTEADY *or* REELING. The condition is ATAXIA (Inco-ordination). Disorders and examination of the Gait are described in § 705.

A. PAROXYSMAL ATAXIA OR VERTIGO (§ 692).

B. CONTINUOUS ATAXIA may result from disease of the Peripheral Nerves, Spinal Cord, Cerebrum or Cerebellum. There are two main clinical types: 1. Sensory Ataxia and 2. Cerebellar Ataxia.

1. **Sensory Ataxia** is due to interruption of sensory impulses conveying the joint-sense (sense of position and passive movement). With the finger-nose and heel-knee tests the ataxia is increased when the patient's eyes are closed.

2. **Cerebellar Ataxia** occurs in lesions of the cerebellum or cerebellar-fibre systems. With the finger-nose and heel-knee tests the ataxia is uninfluenced by the closure of the patient's eyes.

The two types can at once be differentiated by testing the joint-sense in the fingers and toes. In sensory ataxia this is impaired or absent.

(1) *The patient is ataxic from loss of joint-sense (sense of position).* The condition is SENSORY ATAXIA.

§ **810. Sensory Ataxia.**—The sensory pathway for joint-sense may be interrupted in the Peripheral Nerves, Spinal Cord, or Cerebral Hemispheres. The following table of causes will assist in diagnosis.

(*a*) *Peripheral Nerves.*

I. Polyneuritis (especially diabetic), § 793.

(*b*) *Spinal Cord* (Posterior Columns).

II. Tabes dorsalis, § 817.

III. Subacute Combined Degeneration, § 811.

IV. Disseminated Sclerosis, § 755.

V. Spinal Cord Compression, § 757.

(*c*) *Cerebrum.*

VI. Acute Alcoholic Intoxication, § 899.

VII. Post-Rolandic Monoplegia (due to neoplasm, depressed fracture), § 674.

VIII. Thalamic lesions, § 829.

I. In POLYNEURITIS, especially when due to *Diabetes* (Diabetic Pseudo-tabes), sensory ataxia is present, but in that disease the calves are tender, whilst in tabes they are insensitive to deep pressure. Bladder disturbances are frequent in tabes and absent in Polyneuritis. The finding of sugar in the urine usually clears up the diagnosis, although it should be remembered that diabetics may develop tabes. In Friedreich's ataxia, the ataxic dysarthria, nystagmus and family history with associated extensor plantar responses, claw foot and scoliosis, make the diagnosis clear.

II. TABES DORSALIS (Synonym: Locomotor Ataxy).—This disease, if treatment is to be effective, should be diagnosed in the *Pre-Ataxic Stage.* The presenting symptoms in the early stages are usually the characteristic "lightning-pains." For a complete description of the disease see § 817. In the later stages of the malady the tabetic is *ataxic* because of the loss

of afferent sensory impressions from his muscles and joints. At first there is remarkable compensation for the loss of joint-sense, and the patient, by concentrating his attention on his movements, is able to make great use of his remaining powers. In these cases the ataxia is *latent* but may be revealed by the heel-knee or finger-nose tests when the patient's eyes are shut, or by asking him to walk backwards or sideways, stand on one foot or turn round quickly. In the last stages the ataxia is *manifest*. The gait is wide-based and staggering, support is needed in walking, and the patient holds on to the furniture in turning. In walking, the eyes are fixed on the ground for additional visual control of movement and the legs are lifted high in the air and stamped on the heels, owing to loss of joint-sense. The diagnosis of tabes in this stage rarely presents any difficulty.

§ **811.** III. **Subacute Combined Degeneration of the Spinal Cord** is a disease of gradual onset in middle-aged or elderly persons, usually associated with the blood picture of a megalocytic (pernicious) anæmia (§ 539).

Symptoms.—The patient comes to you for his nervous symptoms and not his anæmia. Only rarely does the disease develop in patients who are being treated for pernicious anæmia. (1) Pins and needles and numbness in the hands and feet are early symptoms. The patient may sit in front of you rubbing his fingers and thumbs together as he tells you of these sensations. (2) Tenderness of the calf muscles is an extremely common early symptom. When it is present, and the plantar responses are flexor, it probably indicates that the disease is in the *polyneuritic stage* and treatment may be curative. (3) Objective sensory impairment to all forms of sensibility occurs, of "stocking and glove" distribution in the periphery of the limbs. Girdle pains round the waist and under the costal margins are not uncommon. Deep forms of sensibility are particularly affected, producing (4) Sensory Ataxia, from loss of joint-sense.

The appearance of extensor plantar responses indicates that the disease is in the *spinal stage*. The tendon reflexes may be increased or diminished, depending on the relative degree of pyramidal and peripheral nerve or posterior column involvement. In the late stages appear hesitancy and dribbling after micturition and overflow incontinence of urine and incontinence of fæces. The gait exhibits a combination of spasticity and ataxia—the spastic-ataxic gait. The toes are dragged, they are lifted jerkily from the ground and the patient staggers when he turns or walks, holding on to the furniture. In the polyneuritic stage, flaccidity of the lower limb muscles is common. The patient, at the end of his malady, may show a spastic or flaccid paraplegia, which renders him bed-ridden.

Mental and other Symptoms.—In association with marked polyneuritic symptoms, and in the late stages of the disease, mild mental confusion may occur. Korsakoff's Psychosis has been met with (§ 899). Transient local œdema of the extremities occurs. Optic atrophy has been described. In the later stages, hæmorrhages occur in the retina, due to the severe anæmia.

Clinical Types.—(1) *Flaccid type*—the muscles of the lower limbs are tender and flaccid, the tendon jerks abolished, with absence of sense of position and vibration in the feet and toes. The symptoms resemble those of polyneuritis, but eventually an extensor plantar response appears. (2) *Spastic type*—the lower limbs show clasp-knife rigidity; increased tendon jerks with clonus, and extensor plantar responses are present. Cutaneous sensibility is impaired in minor degree over the distal parts of the limbs. In the final stages the spastic lower limbs become flexed, and there is incontinence of urine. Both clinical types may be observed as stages in the progress of the disease in the same individual. When the process is most marked in the pyramidal tracts, the spastic picture predominates, when more in the posterior and lateral columns and peripheral nerves, the limbs become flaccid.

The *diagnosis* is usually easy when the patient is of advanced years. Clinical diagnosis should be confirmed by blood-counts, fractional test-meal and examination of the spinal fluid. In view of the high incidence of *carcinoma of the stomach* in cases of pernicious anæmia X-ray examination may have to be made and repeated at yearly intervals until that diagnosis can be excluded. For the differential diagnosis from (1) Friedreich's ataxia, (2) polyneuritis, and (3) tabes dorsalis, see Table LV.

Prognosis.—In some rare cases the onset of the disease is *acute* and rapidly fatal in days or weeks. Usually, however, the disease runs a slowly progressive course, with remissions, over many years. In the early polyneuritic stage the malady may be cured by suitable treatment.

Etiology.—The disease commences in some cases as a polyneuritis ("demyelination neuritis"), but the characteristic lesions are found in the spinal cord, particularly in the posterior columns, the pyramidal tracts and to lesser extent, in the spino-thalamic tracts. The cord lesions are *focal*, with ascending and descending degeneration proceeding from the diseased foci. Subacute combined degeneration of the cord is found also in association with diseases other than pernicious anæmia—(1) The cachectic stage of *gastric carcinoma*, (2) Certain nutritional disorders of severe degree, *e.g.*, *pellagra*, *lathyrism*, *q.v.* A related clinical picture, but due to degenerative changes in the posterior nerve roots is found in some cases of *bronchial carcinoma*.

Treatment.—(1) A high vitamin diet of which whole liver forms an important part should be given. If it can be obtained, and if the patient can tolerate it give lightly cooked calves' liver. Kidney, and calves' brains should if possible be added to the diet. Brewer's yeast or marmite are given. (2) *Liver extract* is given in very large doses intramuscularly, until the red-cell count is over 5 million, and the peripheral paræsthesiæ are beginning to disappear. Doses of 20–30 c.cs. weekly are advised in the first instance. Many consider the cruder liver extracts are more efficacious. After two weeks even in a severely anæmic patient, there should be a gain of 1 million or more red blood cells. The maintenance dose of liver extract is 3–4 times larger than that used to control uncomplicated pernicious anæmia. In cases that show sensitivity reactions to liver extracts, vitamin B_{12} has been shown to be effective. Folic acid has no place in the treatment of subacute combined degeneration of the cord;

TABLE LV.

	Subacute Combined Degeneration of the Cord.	*Polyneuritis.*	*Tabes Dorsalis.*	*Friedreich's Ataxy.*
Age.	Usually middle-aged and elderly.	Any age.	Any age (Juvenile Tabes).	Patient a child or young adult.
Speech.	Speech normal.	Speech normal.	Speech normal.	Ataxic dysarthria.
Sensory symptoms.	Pins and needles in the fingers and feet.	Cramp in legs.	"Lightning Pains."	Painless.
Muscular tenderness.	Muscular tenderness.	Marked muscular tenderness in most cases.	Muscles and tendons insensitive to deep pressure.	No muscular tenderness.
Reflexes.	Plantars usually extensor.	Plantars absent.	Plantars flexor.	Plantars extensor.
Special Characteristics.	Achylia and often picture of pernicious anæmia.	Glycosuria in diabetic neuritis.	Characteristic spinal fluid findings in 65 per cent.	Pes cavus and scoliosis.

this substance whilst perhaps controlling the anæmia will not prevent neurological complications. (3) Iron in the form of Blaud's pill may be given in doses up to 50 gr. daily. (4) 30–40♏ of dilute hydrochloric acid in a wineglassful of water is given with meals if the patient will tolerate it. (5) In bed the patient's feet may have to be lightly splinted to prevent dropping. A bed-cradle is advised. Frenkl's bed and walking exercises will help ataxic patients during convalescence. A semicircular frame on wheels with axillary supports, the so-called "Walking Machine," is valuable in re-educating the patient and giving help and confidence when strength and co-ordination are still poor.

IV. In DISSEMINATED SCLEROSIS, a patch of disease in the posterior columns or fillet will cause sensory ataxia, acute or gradual in onset. Here the diagnosis is made by other symptoms and signs: viz. (1) transient attacks of uselessness in the limbs, transient paræsthesiæ, transient diplopia or blindness, or (2) evidence of disseminated lesions in the nervous system, *e.g.*, pallor of the temporal halves of the optic discs, nystagmus (§ 755).

Treatment of Sensory Ataxia.—After removal or treatment of the cause of the ataxia, much can be done by re-education of existing healthy mechanisms, to improve the residual ataxia. The exercises designed by Frenkl are of great use, but short of these, which need the co-operation of a skilled teacher, much can be done by the patient at home (see § 817). (1) *Bed exercises*—Lying on his bed or a couch, the patient should be taught to bring his great toe accurately on to various squares drawn on a smooth board, vertically facing him. Sitting up, with the board flat, he does the same things with each heel. (2) *Walking exercises*—Now getting up he practises walking on foot-marks chalked on the floor or painted on a strip of linoleum. Later, walking sideways, walking on tiptoe, heeling

and toeing a line are practised. Weighting the boots may give increased security. The hands are trained by means of chess-men or draughts and a draught-board, or with marbles on a solitaire board.

V. Spinal Cord Compression.—Lesions compressing the posterior columns of the cord or the cerebellar afferents cause spastic ataxic gait. Increased spinal fluid pressure, other characteristic findings on spinal manometry, and increased protein in the fluid, are found in such cases.

(2) *The patient is ataxic* WITHOUT *loss of joint-sense* (*sense of position*) *and signs of cerebellar disease are present.* The condition is Cerebellar Ataxia.

§ 812. **Cerebellar Ataxia.**—Lesions of the cerebellum or its fibre systems produce ataxia of voluntary movement, without great weakness and without qualitative changes in the reflexes. In acute cerebellar lesions, marked hypotonia of the voluntary muscles occurs.

The syndrome of disease of the cerebellum or its peduncles is very characteristic. There is loss of tone (*Hypotonia*) in the ipsilateral voluntary muscles, most marked in acute lesions, *e.g.*, gun-shot wounds. The affected limbs, if grasped firmly at the forearm, can often be shaken like a flail. The loss of tone may also be revealed by passive hyperextension of the wrists or elbow joints.

Nystagmus is present with a slow, coarse movement, on looking to the side of the lesion, and a rapid, finer nystagmus on looking from the side of the lesion. This lateral horizontal nystagmus is often accompanied by defective conjugate movement of the eyes, *e.g.*, there may be coarse horizontal nystagmus of the right eye on looking to the right, with defective inward movement of the left eye, or a general unwillingness to look to the side of the lesion. Vertical or rotatory nystagmus on upward or downward movement of the eye occurs in lesions of the vermis. The fixation nystagmus of cerebellar disease is probably dependent on lack of tone in the ocular muscles. A rare phenomenon in acute lesions is "skew-deviation" of the eyes.

Cerebellar attitude.—The occiput on the affected side is turned downwards and the chin tilted to the opposite side. Occasionally this position of the head is reversed. In lesions of the posterior vermis, the head may be tilted backwards, with a tendency for the patient to fall backwards.

Cerebellar Speech.—The articulation assumes a syllabic-staccato quality, or articulation is slurred.

Dysdiadochokinesis.—There is difficulty in performing rapidly alternating voluntary movements, *e.g.*, clenching and unclenching the fingers, shaking the forearms, tapping with the palms of the hands on the knees.

Dysmetria.—The inco-ordination of voluntary movement may show itself by a tendency to overshoot the mark with the finger-nose test.

Past-pointing. (Bárány). The patient is asked to stretch out one arm and point the forefinger. He is then made to move the arm in the vertical plane and with closed eyes bring his finger back to its original position. After a unilateral cerebellar lesion the arm deviates outwards on the side of the lesion.

Gait.—This is staggering or reeling. It may be noticed especially when the patient turns in walking. On attempting to walk in a straight line the patient deviates to the affected side. There is greater difficulty in balancing on the leg of the side affected.

When the upper limbs are outstretched, the one on the affected side tends to fall away or, if it is briskly tapped, it bounces or oscillates abnormally compared with the sound side. Tremor, appearing just before the final execution of a finely adjusted voluntary movement (Intention Tremor) or a static tremor, appearing when the affected limb is held outwards, may be noted.

In lesions of the vermis (*a*) the symptoms tend to be bilateral in distribution

and most markedly in the lower limbs, (b) there is ataxic dysarthria, and (c) retraction of the head may be present if the lesion is far back.

Clinically, Cerebellar Ataxia is met with in the following diseases:

(a) *Diseases of the Cerebellar Fibre-Tracts.*

I. Disseminated Sclerosis.
II. Friedreich's Ataxia.
III. Hereditary Ataxia (Marie).
IV. Thrombosis of the Posterior Inferior Cerebellar Artery.
V. Syringomyelia and Medullary Tumours.
VI. Extra-Cerebellar Tumours.

(b) *Diseases of the Cerebellum itself.*

VII. Hypoplasia—Cerebellar Diplegia, Congenital Absence.
VIII. Senile Cerebellar Atrophy.
IX. Thrombosis and Hæmorrhage.
X. Intra-Cerebellar Tumours, Abscesses and Tuberculomata.

The diseases of the fibre-tracts are differentiated by the "neighbourhood symptoms," involvement of contiguous tracts and nuclei.

I. Disseminated Sclerosis (see § 755).

§ 813. II. **Friedreich's Ataxia** is an heredo-familial disease, coming on gradually in childhood or early adult life, characterised by (1) *Deformities*—pes cavus, hammer-toes (usually bilateral), and scoliosis. (2) *Cerebellar Signs*—Ataxic dysarthria, nystagmus and rhythmical oscillation of the head (titubation) with clumsy ataxia of the limbs. (3) *Pyramidal Signs*—diminished or absent abdominal reflexes, and when the plantar response is observed, it is of the extensor type. The gait is spastic and ataxic and there is associated weakness of the limbs. (4) Although the posterior columns are sclerosed, there is seldom any appreciable loss of sensibility until the late stages of the disease, but the *tendon reflexes disappear early*.

The degeneration affects the spino-cerebellar tracts in the cord, the pyramidal tracts and the cells of Clarke's column; the posterior and antero-lateral columns are sclerosed. The disease progresses very slowly and is compatible with long life, although the patients may become confined to a wheel-chair, or bed-ridden. Contractures may occur and should be prevented, if possible, with massage and remedial exercises. Primary optic atrophy, occurring very rarely, may cause partial or complete blindness. For differential diagnosis, see Table LV, § 811.

III. **Hereditary Ataxia** (Marie) is much rarer and resembles Friedreich's Ataxy in the occurrence of Cerebellar Ataxia, ataxic dysarthria, deformities and pyramidal signs. The differences are three: (a) the tendon reflexes are always exaggerated, (b) optic atrophy is common, and (c) the onset is always in adult life. Occasionally, Argyll-Robertson pupils are found in this disease.

IV. **Thrombosis of the Posterior Inferior Cerebellar Artery** (Synonym: Cerebellar Apoplexy).—The onset is sudden, with vertigo; rarely is consciousness lost. The lesion produces hemiataxy, paralysis of the pharynx, soft palate and vocal cord, with sympathetic oculo-pupillary signs (miosis, enophthalmos and pseudo-ptosis), all on the side of the lesion, with a crossed hemi-anæsthesia of the limbs and trunk, and sensory loss on the ipsilateral side of the face, chiefly to pin-prick and hot and cold. The pyramidal tracts, which are not supplied by this artery, remain intact (§ 684).

V. **Syringo-bulbia and Medullary Tumours.**—By interference with the cerebellar tracts, the pyramidal and sensory projection fibres, the bulbar nuclei and oculo-pupillary fibres, these maladies produce characteristic anatomical lesions, bilateral in distribution. They are slow in onset. Characteristic of syringo-bulbia is the dissociated anæsthesia, and there may be evidence of syringo-myelia, wasted hands, painless whitlows and scoliosis (§ 818). Signs of increasing intracranial pressure, headache and vomiting, accompany medullary tumours. These tumours are rapidly fatal.

§ 814. VI. **Extra-cerebellar Tumour.**—The common lesion is a neurofibroma growing from the sheath of the Acoustic Nerve—*Acoustic Neurofibroma.* The tumour is usually solitary and unilateral, rarely bilateral and sometimes associated with von Recklinghausen's Disease (see § 803). It occurs after the age of thirty years.

Symptoms.—(1) A long-standing history of *deafness* extending over years, of the type of *nerve deafness.* (2) *Tinnitus* has been present intermittently before the patient is seen by the doctor. (3) There is *diminution or absence of the corneal reflex* on the side of the nerve deafness. This symptom merits the closest attention: it is due to pressure upon the sensory root of the trigeminal nerve. (4) In some cases facial hemispasm or peripheral facial weakness, due to involvement of the facial nerve. (5) Cerebellar signs, including cerebellar attitude of the head, appear in the ipsilateral limbs. (6) Caloric and rotation tests (see § 860) show that the vestibular part of the acoustic nerve is completely or partially destroyed. (7) Signs of increasing intracranial pressure—vomiting and papillœdema—are late in appearing. The headache is sometimes early and characteristically localised to the occipital region, with agonising pain and retraction of the head. The spinal fluid shows increase of protein.

Treatment.—These tumours can be removed with success by surgeons skilled in intracranial surgery. Permanent deafness may ensue, but the cerebellar signs are quickly recovered from.

VII. **Hypoplasia** of the Cerebellum occurs in *Cerebral Diplegia,* and *Congenital Absence* is also met with.

VIII. **Senile Cerebellar Atrophy** (Synonym: Olivo-ponto-cerebellar atrophy).—This is a slowly oncoming degeneration of the cerebellar cortex and olives, occurring after the age of sixty years. The symptoms are *purely cerebellar* and the malady is not familial. The disease is slowly progressive. It is, relatively, not uncommon.

IX. **Thrombosis and Hæmorrhage.**—The symptoms are those of acute vertigo in a patient with arterial disease. Acute cerebellar signs with skew-deviation of the eyes develop within a few minutes, the eye on the affected side looks down and in, the other eye up and out.

§ 815. X. **Intra-cerebellar Tumours, Abscesses and Tuberculomata.**—Cerebellar tumours are relatively common in children; they are mostly of two types—(1) The malignant *Medulloblastoma,* growing in the mid-line from the roof of the fourth ventricle and killing the patient within a year. (2) The more benign vascular and cystic *Angioblastoma,* usually in a lateral lobe. This latter is sometimes associated with angioma of the retina and cysts and cavernomas of the liver (Lindau).

Symptoms.—Papillœdema is intense, headache and vomiting occur early, with hydrocephalus in children. The signs are those of a mid-line or lateral lobe cerebellar lesion. Cranial nerve palsies, bilateral sixth, sensory fifth, or bulbar palsies and bilateral pyramidal signs, may be present. In children the knee-jerks and ankle-jerks disappear and there may be great hypotonia.

Treatment is surgical. Preliminary tapping of the ventricles may relieve symptoms until decompression can be undertaken. Tuberculomata are almost invariably fatal from tuberculous meningitis when operated upon. Cerebellar abscesses (see § 737) are usually fatal unless they are near the surface and meningeal adhesions present, when they can be successfully treated surgically, without supervening meningitis.

GROUP XI. PAIN AND SENSORY DISORDERS

§ 816. In this section are considered diseases presenting symptoms of pain or sensory disorder. The investigation of pain has been considered in § 693. Apart from neuralgia (§ 821), the following types of pain in neurological disease can be recognised:

TABLE LVI.

Type of Pain.	*Distribution.*	*Accompaniments.*
(1) Peripheral Nerve Pain.	Follows cutaneous distribution of particular nerve involved. Figs. 180 183.	Paræsthesiæ, often sensory impairment and tenderness of nerve and muscles on pressure.
(2) Root Pain.	Follows cutaneous distribution of corresponding spinal nerve root (see Fig. 176).	Intensified by coughing or sneezing.
(3) Central Pain.	When due to disease of Thalamus is of hemiplegic distribution.	Persistent aching or burning.
(4) Referred Pain.	Referred to cutaneous Root Area corresponding to segmental supply of affected viscus.	Visceral disease, *e.g.*, coronary disease.
(5) Psychalgia.	Of no fixed anatomical distribution.	Psychoneurosis, *e.g.*, Anxiety State or Hysteria.

In the investigation of any pain, make it an invariable rule never to omit a careful local examination of that part of the body to which the pain is referred.

(1) **Pain due to Peripheral Nerve Disease.**—These pains follow the distribution of the particular nerve involved. In simple *Neuralgia* sensory loss and paralysis are absent. In *Neuritis*, or inflammatory disease of single peripheral nerves, the following characteristics are present: (*a*) Severe pain over the cutaneous distribution of the nerve and slight impairment of cutaneous sensibility; (*b*) tenderness of the affected nerve on pressure, and pain when it is passively stretched; (*c*) cutaneous hyperæsthesiæ or paræsthesiæ (sensations of crawling under the skin, numbness or pins and needles) in the distribution of the affected nerve; (*d*) deep tenderness of the muscles supplied by the nerve, slight wasting and perhaps fibrillation. There is never great wasting and there is no true weakness; (*e*) diminution of the corresponding tendon reflexes, in the later stages. For Polyneuritis, see § 793. In *Pressure Lesions* of peripheral nerves there is true muscular paralysis and the wasting is much more profound.

(2) **Root-Pains.**—These follow the cutaneous distribution of the corresponding spinal segments. (see Fig. 176). They are intensified by coughing or sneezing and may be accompanied by tingling or feelings of constriction accurately localised to root areas and hyperæsthesia of the skin to dragged pin over the affected areas. The aggravation of the pain on coughing or sneezing is due to the distension of the pial sheath of the posterior nerve root, occasioned by the raised cerebro-spinal fluid pressure. Such root-pains are met with in *tabes dorsalis*, in *meningeal inflammation*, *e.g.*, syphilitic meningomyelitis, *extra-medullary spinal tumours*, in *spondylitis deformans* (associated with creaking and stiffness in the spine and lipping of the edges of the vertebral bodies seen in lateral radiograms of the vertebræ), in secondary *deposits of carcinoma* in the vertebræ, *Pott's Disease*, and in *herpes zoster* before the vesicular eruption.

(3) **Central Pain.**—(*a*) *Spinal Cord.*—In certain central lesions of the spinal cord (*e.g.*, syringomyelia, intramedullary growths) spontaneous inveterate pain may be met with, possibly due to irritation of the adjacent spino-thalamic tracts. (*b*) *Thalamus.*—In thalamic thrombosis and in neoplasms involving this structure, pain of similar character may be met with. It is of hemiplegic distribution, associated with perverted perception of cutaneous sensibility (hemiplegia dolorosa) and, perhaps, involuntary movements—the "thalamic syndrome" of Déjérine and Roussy. These central pains are quite uninfluenced by treatment.

(4) **Referred Pains.**—Familiar examples of this are met with in the pain occurring in the throat and down the inner side of the left arm in angina pectoris, and in the pain referred to the tip of the corresponding shoulder in diaphragmatic pleurisy, subphrenic abscess, liver abscess, and cholecystitis, etc. In these cases, the pain is referred to the cutaneous root-areas corresponding to the segmental supply of the affected viscus (see Table LVII).

TABLE LVII.—VISCERAL DISEASE IN RELATION TO SUPERFICIAL TENDERNESS OR REFERRED PAIN (SIR HENRY HEAD).

Heart, Ventricle	Dorsal 1 (?), Dorsal 2, 3, 4, 5.
Auricle	Dorsal 5, 6, 7, 8, and (?) 9.
Aortic Arch	Cervical 3 and 4, Dorsal 1, 2, 3, 4.
Dorsal Aorta	Dorsal 5, 6, 7, 8, 9.
Abdominal Aorta	Dorsal 10, 11, 12, and Lumbar 1.
Lungs	Cervical 3, 4, Dorsal 3, 4, 5, 6, 7, 8, 9.
Œsophagus	Dorsal 5, 6, 7.
Stomach	Dorsal 6, 7, 8, 9, 10.
Intestine—	
1. Duodenum to Sigmoid Flexure	Dorsal 10, 11, 12.
2. Rectum	Sacral 2, 3, 4.
Liver and Gall Bladder	Dorsal 7, 8, 9, 10. (Right side.)
Kidney and Ureter	Dorsal 10, 11, 12, Lumbar 1, Lumbar 2.
Bladder	Sacral 2, 3, 4.
Prostate	Sacral 2, 3, 4, Dorsal 10 and (?) 11.
Testicle	Dorsal 10.
Epididymis	Dorsal 11 and 12.
Ovary	Dorsal 10.
Uterine Appendages	Dorsal 10, 11, 12, Lumbar 1, and (?) Lumbar 2.
Cervix Uteri and Lower Segment of Uterus	Sacral, 2, 3, 4.

(5) **Psychalgias.**—These are pains of mental origin; they do not follow anatomical boundaries, are constant and are accompanied by a disordered mental make-up. They are produced by suggestion, are perpetuated by anxiety, and can be influenced by suggestion. Such are the bilateral facial pains following dental extractions, or pains in the body associated with phobia of cancer or tuberculosis.

There are two important diseases, the diagnosis of which depends upon characteristic sensory findings: I. TABES DORSALIS; II. SYRINGOMYELIA. Two other diseases may be considered within this group: III. SYPHILITIC MENINGO-MYELITIS, and IV. HYPERTROPHIC CERVICAL PACHYMENINGITIS.

§ **817. I. Tabes Dorsalis** (Locomotor Ataxia).—Tabes Dorsalis may be defined clinically as a chronic syphilitic disease, characterised by (*a*) Disturbances of sensation, special and general, in the form of lightning pains, paræsthesiæ and anæsthesiæ; (*b*) loss of the tendon reflexes and muscular hypotonia; (*c*) sensory ataxia (symptoms referable to disease of the posterior nerve-roots and posterior columns); (*d*) loss of the pupillary light reflex; and (*e*) disorders of micturition. Muscular power usually remains intact until near the end. Ten years is the average date of onset after the primary sore; it rarely begins within five years after infection.

The *Symptoms*, in tabes, run a very prolonged course over many years and usually last all the patient's lifetime. The development of the disease is most insidious and it may not be apparent until it is well established. There is no disease of the nervous system which is so frequently missed, notwithstanding the fact that it is relatively common. The patient will only rarely come to you for his ataxia. He comes before this because of what he calls "*rheumatism*" (lightning pains), *bladder troubles*, hesitancy or dribbling after micturition, *acute abdominal pain and vomiting* ("crises"), *failure of vision* and *diplopia*, or a *fracture, dislocation* or *swollen joint* (Charcot joint). Your attention is focussed on the local condition and the diagnosis may be missed. Remember that it is possible to make the diagnosis *from the characteristic lightning pains alone* and to confirm it afterwards by further clinical and serological examinations. The symptoms can be discussed categorically in order of frequency of their early appearance.

A. *The Symptoms referable to Disease of the Posterior Roots.*

(1) *Lightning Pains* ("rheumatism") are the commonest early symptoms. They may precede all the other symptoms by years, and occur in no other disease. They may be unilateral or bilateral, and are described as stabbing, burning, tearing or bursting pains. They are commonly in the legs (since tabes usually starts in the lumbo-sacral region) and like "stabs with a knife" seem to *pierce the transverse axis* of the limbs. They are *paroxysmal* and vary with the weather, a point which should not mislead you into the diagnosis of rheumatism. They are diagnosed, not by the severity of the pain, but by (i.) their paroxysmal character, (ii.) their distribution in the legs, especially in the calves and heels, (iii.) their "transverse" direction. They are increased temporarily after intravenous neoarsphenamine B.P. (N.A.B.). Pins and needles, and "girdle-sensations" of constriction round the trunk, occur. (2) *Sensory Impairment.*—All forms of sensibility are affected, cutaneous and deep. Cutaneous sensory impairment occurs as (*a*) a "cuirass" over the thorax and down the inner aspect of both upper limbs, including the ring, and little fingers. (*b*) A "butterfly-area" over the nose and cheeks (masque tabétique of Duchenne), (*c*) on the soles of the feet and perinæum, in sacral tabes. Deep sensibility is affected early; especially common are loss of pain sensibility of the tendo Achillis to pressure (Abadie's Sign), and loss of vibration sense over the tibial malleoli or sacrum. Pressure over the ulnar nerves at the elbow may fail to produce the normal "funny-bone" sensation in the ring and little fingers (Biernacki's Sign) and the external popliteal nerve as it winds below the head of the fibula, may be similarly insensitive (Sarbo's Sign). (3) *Diminution or Loss of Tendon Reflexes and Hypotonia.*—The disappearance of the ankle-jerks (S1, 2) precedes the disappearance of knee-jerks by years, because tabes commences in the sacral roots. The arm-jerks are retained late, except in cervical tabes. The cutaneous and plantar reflexes are normal, unless

tabes is complicated by another form of neuro-syphilis. In uncomplicated tabes the plantars are always flexor. The disease in the posterior roots interrupts spinal reflex-arcs subserving muscular tonus as well as those connected with the tendon reflexes, and hypotonia, with ability to hyperextend the knee-joints or hip-joints into grotesque attitudes, may be present. (4) *Sensory Ataxia* is due to disease of sensory and non-sensory afferents concerned with joint sense and posture. It is usually, in the early stages of the disease, compensated for by the patient, and the ataxia may only be made manifest by the heel-knee or finger-nose test, the patient's eyes being closed. Romberg's sign is fallacious and need not be employed; it may occur in nervous individuals who are organically sound. In the later stages the ataxia is manifest, the gait wide-based, the heels being brought down jerkily with a stamp and the feet lifted high in stepping, owing to errors in projection of the limbs. The eyes are fixed on the ground for additional visual guidance.

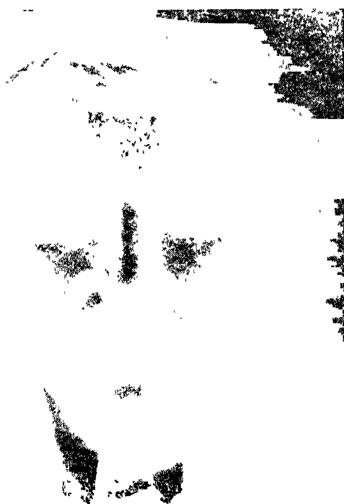


FIG. 184.—TABETIC FACIES, showing bilateral pseudo-ptosis, with compensatory over-action of the frontalis, causing wrinkling of the forehead.

B. *Symptoms referable to other parts of the Nervous System.*

Pupillary and Ocular Phenomena.—(*a*) *The pupils* gradually become smaller and irregular in outline, often oval. They become eccentrically placed, usually to the nasal side of the iris. They are commonly unequal. Miosis is not constant, one pupil may be widely dilated. Later, the pupils, whether dilated or contracted, fail to react to light but preserve the accommodation-convergence reaction—the *Argyll-Robertson phenomenon*; this may occur in large or small pupils, in one eye or both. The consensual reflex may be lost. "Fixed pupils" are also met with, which do not react to light, nor accommodation. (*b*) *Diplopia*, in the early stages, is caused by transient weakness of external ocular muscles and lasts a few days. Later, permanent frank external ophthalmoplegias occur. (*c*) *Pseudo-ptosis* is due to paralysis of the cervical sympathetic, and is bilateral, with compensatory wrinkling of the forehead, producing the "tabetic facies." (*d*) *Optic Atrophy* occurs and usually progresses to complete blindness. For years it may be confined to one eye. The earliest signs are pallor of the disc, with peripheral constriction of the visual fields, when perimetrically charted. Later, the disc shows a typical clear-cut yellow appearance, the colour of candle-

wax (Plates V and VI). Tabetics with optic atrophy rarely show advanced tabetic signs elsewhere (Benedict's Law).

Vesical and Sexual Disabilities.—Hesitancy and dribbling of urine after micturition appear early. Incontinence during the night may occur. Cases of tabes have residual urine which becomes infected, leads to cystitis with ascending pyelonephritis and general debility. Impotence is an early symptom in sacral tabes. Incontinence of fæces may occur, especially after aperients. *Visceral Crises.*—Acute abdominal pain may occur, with vomiting of pints of acid gastric juice, lasting days, and may be mistaken for an acute abdominal catastrophe (§ 242). The pains



FIG. 185.—CHARCOT ELBOW JOINTS, in a case of tabes dorsalis. Patient was the driver of a locomotive and frequently sustained minor injuries to both elbows from the recoil of the brake lever of his train.

of gastric crises are in no way related to food. Crises may occur in other organs, *e.g.*, spasm in the larynx, the colon or rectum, producing tenesmus; or in the bladder, with painful micturition or retention.

Trophic Changes.—*(a) Charcot joints* (§ 591. X).—This arthropathy is probably always occasioned by local trauma. The joint becomes swollen, but pain, heat and redness are absent. Several joints may be affected. Recurrent effusions cause great disorganisation and may produce a flail-limb or genu recurvatum, if the knee-joint is involved. The spine, or the thumb or finger-joints may be affected. Usually it is large joints in the lower limbs (*cf.* syringomyelia, in which the large joints of the upper limbs are similarly affected). Hypertrophic and atrophic changes occur. X-ray shows great rarefaction, irregular large osteophytes, erosion,

linear fractures or a dislocation. (*b*) *Perforating ulcers* occur in the ball of the great toe; they become keratinised and covered over temporarily; then the callosity sloughs out leaving the cylindrical cavity unhealed as before. They may extend into the joint. *Painless ulceration* may occur in the nail-bed.

Tabes associated with other forms of Neuro-Syphilis.—Signs of *G.P.I.* (§ 902) may co-exist, with tremor of the lips and tongue, dysarthria and character-change—*Tabo-Paresis.* Signs of *Syphilitic Amyotrophy* may be present (§ 789) with local wasting of the peripheral upper limb muscles.

VISCERAL SYPHILIS.—Aortitis, thoracic aneurysm, or syphilitic aortic regurgitation, occur in about a third of the cases (§ 80). Recurrent laryngeal paralysis may be present, however, apart from aneurysm (§ 176).

Juvenile Tabes is rare and may be combined with signs of paresis. The symptoms are congenital, but may not show themselves until after the first decade. The younger the patient at the onset of symptoms, the more severe the disease. Choroido-retinitis, bilateral deafness, Hutchinsonian teeth, sabre-shaped tibiæ and rhagades, may be present; otherwise the disease is identical with the adult form.

Diagnosis.—The presence of lightning pains alone is sufficient to make the diagnosis, combined with evidence of past syphilis. Evidence of past syphilis may be obtained by the patient admitting infection (usually imperfectly treated). Patches of leukoplakia on the palate, tongue or cheeks are suggestive, also repeated miscarriages or absence of pregnancy after many years of marriage. The diagnosis rests on clinical grounds and is confirmed by examination of the blood and spinal fluid. The Wassermann reaction is positive in the blood in 65 per cent. of cases, and in the spinal fluid in about the same percentage. The spinal fluid, when positive, shows lymphocytic pleocytosis, 10–80 cells per c.mm. (corresponding to the degree of meningeal inflammation present), globulin increased, protein 0·03 to 0·08 per cent., and a "luetic" type of Lange curve—viz., 1233210000. The *myotonic pupil* (§ 839) with absent tendon reflexes is a benign condition, sometimes confused with tabes dorsalis. In unsuspected *polyneuritis* (alcoholic or diabetic) absent knee and ankle jerks are found: occasionally these reflexes are absent as an isolated and unexplained finding in an otherwise normal individual. For *Differential Diagnosis*, see Tables LV, LXI.

Etiology.—(1) As with General Paralysis of the Insane, tabes usually begins between twenty-five and forty years of age. It is a rare result of congenital syphilis. (2) The majority of cases are males. Husband and wife may both be affected (conjugal tabes). (3) The disease is at first meningo-vascular, and is due to the spirochæta pallida affecting the posterior root between the ganglion and the spinal cord. The sensory fibres are involved earliest in the sacral region. (4) Column degenerations follow, and the posterior column degenerates as high as the nucleus gracilis and nucleus cuneatus; the spino-thalamic fibres being relayed afresh on entering the posterior horn, are unaffected. (5) Similar changes may occur in the optic and other cranial nerves. (6) The Argyll-Robert-

son pupil is thought to be due to degeneration of the colliculo-ocular fibres in the mid-brain.

Prognosis.—Effective treatment of primary syphilis probably prevents the later onset of tabes. Cases treated late may develop tabes or dementia paralytica in spite of treatment. The longer the period elapsing between infection and the first tabetic symptom, the milder will the tabes be. The disease probably never ceases to manifest itself during life, but long periods of quiescence are observed lasting for many years when the patient is untroubled by lightning pains or visceral crises and the signs in the nervous system are at a standstill. The disease appears in these cases to have burnt itself out. The factors underlying periods of activity are imperfectly understood. Activity is possible so long as the spinal fluid shows pleocytosis; but in serologically negative cases symptoms may progress from the effects of intercurrent illness or injury, especially if necessitating prolonged rest in bed. It is relatively easy in most cases by anti-specific treatment to produce a normal or almost normal spinal fluid, but this does not mean that the disease is arrested, or that lightning pains and crises, if ameliorated, have ceased. The blood and C. S. F. Wassermann reaction sometimes remains persistently positive after a full course of treatment. Further treatment of the infection in such cases is not usually necessary.

Ocular palsies may be transient, but optic atrophy almost invariably progresses to total blindness. Bladder infections are a most serious cause of cachexia, and emaciation; and with other intercurrent infections and the aortic complications of the disease are liable to cause death.

Treatment.—(*a*) *Anti-syphilitic Treatment.*—In patients with progressive symptoms, those with pleocytosis in the spinal fluid, or marked changes in the Lange curve, treatment usually causes marked improvement in the patient's general and neurological condition. It is doubtful, in other cases, if anti-syphilitic treatment radically influences the course of the disease. Many benign and quiescent cases have had little or no treatment. In chronic as well as acute cases the possibility of dangerous Herxheimer reactions in the nervous or cardio-vascular systems should be borne in mind. They are less likely to occur if for two weeks before commencing intensive treatment the patient is given by mouth—℞ liq. hydrarg. perchlor. ♏ 30, pot. iod. gr. 12, infus. quassiæ ad fl. oz. ½, thrice daily. Then follow with bismuth and penicillin as described for tertiary syphilis (§ 552)—penicillin must be in doses of at least 10 mega-units in 2 weeks to be effective in neuro-syphilis. Short courses of intravenous N.A.B., say six injections each of 0·15 to 0·45 G., given at weekly intervals every six to twelve months, may diminish a tendency to lightning pains or crises. There may be an exacerbation of the pains during the course of injections, but later they pass off.

In cases of optic atrophy arsenical preparations, especially tryparsamide, should be used with caution. Malarial therapy has been advised to check the progression of the blindness, but it is doubtful if it does so. Similarly,

malarial therapy is sometimes advised in cases which show a paretic type of Lange curve in the spinal fluid, even if, clinically, signs of dementia paralytica are lacking. Paretic Lange curves in tabes may alter as the result of treatment with heavy metals, arsenicals or penicillin, and they do not always portend dementia.

(*b*) *Symptomatic Treatment.—Bladder.*—The patient should be taught to empty the bladder every three hours whether he feels the need or not. Pills of belladonna and ergot may help hesitancy of micturition. Urinary infections may respond to short courses of sulphathiazole given by mouth for five days with plenty of bland fluids. Calcium mandelate 3 G. four times daily, with ammonium chloride in capsules when necessary (§ 412) may be given. Bladder infections are associated with residual urine and with urinary calculi in some cases, and permanent suprapubic drainage may be required. *Lightning pains and crises* are most difficult to treat. Never give morphia or heroin which are liable to produce addiction. Even pethidine may cause addiction. Aspirin gr. 10, or codein gr. ½ repeated as necessary should first be tried: chloretone gr. 10 in a cachet may be given and repeated. Such pains and crises may abate after a short course of N.A.B. Chordotomy is practised for severe lighting pains. *Charcot's Joints.*—The acute stages are best treated by rest in bed. Even large effusions will become absorbed without aspiration. Heavy calipers are of doubtful value and some patients cannot wear them. Using the joint inevitably causes erosion of bone fractures, usually painless and with much bony hypertrophy. An orthopædic opinion should be obtained at an early stage in each individual case. *Perforating ulcers* on the feet will seldom heal until the patient is put to bed for four to six weeks. They should be curetted and covered with strapping having been dressed with gauze soaked in one of the following—zinc and castor oil, red lotion, acriflavine, sulphonamide cream, penicillin cream, sterile paraffin or cod-liver oil. *Ataxia* is markedly improved by a course of Frenkl's exercises.[1]

§ 818. II. **Syringomyelia** usually manifests itself by the appearance of a wasted hand and "dissociated anæsthesia" in a young adult, in association with kyphoscoliosis and other deformities. The disease is very slowly progressive, with intermissions, and extends over many years.

Symptoms.—Five chief groups of symptoms occur: (1) *Sensory Changes*: "dissociated anæsthesia" arises from interruption of the fibres subserving pain, temperature and tactile cutaneous sensibility, as they cross in the anterior commissure of the cord to attain the spino-thalamic tract. Touch has two pathways: thus the fibres for touch and those subserving deep sensibility, which travel up the cord in the posterior columns, are intact. Consequently, over the affected areas the patient is unable to feel pin-pricks and hot objects (often blistering himself with cigarette ends), yet he may be able to feel *light touches* with cotton-wool, and joint and vibration sense are normal. In later stages, all forms of sensibility may be impaired. The distribution of the cutaneous sensory impairment is commonly that of a jacket, as the disease notably affects the cervical enlargement. There is always (*a*) an upper

[1] *The Treatment of Tabetic Ataxia*, by S. H. Frenkl. Heinemann, London, 1917.

and (*b*) lower level to the sensory loss, whether it is unilateral or bilateral. When the enlargement extends high in the cervical enlargement the upper level will be a zone of analgesia on the periphery of the face, due to involvement of the spinal root of the trigeminal nerve which extends as low as the third cervical segment. Impairment of sensibility may also occur over the lower limbs. (2) *Motor Symptoms.*—*Muscular Atrophy* occurs frequently in the intrinsic hand and forearm muscles on one or both sides. "Claw-hand" may be produced or contractures occur with reaction of degeneration in the wasted muscles. *Spastic paralysis* of the legs is a late and inconstant feature, due to compression of the pyramidal tracts as these traverse the affected cord segments. Spasticity of the lower limbs on one side, with impaired cutaneous sensibility on the contralateral lower limbs (Brown-Séquard phenomenon), occurs if the lesion extends laterally. (3) *Trophic and vaso-motor changes* occur: (*a*) Blue œdema of the hands, with diffuse thickening of the subcutaneous tissues (*main succulente*) is found. (*b*) The hands and fingers are often scarred, necrosed and blistered, or ulcerated, as the result of painless whitlows, infected abrasions and burns. (*c*) Trauma of the shoulder, elbow, wrist or finger-joints leads to the production of painless arthropathies (Charcot joints). (4) *Skeletal deformities* are extremely common: (*a*) kypho-scoliosis, from weakness of the spinal muscles, (*b*) pes cavus, (*c*) spina bifida, (*d*) cranial asymmetry, or (*e*) acromegaly (rare). (5) *Cervical Sympathetic involvement* (C8 and Th1), with enophthalmos, miosis, pseudo-ptosis and narrowing of the ocular fissure on one side. Owing to vaso-dilatation on the affected side of the face, the eyebrow, moustache and beard may grow more profusely on that side. Any known neurological symptom may occur in syringomyelic patients. Fits, tetany, fibrillation, hydrocephalus, retrobulbar neuritis, etc., have all been described.

Cerebro-spinal Fluid.—In most cases there is no alteration from the normal, or else protein is present in slight excess. Heavy excess of protein suggests spinal tumour. When the lesion extends to the bulb (*Syringobulbia*, § 813. V.) there may be: (1) Nystagmus (from involvement of the posterior longitudinal bundle), (2) Hemiatrophy of the tongue, with palatal, pharyngeal and laryngeal palsies (from involvement of the bulbar nuclei), and (3) Sensory analgesia over the peripheral area of the face (from involvement of the descending sensory root of the trigeminal).

The *Diagnosis* depends on the finding of "dissociated anæsthesia" with muscular wasting and skeletal deformities. The differentiation from other conditions producing wasted hands has been considered in § 786. *Cervical* rib (§ 800), either unilateral or bilateral, is a common abnormality found with syringomyelia. When, in a case thought to be cervical rib, any physical sign is found *outside the territory of the upper limbs and neck*, the correct diagnosis is probably syringomyelia. The finding of dissociated sensory loss on the trunk, or alteration in the abdominal or plantar reflexes in such a case, indicates syringomyelia.

Prognosis.—The disease runs a very slow but progressive course; the patient may live to an old age or may die of some intercurrent disease. If the disease extends rapidly upwards, death may occur from respiratory paralysis; or a sudden hæmorrhage into a cavity may cause rapid increase in the severity of the symptoms (*Hæmatomyelia*, § 759). With spastic paralysis of the legs and sphincter disturbance there may be great incapacity, but in most cases the incapacity is only moderate.

Etiology.—The disease is probably congenital, but the symptoms are rarely noticed first before the age of fifteen years or over the age of thirty. Two-thirds of the cases occur in males. Familial cases are rare but are recorded. To the naked eye, the cord on section shows a central gelatinous mass of glial tissue, containing an irregular cavity, usually most marked in the cervico-dorsal and lumbar enlargements; it may extend into the floor of the fourth ventricle or mid-brain. Surrounding normal structures are compressed by the enlarging cavity and proliferating cells.

Treatment.—Protect the affected hands from changes of temperature and from trauma. Splinting at night prevents deformities from contracture. The postural scoliosis is improved by appropriate exercises. Deep X-ray to the cervical spine may relieve the aching pain of the malady, but is not curative.

HÆMATOMYELIA or hæmorrhage into the substance of the spinal cord is considered in § 759.

§ 819. III. **Syphilitic Meningo-myelitis** produces pain in the back, root- and girdle-pains, and numbness in the limbs. The signs are those of spinal cord compression, and the onset of these may be sudden or gradual and may simulate cord compression from spinal tumour or vertebral disease. Argyll-Robertson pupils may be found. The diagnosis is made by examination of the spinal fluid. Lymphocytes are present in excess, according to the degree of the meningeal reaction (10–50 cells per c.mm.). Pleocytosis is greatest in acute cases (50–500 cells per c.mm.). The total protein and globulin are increased and the Wassermann Reaction is positive in 80 per cent. of cases. The Lange curve is constantly of the luetic type, viz., 1122211000.

§ 820. IV. **Hypertrophic Cervical Pachymeningitis** is another syphilitic malady producing severe root-pains, radiating down the upper limbs and across the neck, with wasting and weakness of the hand and forearm muscles, sensory changes and alteration in the tendon reflexes. In the later stages the legs become spastic. Protein is greatly increased in the spinal fluid, but cellular increase is slight or absent. The Lange curve is of the luetic type and the Wassermann reaction is often strongly positive.

For *Treatment* of these conditions, see §§ 552 and 817.

PAIN IN THE HEAD, FACE AND NECK

HEADACHES AND PAINS IN THE FACE due to Migraine, Ciliary Migraine, Trigeminal Migraine, Supraorbital Neuralgia, Sinusitis, Intracranial Tumour, Anxiety States, etc., are described in §§ 695, 696.

§ 821. **Neuralgia** is a term employed to describe shooting, radiating pains in the territory of a peripheral nerve, or spreading wider than this. It is more frequent in the upper part of the body. In *neuralgia* there is never any sensory loss, nor alteration in the tendon reflexes, as contrasted with *neuritis*, or inflammation of a peripheral nerve, in which these findings are common. Trigeminal neuralgia (§ 822), migrainous neuralgia, ciliary neuralgia (§ 696) and glossopharyngeal neuralgia (§ 823) are believed to be diseases *sui generis*. In other cases neuralgia occurs: (1) following inflammation of a nerve (*e.g.*, post-herpetic neuralgia, neurofibrositis), (2) in pressure lesions of a nerve, (3) following trauma of a nerve (*e.g.*, causalgia), (4) reflexly, as in acute pulpitis affecting a tooth, or (5) in the psycho-neuroses and hysteria. *Facial neuralgia* may be accompanied by salivation, lachrymation, flushing and even œdema or local greying of the hair. *Post-Herpetic Neuralgia.* In the elderly and aged the pain of an attack of herpes zoster (§ 826) may persist, particularly after herpes ophthalmicus. The condition is distinguished from other root pain by the history of vesiculation and the presence of scars and sometimes pigmentation. The severity and persistence of the pain may make life a burden.

DENTAL NEURALGIA is described in § 205.

Treatment.—The treatment of neuralgia is the treatment of its cause. The pain may be relieved by aspirin gr. 10, veramon gr. 6, or antipyrin gr. 10, or by painting the affected area when it is below the eye with chloral hydrate, menthol, and camphor āā gr. 60. Sedative galvanism is useful in some cases, but a thorough search must be made first for an irritative focus. Post-herpetic pain is little influenced by sedatives. Opium or morphine should never be given for this or any other neuralgia. In early cases X-ray irradiation of the affected nerve roots may be helpful. More chronic cases have been treated by section or alcohol injection of nerve roots and ganglia, even chordotomy to relieve pain.

§ 822. **Trigeminal Neuralgia** (Synonym : Tic douloureux) is a complex neuralgia involving three groups of nerves, characterised by (1) paroxysms of intense pain in the distribution of one or more divisions of the *fifth nerve*, (2) clonic twitchings of the facial muscles on the side affected (*seventh nerve*), and (3) unilateral lachrymation, rhinorrhœa or sialorrhœa and flushing of the face during the paroxysms of pain (*cervical sympathetic*). It runs a cyclical course, with intermissions over a great number of years, and differs from other neuralgias in that no structural lesion of the trigeminal nerve ever develops.

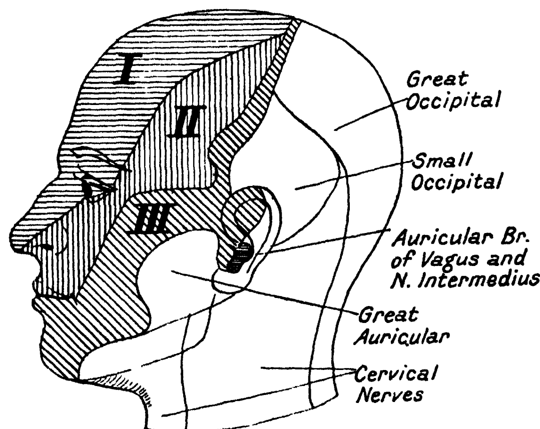


FIG. 186.—CUTANEOUS SENSORY SUPPLY of the face, scalp and neck. The distribution of the trigeminal nerve is shaded.

Symptoms.—The pain commonly commences in the second division, less often in the third (see Fig. 186) and rarely in the first division. The pain is described as stabbing, " like red-hot needles or wires," " like an explosion of fireworks on my face," and is one of the most severe pains that an individual may be called on to experience. It is brought on by cold air on the cheek, talking, washing the face, eating, wearing dentures, even a sudden jar when walking. Pressure over certain " trigger-zones," the red margin of the lip, the angle of the ala nasi and cheek, may

start off paroxysms of pain. Such sufferers will leave off wearing their dentures, they walk rigidly lest a jar should start a bout of pain, they speak without moving the jaws and indicate the site of the pain with the forefinger held over the cheek but rarely touching it. The cheek or eyebrow on the affected side may show an accumulation of desquamated epithelium and dirt on that side, from lack of washing, and in third-division neuralgias unilateral furring of the tongue may be present. In a severe bout, lasting two or three weeks, the patient may waste rapidly from lack of food and sleep. At first, paroxysms extend over some weeks, at intervals of some months. Later, years may elapse between the paroxysms, and the pain lessens in intensity.

Diagnosis.—The malady should never be diagnosed unless (1) the pain is confined to the anatomical boundaries of the fifth nerve, (2) the pain is strictly unilateral, (3) no objective impairment of sensibility is present, (4) cranial nerve palsies are absent. The explosive character of the pain and its strict localisation to the anatomical distribution of the divisions of the fifth nerve affected is characteristic. For example, in a second division neuralgia, the pain is felt in the cheek, on the upper lip and alveolus, as far as the mid-line and in the hard palate up to the mid-line. The corneal reflex is intact, cutaneous sensibility is normal, the masseters and temporals contract normally unless there has been some previous destructive operation on the nerve. When objective sensory loss is present, suspect growth at the base of the skull or gummatous meningitis. Bilateral pains in the face are commonly hysterical. Trigeminal migraine has been described in § 696.

Etiology.—This is a disease of the aged, although it occurs rarely during middle life or in young adults. The average age of onset is fifty years. The writer has seen it come on in a woman of ninety-one years. Both sexes are equally affected. A few cases are recorded associated with disseminated sclerosis. It may develop in completely edentulous patients; the pathology is obscure.

Treatment.—(*a*) In the slighter paroxysms the patient should be advised to remain indoors in a uniform temperature and to take semi-solid food through a glass tube or straw. The following mixture should be prescribed: Tinct. gelsemium ♏15, sodium bromide gr. 10, liquor arsenicalis ♏ 2, in peppermint water thrice daily. In more severe cases, sodium phenobarbitone, gr. ½, may be added to this mixture. (*b*) In other cases, the maxillary and mandibular divisions of the nerve may be injected with 90 per cent. alcohol (1–2 c.c.) at the foramen rotundum and foramen ovale respectively. Successful injection produces anæsthesia and relief of pain for one to three years. Injection of the supra- or infra-orbital and mental foramina affords relief for lesser periods. If the Gasserian ganglion itself is injected or removed there is liability to the formation of neuro-paralytic keratitis or corneal ulceration. Alcohol injections should never be undertaken except by an expert, owing to the danger of producing retrobulbar neuritis, facial palsy or otitis media and deaf-

ness, from misdirection of the injection. (*c*) Surgical treatment consists in fractional division of the sensory root at its exit from the pons, or section of the bulbo-spinal trigeminal tract within the pons (Sjöqvist). The latter operation destroys pain and temperature sensation, chiefly over the distribution of the first and second divisions, touch being preserved.

§ 823. **Dental Causalgia.**—The symptoms follow upon dental operations and are probably due to involvement of filaments of the trigeminal nerve in scar tissue. The symptoms simulate trigeminal neuralgia and the pain is always *burning, continuous* and superficial in character. In distribution, it exceeds the anatomical distribution of the fifth nerve and psychological factors are commonly present. Objective sensory impairment is never found. Such cases should never be injected with alcohol or operated upon. Cold compresses or hot fomentations of opium and belladonna liniment are useful, with bromide and gelsemium by mouth. Mild doses of diathermy and of galvanism help many cases. Opium or morphine should never be given internally or hypodermically. The ultimate prognosis is good. Hysterical pains in the face may follow dental extraction and are commonly bilateral and continuous. Paroxysmal trigeminal neuralgia is very rarely bilateral in distribution.

Glossopharyngeal Neuralgia.—The pain occurs in the region of the tonsillar fossa, deep in the ear and in front of or behind the auricle, which may be hyperæsthetic. The possibility of malignant disease in the pharyngeal region must be excluded. The malady is analogous to tic douloureux and may be cured by avulsion of the glossopharyngeal nerve and cutting the pharyngeal branch of the vagus. Severe supraorbital pain may follow *herpes ophthalmicus* in old people. See § 856.

Occipital Neuralgia.—Neuralgic pains in the occipital region or the cervical region are commonly due to neurofibrositis or to cervical spondylitis. If persistent, the pain can be relieved by mobilisation of the neck under general anæsthesia, with subsequent exercises, massage and radiant heat, or by injecting the tender nodules in the muscle with ♏7 of 90 per cent. alcohol, after producing local anæsthesia with procaine 2 per cent.

Pain in the Upper or Lower Limbs

It should be borne in mind that pain in a limb may be due to disease of the muscles, bursæ, blood-vessels, bones, joints, peripheral nerves or central nervous system, or may be referred, as in coronary disease. You should examine each of these structures systematically, palpating the bones, vessels and peripheral nerves, testing the mobility of all the joints, testing sensibility and the tendon reflexes. A general examination of all the systems is necessary, together with testing of the urine and often an X-ray examination, for evidence of arthritis, cervical rib, or spondylitis. Spinal puncture may be necessary to clinch the diagnosis.

Pain in the Upper Limbs.—*Periarticular adhesions* round the shoulder-joint, or *cervical spondylitis, bursitis,* and *fibrositis* are common causes of neuralgic pains. Such pains are increased by movement of the neck, shoulder-joint or arm in certain directions, and there are limitation of movement and tender points in these structures. One mobilisation (or more) under general anæsthesia, with subsequent massage and radiant heat, usually improves the condition greatly. *Radiculitis* is accompanied by muscular paralysis of root distribution and, occasionally, sensory impairment (§ 733).

§ 824. **Brachial Neuritis** is a form of interstitial neuritis, affecting both sexes, with a subacute onset after strain or cold. The pain is severe and

TABLE LVIII.—CAUSES OF PAIN IN THE LIMBS

Cause In.	*Upper Limbs.*	*Lower Limbs.*
Muscles.	Neurofibrositis.	Neurofibrositis. Cramp and Heat-Cramp.
Blood-Vessels.	Raynaud's Disease.	Intermittent Claudication. Thrombo-angiitis Obliterans. Syphilitic Endarteritis.
Bones.	Periostitis or Osteomyelitis.	Periostitis or Osteomyelitis. Paget's Disease of Bone.
Joints.	Periarticular Adhesions. Arthritis (especially shoulder joint). Cervical Spondylitis. Pott's Disease.	Disease of Sacro-iliac or Hip Joints (Osteoarthritis, Tuberculous Disease, Acute Rheumatism, etc.). Lumbar Spondylitis. Prolapsed Intervertebral Disc.
Peripheral Nerves.	Rib pressure Syndromes. Pressure Lesions (enlarged supraclavicular glands, fibrositis, etc.). Brachial Neuritis. Median and Ulnar Neuritis. Causalgia. Infective Radiculitis. Referred Pains in Coronary Disease.	Sciatic Neuritis. Pressure Lesions, *e.g.*, Pelvic Tumours. Meralgia Paræsthetica. Diabetic and other types of Polyneuritis. Infective Radiculitis.
Spinal Cord.	Spinal Tumour (Primary and Secondary). Meningomyelitis. Tabes Dorsalis.	Primary and Secondary growths in Cauda Equina. Meningomyelitis. Tabes Dorsalis.

is described as "stabbing" or "boring," with paræsthesiæ, and radiates from the shoulder to some or all of the fingers. The brachial plexus and the nerves derived from it are tender on deep palpation, (*a*) in the supraclavicular region or (*b*) on the inner aspect of the arm. Passive flexion of the limbs at the elbow and abduction at the shoulder (Bikeles's Sign) stretches the plexus and nerves and causes acute pain. The muscles of the upper limb are hypotonic and tender, but there is rarely any true loss of power, gross wasting, or objective sensory impairment. The tendon reflexes of the affected limb are commonly increased in the early stages of the disease; they may be abolished later. The skin may become shiny and atrophic and the nails brittle. Subungual hæmorrhages may occur in severe cases.

Cases last from a few weeks to many months. Mild cases clear up in six weeks. Secondary malignant deposits, compressing the brachial plexus, cause a more gradual onset of symptoms, with muscular weakness and sensory loss and a mass can commonly be felt in the supraclavicular fossa.

Treatment.—Preliminary rest in bed, with the limb between sandbags, in the abducted position, is of value. Aspirin gr. 10, pyramidon gr. 5, heroin gr. $\frac{1}{12}$, night and morning, will relieve pain. Later, the arm should be put in an abduction frame, as for fracture of the head of the humerus, for three to four weeks or longer. Daily passive movements will prevent periarticular adhesions and contractures. Diathermy is useful, after the acute stage, in cases which can bear heat, and, after the arm

comes out of the splint, massage and a sling should be prescribed until the pain has entirely disappeared.

The symptoms of Rib pressure syndromes have been described in § 800. A rib may be palpable as a bony mass in the supraclavicular fossa, and pressure upon it may cause pain down the middle finger or obliteration of the radial pulse. Cervical oculo-pupillary phenomena may be present on the side of the lesion.

Median Neuritis is the result of interstitial inflammation or fibrosis of the nerve, sometimes in the carpal tunnel. It may be secondary to arthritis of the wrist, or prolonged pressure on the palm of the hand with a file or scrubbing brush (occupational neuritis). Pain and impairment of sensibility occur on the radial half of the palm, over the *palmar* surface of the thumb, index, middle fingers and the radial half of the ring-finger. The dorsal aspect of the terminal phalanges of these digits are similarly affected. Wasting and weakness occur in the abductor, opponens and flexor brevis pollicis. Appreciation of light touch, tactile discrimination, and sensitivity to pinprick are impaired over the distal parts of the affected digits, with subjective burning and tingling sensations.

Ulnar Neuritis is commonly traumatic on account of the exposed position of the nerve as it runs behind the internal condyle. Repeated slight traumata cause palpable thickening of the nerve-trunk. The deep palmar branch may be affected by long-continued pressure of some tool or instrument in certain occupations (*e.g.*, scrubbing women). Pain is referred to the dorsal and palmar surface of the hand over the ulnar border, the little finger and ulnar half of the ring-finger. Sensory impairment and paræsthesiæ may be present over this area and never occur above the wrist-line. There is weakness and wasting of the intrinsic muscles of the hand, except the abductor, opponens and flexor brevis pollicis, which are supplied by the median. The two ulnar fingers may be hyper-extended at the metacarpo-phalangeal joints, and slightly flexed at the interphalangeal joints (" claw-hand," § 803, Table LIII).

Treatment.—An anterior forearm and cock-up hand splint should be worn at night to prevent contractures, and, in the acute stages, the arm should be in a sling. Diathermy is useful and aspirin will relieve the pain. In the later stages, massage and re-educative movements are of value. In median neuritis due to thickening of the nerve in the carpal tunnel relief may sometimes be obtained by surgical exploration with neurolysis, and decompression of the swollen nerve. Operation may help cases of ulnar neuritis due to pressure on the ulnar nerve in the condylar groove at the elbow, or in cases where the nerve is involved by callus or osteophytic outgrowths, transposition of the nerve being performed. Cases of ulnar neuritis may take many months to recover.

Median or Ulnar **Causalgia** follows partial lesions of these nerves. (1) The pain is severe and burning in character and continuous. (2) It occasionally exceeds the anatomical distribution of the affected nerve. (3) Psychical factors are present. Such cases should never be given morphine. Hot fomentations of lead and opium

lotion or cold compresses will relieve the pain, with gelsemium and bromide by mouth. The ultimate prognosis in most cases is good. In severe cases Turrell advised a weak interrupted galvanic current applied twice a day.

PAIN IN THE LOWER LIMBS may be due to lesions of the muscles, bones, joints, peripheral nerves and blood-vessels, or central nervous system. A general physical examination, with testing of the urine for sugar, and a rectal examination, are necessary in all cases. Pain, in association with *chronic arthritis of the hip-joint,* may be referred to below the knee or to the outer side of the ankle. There is limitation of abduction and internal rotation, and the patient lies with the limb everted. There is difficulty in crossing one knee over the other.

§ **825. Sciatic Neuritis** (Synonym: Sciatica).—The main trunk of the nerve is made up of the union of the upper sacral and the 4th and 5th lumbar nerves. The onset of sciatica is acute or subacute and frequently follows an attack of lumbago. The pain is aching, and follows the distribution of the nerve. Sensations of "crawling," "pins and needles" and burning are frequently described by sufferers. The nerve is tender on stretching and on deep palpation along its course—the sciatic notch, middle of the back of thigh and popliteal space, behind the head of the fibula and external malleolus and on the sole of the foot. With the patient in the horizontal position, straight-leg raising from the couch brings on the pain. The muscles show a slight degree of softening or atrophy, most marked in the anterior tibial group, rendering more prominent the anterior ridge of the tibia on the affected side. Fibrillation may be observed. In severe cases there is weakness of dorsiflexion of the foot or great toe, even drop-foot. Sensory impairment may be present on the outer aspect of the leg, sole or dorsum of the foot, but is always slight. The ankle-jerk is diminished on the affected side in severe cases, the other tendon reflexes being unaltered. The patient has difficulty in sitting on the affected buttock and walks with the limb slightly flexed, and on the toes, with the painful extremity internally rotated. Compensatory scoliosis may accompany the pelvic tilting of the erect posture. In *Radicular Sciatica* the pain is chiefly in the buttock and is intensified by coughing or sneezing.

Prognosis.—Favourable cases recover in four to six weeks. Second or third attacks are occasionally observed in the same or opposite limb. After recovery the ankle-jerk may remain in abeyance. A valuable index of prognosis is the degree to which the affected limb can be raised with the patient horizontal and the knee held straight.

Etiology.—It is a disease of early and middle life, chiefly affecting males. Predisposing causes are exposure to damp, trauma, violent exercise, continued pressure on the nerve as in motor-driving, tumours or the frequently associated lumbo-sacral fibrositis. Sciatica may appear as an acute or chronic symptom in diabetes, and may then be bilateral. Pain of sciatic distribution occurs with carcinoma of the rectum or sigmoid or other pelvic tumours, morbus coxæ, sacro-iliac subluxation and arthritis. For pressure lesions, see §§ 802, 803.

Diagnosis.—The distribution of the pain, tenderness of the nerve and of the muscles supplied by it with diminution or absence of the ankle-jerk, is characteristic. Rectal examination, urine testing for sugar, and a good X-ray of the hip and sacro-iliac joints, to exclude arthritis, should be routine in all cases.

Treatment.—In the acute stages order strict rest in bed, with blanket baths and bed pan or commode, and splinting with a long Liston's splint reaching from the axilla to the ankle. The early pain may be relieved by aspirin gr. 10, pyramidon gr. 5 and heroin gr. $\frac{1}{12}$, night and morning. Warmth, blisters and counter-irritation along the course of the nerve may abort the disease. The underlying condition should be treated. In more severe cases, a plaster shell from the mid-thoracic region to the toes, or a plaster spica fixing the hip- and knee-joints, may be applied for from four to six weeks. In milder cases, and after removal of the plaster, massage or diathermy may be given daily. Persisting pain should be treated by injection of 50 c.c. of sterile normal saline into the neighbourhood of the nerve where it leaves the sciatic notch. Other useful measures are galvanism (100 to 200 m.a.), radiant heat and the Kromayer lamp. Small filtered doses of X-ray may be applied over the exits of the nerve-roots, from the spine. Epidural treatment is described in § 919.

Prolapsed Intervertebral Disc.—A unilateral or bilateral sciatic nerve syndrome may follow traumatic extrusion of the nucleus pulposus of an intervertebral disc through its annulus fibrosus. The herniated disc is usually that between L4, 5 or L5, S1 vertebræ and it impinges (perhaps intermittently) on the anterior or antero-lateral aspect of the theca with its contained lumbo-sacral nerve roots. The *Signs* are those of radicular sciatic neuritis. The *Condition may be suspected* with (1) a history of sudden, immobilising low back pain, (2) a long history of recurring unilateral or bilateral sciatic pain in an active muscular subject, (3) the normal lumbar lordosis being replaced by a rigid flattening of that part of the spine. (4) Moreover, the C.S.F. protein is more likely to be raised in this condition than in sciatic neuritis owing to the partial thecal block. (5) The knee as well as the ankle jerk may be diminished or absent if L2, 3 roots are affected. (6) Contrast myelography by means of radiographs taken after intrathecal injection of air or iodised oil may or may not succeed in demonstrating a prolapsed nucleus pulposus.

Treatment is as for sciatic neuritis. Rest in the horizontal position in the early stages for 4–6 weeks is advised with a plaster jacket when the patient is ambulant. The results of surgical removal of the extruded nucleus pulposus are at present uncertain and conservative treatment is generally to be advised.

Meralgia Paræsthetica.—In this condition, numbness or paræsthesia to light touch over the outer aspect of the thigh in the distribution of the external cutaneous nerve occurs. It is probably due to a rheumatic affection of the nerve where it pierces the fascia lata.

Femoral Neuritis (see § 803) is a rare affection causing pain on the anterior aspect of the thigh and diminution or loss of knee-jerk. It may be due to syphilis, to chronic rheumatic or pressure lesions in the pelvis.

Intermittent Claudication.—See § 580.

Pains in the Trunk, due to muscular or aponeurotic fibrositis, spondylitis, caries or tumours of the vertebral column, intrathoracic aneurysm and new growth, and pleurisy, are discussed in other sections. The

referred pain of renal and biliary colic and coronary disease are also discussed in their appropriate sections. The chronic *pain under the left breast* complained of by many young women is not symptomatic of visceral disease, but is part of an anxiety neurosis.

The *neurological* causes of pain in the trunk are varied. Many are dealt with in the appropriate chapters. Firstly, there are the root-pains of inflammatory meningeal disease (syphilis, tubercle, spondylitis) or of pressure lesions (primary and secondary tumours of the thorax, vertebral column, meninges, or nerve-roots). Chronic root-pains, commencing unilaterally and later becoming bilateral, are almost pathognomonic of *spinal tumour*. The "lightning pains" and "visceral crises" of *tabes dorsalis* should be borne in mind; other signs of the disease should be looked for.

§ 826. **Herpes Zoster** produces unilateral root-pains either preceding or succeeding the eruption of herpetic vesicles. The condition is due to inflammation of a posterior root ganglion. It is caused by a virus infection, with an initial febrile reaction, and is particularly liable to occur in tabes dorsalis, in patients taking arsenic, and in spinal caries. Attacks of herpes may follow chicken-pox after an interval of fourteen days (§ 476).

Symptoms.—(1) Fever and pain of root distribution are present for three to four days, and pleurisy may be suspected. (2) Between the second and the sixth day, an erythema of the skin appears, upon which groups of tiny vesicles, filled with clear fluid, develop. From the fifth to the tenth day the vesicles coalesce and rupture, with the formation of scabs. The distribution of the eruption is always that of a nerve segment. Pain, itching or intense burning last until the third or fourth week, but in elderly patients may persist in spite of treatment for months or years.

Complications.—(1) Scarring and (2) pigmentation are fairly common after the vesicles heal. (3) Persistent root pains, (4) objective sensory impairment, and (5) flaccid paralysis of root distribution, may last for months. (6) Pleurisy or (7) effusion into joints are rarer complications. Ophthalmic herpes is described in § 856, and Geniculate herpes in § 858.

Treatment.—The vesicles should be covered with collodion and gauze, and the collodion re-applied daily until healing occurs. Pituitary extract ½–1 c.c. intramuscularly may relieve the pain, but in some cases morphia may be necessary. Post-herpetic neuralgia seems to be uninfluenced by any form of treatment, and measures should be instituted to improve the general health. Deep X-ray therapy may be tried, and ultra-violet light (third degree erythema).

GROUP XII. PROGRESSIVE HEADACHE AND VOMITING

§ **827.** The various types of headache and their causes have already been considered (see §§ 695, 696). Progressively severe headache, with vomiting, occurs in five conditions, the most important being INTRACRANIAL TUMOUR. These five conditions are:

I. Intracranial Inflammations, *e.g.*, Abscess, Encephalitis, Meningitis.

II. Slow Intracranial Bleeding, *e.g.*, Chronic Subdural Hæmatoma, following head injury.

III. Intracranial Tumour.
IV. Progressive Toxæmia, *e.g.*, Chronic Renal Disease, Industrial Chemical Poisoning, *e.g.*, lead.
V. Acquired Hydrocephalus.

I. **Intracranial Inflammations** are accompanied by *pyrexia* and characteristic pleocytosis in the spinal fluid. Their diagnosis, including the diagnosis of *Intracranial Abscess*, is considered in § 737 *et seq.* Reference must be made to a condition occurring *in association with suppurative otitis media, which simulates intracranial tumour.*

Otitic Hydrocephalus occurs in children and young adults. Headache, vomiting and papillœdema occur, without localising signs. The spinal fluid, although under pressure, is sterile and may be free from excess of cells. The condition is relieved by thecal punctures and subsides spontaneously.

II. **Slow Intracranial Bleeding** is usually rapidly productive of convulsions and coma and its diagnosis is considered in § 711. Reference must be made to one condition which closely simulates intracranial tumour—Chronic Subdural Hæmatoma.

Chronic Subdural Hæmatoma results from traumatic tearing of small cortical veins draining into the superior longitudinal sinus. Slow venous oozing into the subdural space then occurs when anything raises the intracranial venous pressure, *e.g.*, coughing, straining at stool. The blood clots and is encysted in the meshes of the pia-arachnoid. The serum separates from the clot, which slowly increases in size, and a " blood cyst " is formed, indenting the cerebral cortex. These blood cysts may be bilateral, on both sides of the superior longitudinal sinus. Symptoms like those of cerebral tumour gradually supervene after a latent interval of from one to six weeks. There is often a story of head injury, which may have been trivial, weeks or months before. Headache, vomiting, papillœdema, progressive apathy with focal Jacksonian fits starting in the leg, unilateral hemiparesis, or bilateral hemiplegia with extensor plantar responses, apraxia and aphasia occur. The variability of the symptoms from day to day is held to be characteristic. The pulse is often slow even in the early stages of the condition. The spinal fluid may be tinged yellow from altered blood, but is usually colourless.

Etiology.—These blood cysts may occur in normal individuals as the result of trauma, but commonly they arise in conditions when the brain is *shrunken, e.g.*, cerebral arterio-sclerosis, alcoholism, G.P.I., senile dementia, when after a trivial head injury the shrunken brain is thrown rapidly forwards or backwards in the skull-case, thus rupturing the cortical venous tributaries of the superior longitudinal sinus.

Treatment is exclusively surgical. The results of operation are often good. Bilateral decompression is necessary if the blood cysts are bilateral.

§ **828.** III. **Intracranial Tumours** are *relatively common.* Moreover, *they may occur in the young, even in children, as well as in the aged,* and may be present for weeks or months, even years, without producing focal

symptoms. Intracranial tumour should be suspected in any of the following conditions: (1) In any case of slow progressive headache and vomiting. The vomiting need not be of the projectile type nor is it necessary for papillœdema to be present. (2) In any case of epileptiform fits, occurring for the first time in an individual over the age of twenty-five years, especially if these fits are Jacksonian with transient residual paralysis. (3) In any progressive focal lesion of the nervous system, *e.g.*, a slowly progressive hemiplegia, slowly progressive nerve deafness, or visual failure; especially if the progress of the lesion is punctuated with Jacksonian convulsions. (4) In any case of pituitary endocrine disturbance.

THE COMPLETE DIAGNOSIS of intracranial tumour comprises—

A. The diagnosis of existence of a tumour.
B. The diagnosis of the localisation of the tumour.
C. The diagnosis of the nature of the tumour.

A. EXISTENCE OF A TUMOUR.

The presence of an intracranial tumour is manifested by *general* and *focal* signs of a *progressive* nature.

(*a*) *General Signs.*—These are (1) Headache. (2) Vomiting. (3) Papillœdema. (4) Increasing apathy or torpor. (5) Fits. These are all due to increasing intracranial pressure.

1. The *Headache* is a constant dull pain with paroxysms of intensity, usually on waking in the morning. If localised, its site does not necessarily indicate the site of the growth. Local tenderness of the skull, or pain on percussion may be present. Headache is early and severe in tumours of the posterior fossa and in pituitary growths. 2. *Vomiting* occurs later and is due to hydrocephalus. It occurs characteristically on waking or during the night; it is not necessarily projectile and may be preceded by nausea. 3. *Papillœdema* (Plates V and VI) should be looked for in every case where an intracranial tumour is suspected. Severe degrees of swelling may occur without visual failure. In cerebellar growths papillœdema is severe in degree and early in its appearance, whereas in pontine growths it is slight and late to appear. 4. *Increasing apathy or torpor* and dulling of alertness is very characteristic of cerebral growths. In cerebellar growths the mentality is often clear when the intracranial pressure is high. 5. *Fits*, indistinguishable from those of idiopathic epilepsy, arise as the result of increased intracranial pressure. They may be focal, in which case they may have localising value (§ 722), or generalised. They may precede all the other symptoms and for a long time, be the only symptom of the growth. The pulse rate commonly drops to fifty per minute, the blood pressure rises and slow or periodic breathing may be present. The hydrocephalus responsible for these symptoms results from displacement and compression of the vein of Galen draining the choroid plexuses, or from obstruction in the ventricular system.

(*b*) *Focal Signs.*—These, too, are *progressive*. They are commonly the result of local circulatory changes in the neighbourhood of the growth,

with anoxæmia, rather than actual disruption or distortion of nerve fibres. Even if the growth is not removed, they often clear up almost completely after simple decompression, which restores the local circulation.

The *earliest signs have the greatest localising value.* Tumours of the frontal lobes may produce in their late stages contralateral cerebellar signs—"*false localising signs.*" In such a case a history of *early* mental or emotional changes, or monoplegic weakness (prefrontal lobe), is of extreme importance in focal diagnosis. The focal signs vary with the area of brain involved.

§ 829. B. The Localisation.

Frontal Lobes.—(1) Mental changes—early alteration in personality, memory failure, euphoria and childish facetiousness, falling off in nice habits, carelessness in dress and incontinence, may be present, with shallowness of emotional feeling. (2) Unilateral anosmia or feelings of irritation in the nose from downward pressure on the olfactory bulbs. Rubbing of the nose is frequent. (3) Unilateral proptosis. (4) Deep lesions may cause slight contralateral pyramidal signs, usually monoplegic. (5) In lesions of the posterior parts of the first and second frontal convolutions the "grasp reflex" may be observed (see § 707).

Motor Area.—The irritative and paralytic signs in lesions of the motor cortex have already been described (see § 668). Deep lesions cause hemiplegia with marked spasticity, and motor aphasia is present if the lesion be left-sided.

Sensory Area.—The irritative and paralytic signs in lesions of the sensory cortex have already been described (see § 674).

Occipital Lobes.—*Irritative lesions* of the calcarine cortex produce visual Jacksonian fits ("sheets of flame," "moving lights") of hemianopic distribution followed by a transient hemianopia. *Paralytic cortical lesions* cause homonymous hemianopia (a hemiplegia of the fields of vision on the contralateral side of the lesion), while destructive partial cortical lesions will produce quadrantic hemianopia or altitudinal hemianopia (see § 676). In lesions of the left angular or supra-marginal gyri "word-blindness" occurs. There may be slight contralateral sensory and motor pyramidal signs.

Temporal Lobes.—(1) Homonymous hemianopia or quadrantic hemianopia of the contralateral visual fields occurs from involvement of the "temporal knee" of the optic radiations. Complex visual hallucinations of hemianopic distribution occur (see § 676). (2) Facio-brachial monoplegia may be present. (3) Lesions involving the subcortical tissues beneath the superior temporal convolution on the left side produce "jargon aphasia" or "word-deafness." (4) Irritative lesions of the uncinate gyrus produce *uncinate fits* with an aura of pungent odours, champing or spitting movements and a transient "dreamy state."

Corpus Striatum and Optic Thalamus.—Lesions involving the internal capsule produce hemiplegia, hemianæsthesia and hemianopia, depending on the posterior extent of the lesion (see § 670). In lesions of the optic thalamus the hemiplegia tends to be transient, but the "thalamic syndrome" of Déjèrine and Roussy, persists, viz.: (1) Sensory loss, chiefly sense of position, of hemiplegic distribution. (2) Involuntary movements and (3) Spontaneous pains in the affected side of the body and limbs, with over-reaction to affective sensory stimuli, *e.g.*, hot stimuli are felt as burning.

Corpus Callosum.—Early apathy or fits, bilateral pyramidal signs, rarely symmetrical, and yellow spinal fluid, are common. In lesions of the genu, motor apraxia occurs in a considerable proportion of cases. Cranial nerve palsies are rare.

Superior Corpora Quadrigemina and Third Ventricle.—Tumours in this region produce internal hydrocephalus. Pineal tumours are uncommon and may be associated with precocious puberty and excessive growth generally. They occur chiefly in boys and may be dermoid cysts. The *pineal body* lies in close apposition to the superior corpora quadrigemina. Lesions in this area produce (1) Loss of conjugate upward

movement of the eyes. (2) Internal ophthalmoplegia. Lesions involving the grey matter of the *floor of the third ventricle* produce obesity, amenorrhœa, diabetes insipidus, glycosuria, pathological drowsiness and other disorders of sleep.

Inferior Corpora Quadrigemina and Superior Cerebellar Peduncles.—Lesions in this region produce bilateral auditory disturbances, ophthalmoplegia and bilateral ataxy of the limbs and trunk. The pyramidal fibres may be involved, with spasticity, especially of the facial muscles.

Optic Chiasma.—The commonest lesion in this region is **Pituitary Tumour.** The symptoms produced are (1) *Endocrine disturbances*—Acromegaly (Fig. 6) or Gigantism, from anterior lobe lesions, Fröhlich's dystrophia adiposo-genitalis (Fig. 7), or Lorrain-Levi infantilism in lesions of the pituitary stalk. These may, however, be absent. (2) *Increased intracranial pressure symptoms*—rare except in tumours of the pituitary stalk. (3) *Neighbourhood symptoms,* due to pressure on surrounding structures. From pressure on the chiasma central scotomata and quadrantic temporal field defects are common (superior quadrants affected if the pressure is from below and *vice versa*). Blindness in one eye, with optic atrophy, is common from direct pressure on the nerve; later, the optic atrophy is bilateral and complete. Unilateral proptosis may occur. "Uncinate fits" (§ 675. III*b*) are common from irritation of the uncinate cortex, which lies just lateral to the pituitary fossa. (4) *Radiographic Signs.*—X-rays (preferably stereoscopic) will show erosion of the posterior clinoid processes and deepening of the pituitary fossa in many cases (Fig. 187*b*). These X-ray signs may be present in chronic hydrocephalus from any cause, and are not pathognomonic.

Brain-Stem (see §§ 670, 683).—(*a*) *Crus Cerebri.*—Oculo-motor paralysis on the side of the lesion with hemiplegia on the opposite side (Weber's syndrome). If the tegmen is involved, contralateral hemiataxy, from involvement of the superior cerebellar peduncles. In tumours these symptoms are often bilateral. Interferences with the fillet may cause hemianæsthesia. (*b*) *Pons*—very varied symptoms. The trigeminal, abducens and facial may be paralysed on the side of the lesion with crossed hemiparesis (Foville's syndrome). In lesions of the upper pons there will be contralateral hemianæsthesia. The signs may be bilateral in tumour. (*c*) *Medulla.*—Paralysis, unilateral or bilateral, of the lowest cranial nerves and nuclei from the ninth to the twelfth, producing dysarthria, dysphagia; with signs of interruption of long sensory and motor projection tracts. Medullary symptoms are often secondary to tumours of the vermis or the pons.

Cerebellum (see § 812) and *Cerebello-Pontine Angle* (Lateral Recess) (see § 814).

RADIOGRAPHY IN CEREBRAL TUMOUR.—In every case of suspected intracranial tumour the chest should be X-rayed as well as the skull, so as to exclude an unsuspected primary intrathoracic neoplasm. Stereoscopic radiograms of the skull should be taken in all cases where a tumour is suspected. They may show (*a*) *Abnormalities in the bones.*—Localised bony thickening or rarefaction or widening of the diploic venous channels in the neighbourhood of a cortical meningioma. The X-ray appearances found in pituitary tumour, described above, are met with in hydrocephalus from any cause. Unilateral widening of the internal auditory meatus in acoustic neurofibroma can rarely be demonstrated. (*b*) *Calcification.*—Suprasellar cystic adamantinomata may show calcification (Fig. 187*b*), as may angiomata, gliomata and tuberculomata. Calculi in the brain occur in the aged after cerebral thrombosis. In 50 per cent. of adults over the age of twenty-five years the pineal gland is calcified, and in accurately centred antero-posterior radiograms the shift of the calcified pineal shadow ("pineal shift") to one or other side may confirm the diagnosis of the side of the lesion in tumours of the hemispheres. (*c*) *Encephalography.*—60 to 100 c.c. of filtered air are introduced, 5 c.c. at a time by fractional replacement of spinal fluid, using the lumbar route. 5 c.c. of spinal fluid are withdrawn at a time and replaced by 5 c.c. of air. The cerebral convolutional markings or the ventricular system are rendered visible in successful cases. The method is not without serious dangers. (*d*) *Ventriculography.*—This is probably safer. Both

lateral ventricles are punctured and 5 c.c. of cerebro-spinal fluid are withdrawn at a time and replaced by 5 c.c. of air until 80–100 c.c. of filtered air have thus fractionally been introduced. Displacement of the ventricles, filling defects, etc., may be demonstrated in antero-posterior, postero-anterior and lateral radiograms, in a large percentage of cases (Fig. 187*c*, *d*). (*e*) *Cerebral Angiography.*—The arterial and



FIG. 187 (*a*).—NORMAL OVAL SELLA TURCICA. Round and flat sellæ are also met with in the normal. Union between anterior and posterior clinoid processes, producing "roofing" of the sella, is of no clinical significance.



FIG. 187 (*b*).—SUPRA-SELLAR CALCIFICATION with erosion of the posterior clinoid processes in a case of pituitary stalk tumour (adamantinoma).

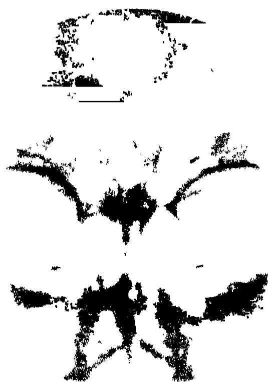


FIG. 187 (*c*).—NORMAL VENTRICULOGRAM (antero-posterior) showing the lateral and third ventricles. The septum lucidum is in the mid-line.

Two circular burr holes are also seen where air has been introduced, and ventricular fluid removed.



FIG. 187 (*d*).—VENTRICULOGRAM (antero-posterior) from a case of right temporal tumour, showing hydrocephalus, deformity of the right lateral ventricle and displacement of the septum lucidum to the left.

venous cerebral circulation can be visualised by injecting contrast medium into the internal carotid artery on the suspected side of the lesion (Fig. 188). The injection is made either through the skin or else through a transverse incision in the neck. Very considerable practice is required. Antero-posterior and lateral pictures are made. The arteriogram is taken after the first 4 c.c. of contrast medium is injected; the phlebogram after a subsequent 4 c.c. The procedure is repeated with the head



(*a*) (*b*)

FIG. 188.—PERCUTANEOUS ARTERIOGRAM OF NORMAL SUBJECT, USING 35 PER CENT. DIODONE AS THE CONTRAST MEDIUM. (*a*) Lateral view showing good filling of internal carotid, anterior, middle and posterior cerebral arteries. (*b*) Frontal view, note typical T-shape of carotid bifurcation behind frontal sinus, and that arterior cerebral artery lies exactly in the mid-line.



(*c*) (*d*)

FIG. 188.—PERCUTANEOUS ARTERIOGRAM IN A CASE OF PTERION MENINGIOMA (CONFIRMED BY OPERATION). (*c*) Lateral view showing a very vascular tumour outlined by contrast medium contained in vessels derived from the middle cerebral artery. The terminal internal carotid and proximal middle cerebral arteries are stretched by the tumour. Note the abnormal vascular channels in left frontal bone remain unfilled. (*d*) Frontal view of same case. Little contrast medium has entered the anterior cerebral artery which can be just seen displaced across the mid-line. This investigation gives the size, shape, position and pathology of the tumour.

[Mr. Wylie McKissock's case from angiograms by Dr. J. Bull.

turned at right angles. The needle is left in the artery during the change of position, and must be kept patent by injection of normal saline between the two injections of 8 c.c. of contrast medium. The method is of considerable value in demonstrating intracranial neoplasms and aneurysms but it is not without risk. Symptoms may worsen, Jacksonian attacks and hemiplegia are reported after such injections.

C. The Nature of the Tumour.

It is increasingly apparent that different types of tumour have characteristic sites of origin in relation to the brain; the age incidence tends to be characteristic in certain types of tumour; moreover, each tumour tends to behave characteristically in its natural history. Though all intracranial tumours may have clinical features in common, we may say that each type is a distinct clinical as well as pathological entity. Observations made on one type of tumour do not necessarily hold for another type.

Gliomas of varying degree of malignancy form 40 per cent. of all intracranial tumours. The *astrocytoma* which grows from the neuroglia in the frontal and temporal lobe and also in the lateral lobe of the cerebellum, is relatively benign. It may form a cyst containing yellow fluid on the wall of which is found tumour tissue. Almost as frequent is the highly malignant *glioblastoma multiforme* (progressing rapidly and fatally) in the cerebral hemispheres of adults of about the age of 40. The symptoms of onset are often sudden, as the tumour is vascular and the vessels within it liable to hæmorrhage and thrombosis. Life is rarely prolonged beyond 12 to 15 months.

Secondary Carcinoma forms about 20 per cent. of all brain tumours. Deposits are often multiple, and so frequently do they arise from an unsuspected primary bronchial carcinoma that X-ray examination of the chest should be performed as routine in all cases where an intracranial growth is diagnosed.

Meningioma (15 per cent.) is a benign tumour arising in the arachnoid villi on the dural walls of the venous sinuses, the venous tributaries of which become adherent to the tumour mass. It does not invade the brain but indents it: it tends, on the other hand, to invade and cause reactive changes in the overlying skull bones, and these may be visible in a straight X-ray picture.

An Acoustic Neurinoma (a benign neurofibroma growing from the acoustic nerve or its sheath) may be the cause of progressive nerve deafness, especially if unilateral. Such a tumour grows slowly, pushing aside the brain-stem and cerebellum causing first a hydrocephalus from obstruction of the iter and later, herniation of the cerebellar tonsils through the foramen magnum in a " pressure cone ". It extends deeply into the internal auditory canal and stretches the facial nerve. By its growth it compresses the fifth, ninth, tenth and eleventh cranial nerves.

Pituitary Tumours. When acromegaly is due to pituitary tumour, that growth is invariably a *chromophil adenoma* of the anterior lobe. *Chromophobe adenoma* also occurs in the anterior lobe and may be unassociated with dyspituitarism. Both these tumours occur within the sella. Tumours arise in the pituitary stalk (infundibulum) chiefly in early life; they are usually malignant *adamantinomata*, cystic epithelial carcinomata arising from an unobliterated Rathke's pouch. They are found from the age of 10 years into early adult life, in the X-ray tend to produce shadows above the sella, due to calcification in the growth, and with clinical signs of hypopituitarism (infantilism or obesity). *Meningiomata* occur after the age of 30 years growing from the dural diaphragm of the sella.

Tumours of Blood Vessels.—*Hæmangioma* is a benign cystic tumour found characteristically in the cerebellum of middle-aged males. The large cyst contains xanthochromic fluid, the tumour existing as a small nodule in its wall. Cysts may be present in the liver or pancreas and other allied tumour lesions may exist in other organs.

As well as the small " berry aneurysms " on the hexagon of Willis which are the

common cause of subarachnoid hæmorrhage, we may find a solitary aneurysmal swelling of the internal carotid artery behind the ocular fissure, just where the artery enters the skull. Venous angiomata in young adults are commonly associated with epilepsy, and there may be a congenital nævus of the face on the same side as the lesion. The diagnosis may be aided by an appearance of calcification found in X-rays of the skull of such cases, casting parallel tortuous shadows on the plate. Arteriovenous aneurysms may cause an intracranial bruit, audible to the patient, and which can be heard by the observer if he applies his ear or stethoscope to the patient's skull.

In CHILDREN the *medulloblastoma* is a highly malignant type of glioma found in the roof of the fourth ventricle or the pons. Local metastases occur and death is common within a year. In the cerebellum of children and young adults we find *cystic astrocytoma* and *hæmangioma*. *Tuberculoma* and *abscess* occur in children and young adults, the former lesion tending to calcify if it is chronic.

Tuberose Sclerosis is a rare congenital disease occurring in mental defectives, (§ 907 C) producing multiple tumours in the brain and other organs, and sebaceous adenomata of the face and scalp. The diagnosis is made from the cutaneous condition (§ 625).

LUMBAR PUNCTURE IN INTRACRANIAL TUMOUR is often highly dangerous, owing to the likelihood of the cerebellar tonsils and medulla descending into the foramen magnum as a "pressure cone," with sudden increase of intracranial pressure, coma and death. The fluid, in intracranial tumour, is under considerable pressure but is otherwise normal except in tumours impinging on the subarachnoid space or ventricles, where excess of protein may be found and, in some cases where the tumour is vascular, yellow coloration and slight lymphocytosis (10–15 cells per c.mm.).

Prognosis of Intracranial Tumour.—Blindness, coma and death will ensue if the tumour is left untouched. The operation results are best in acoustic neurofibromata and meningioma of the vertex. Calcification and spontaneous cessation of growth sometimes occur in tuberculomata, but this event is an extreme rarity. Sudden coma, occurring before surgical decompression, is of the gravest omen.

Treatment of Intracranial Tumour.—The treatment is exclusively surgical. Prior to operation, 50 c.c. of 50 per cent. dextrose solution may be given intravenously (3 c.c. a minute) to reduce intracranial pressure. Preliminary ventricular puncture is of great value also in this respect.

An osteoplastic flap is usually turned down and the tumour, if accessible, is removed or destroyed by endothermy. Cysts are drained and their walls removed or destroyed. Where the tumour is inaccessible a decompression operation is performed. In the posterior fossa this involves removal of the posterior segment of the foramen magnum and the arch of the atlas, to accommodate the "pressure cone."

Tumours of the brain-stem are not helped by decompression, which may precipitate death. They are best treated by hypertonic intravenous solutions (§ 919) and by tapping of the lateral ventricles, if headache is severe. Tuberculomata, recognised at operation, should not be removed, as tuberculous meningitis will probably ensue. Simple decompression is sufficient. Deep X-ray inhibits the growth of pituitary tumours, sarcoma of the base of the skull and the malignant medulloblastoma of children.

IV. **Progressive Toxæmias.** CHRONIC RENAL DISEASE.—Headache, vomiting and optic neuritis occur and the symptoms may closely simulate those of intracranial tumour. The late appearances of papillœdema closely simulate those of albuminuric retinitis. In such cases extensive focal softenings of the cerebrum may occur *gradually*, simulating the focal march of a cerebral tumour. High blood pressure is common in both conditions. In these cases it is often extremely difficult to make the diagnosis. A very high blood-urea reading is characteristic of renal disease.

INDUSTRIAL CHEMICAL POISONING (*e.g.*, lead encephalopathy, § 553) may simulate intracranial tumour with its headache, vomiting and optic neuritis. Epileptiform fits may occur. The diagnosis is sometimes difficult. The poisonous substance may be demonstrated in abnormal concentration in the blood or excreta.

§ 830. V. **Acquired Hydrocephalus** is usually the result of meningitis or neoplasm. When secondary to meningitis, there is usually much meningeal scarring and adhesions are present in the roof of the fourth ventricle and tentorium, round the veins of Galen. There is persistent headache, vomiting, mental impairment, convulsions and sometimes, papillœdema or optic atrophy. In children, enlargement of the head is a striking feature, and in young children the sutures separate with bulging of the fontanelles. There is marked general wasting. Neoplasms, which cause hydrocephalus, are commonly situated in some part of the brain-stem from the third ventricle to the medulla. In such cases, X-rays may show enlargement of the pituitary fossa from distension of the infundibulum. Such a finding should not lead one to the diagnosis of pituitary tumour unless neighbourhood symptoms are present.

Hydrocephalus from stenosis of the Aqueduct of Sylvius may be *Congenital* and here the presenting symptom is enlargement of the cranium. Later mental failure, fits, ataxia, spastic weakness of the limbs, nystagmus and optic atrophy may occur. The *diagnosis* from other causes of enlargement of the head may be difficult. In most cases hydrocephalus is slowly progressive and uninfluenced by treatment. A few cases survive to adult life with their faculties more or less impaired.

GROUP XIII. THE CRANIAL NERVES AND SPECIAL SENSES

The investigation of the cranial nerves and special senses is of great importance, from the standpoint both of general medicine and neurology. A tabular statement of the cranial nerve-functions will be found in Table LIX. The applied physiological anatomy of the special senses and cranial nerves has been considered in §§ 675 to 678 and § 683, while the methods of examination have been described in § 703.

§ 831. **The Olfactory Nerve** (§ 675) consists of fibres which arise in the upper nasal mucosa, penetrate the cribriform plate of the ethmoid bone, and terminate in the olfactory bulbs. The methods of examination are described in § 703. *Anosmia* is loss of smell. Bilateral anosmia may arise from local disease in the nose (§ 178) and is not necessarily of neurological significance. It not uncommonly follows cerebral

TABLE LIX.—CRANIAL NERVES AND THEIR FUNCTIONS

Cranial Nerves.	*Functions.*
I. Olfactory nerve.	Smell.
II. Optic nerve.	Sight. Afferent for pupillary light reflex.
III. Oculo-motor.	Supplies all the extrinsic muscles of the eyeball (except the superior oblique and external rectus) and the levator palpebræ superioris; also the sphincter pupillæ and ciliary muscle.
IV. Trochlear.	Supplies the superior oblique; turns the eye down and outwards.
VI. Abducens.	Supplies the external rectus; turns the eye outwards.
V. Trigeminal nerve. First division, Ophthalmic.	*Sensory* to forehead and part of vertex, anterior part of nose to tip, upper eyelid and temple, eyeball and lachrymal gland. Contains dilator pupillæ fibres from sympathetic.
Second division, Maxillary.	*Sensory* to cheek, lower eyelid, side of nose and upper lip; the upper teeth and gum; lining membrane of nose, roof of mouth, soft palate, tonsils and roof of pharynx. *Taste* of anterior two-thirds of tongue (through Meckel's ganglion by chorda tympani nerve). *Trophic* and vaso-motor fibres.
Third division, Mandibular.	*Sensory* to lower part of face, lower lip, side of head, ear, tongue lower teeth, gum, and inner side of cheek. *Motor* to masticatory muscles, temporal. masseter, pterygoids, anterior belly of digastric and mylo-hyoid, tensor tympani and? tensor palati. *Taste* of anterior two-thirds of tongue (by chorda tympani from lingual nerve); of posterior one-third of tongue through glosso-pharyngeal nerve, Jacobson's nerve, and otic ganglion.
VII. Facial nerve.	*Motor* to all muscles of face and scalp (excepting levator palpebræ superioris), platysma, posterior belly of digastric, and stapedius muscle. It is joined by the chorda tympani (conveying taste fibres of anterior two-thirds of tongue from lingual branch of V. to Meckel's ganglion).
VIII. Auditory nerve.	Hearing and Equilibration.
IX. Glossopharyngeal nerve.	*Sensory* from pharynx. Collects taste fibres from posterior one-third of tongue, which ultimately join V. *Motor* to middle constrictor of pharynx and stylo-pharyngeus.
X. Vagus nerve.	*Motor* for soft palate (except tensor palati), pharynx and larynx (through accessory portion of XI). *Motor* (involuntary) and *sensory* for heart, respiratory passages and abdominal viscera.
XI. Spinal accessory nerve.	*Motor* to sterno-mastoid and trapezius. (Supplies vagus with motor fibres for larynx, pharynx, and palate).
XII. Hypoglossal nerve	*Motor* to tongue and depressors of hyoid bone.

concussion, from tearing of the olfactory filaments and is usually permanent. Both bilateral and unilateral anosmia may result from pressure of a prefrontal or pituitary tumour upon the olfactory bulbs. For hysterical anosmia, see § 888.

The olfactory filaments are also of importance, as they may constitute an axonic pathway of invasion of the nervous system in certain virus infections, *e.g.*, poliomyelitis.

§ 832. The **Eye** is innervated mainly by four cranial nerves, the *second, third, fourth,* and *sixth.* The *fifth* and the cervical *sympathetic* are also concerned in its innervation. Careful examination of the eye is of the greatest importance in many diseases.

The symptoms which reveal disease of the eye may be arranged under six headings: I. Pain, II. Superficial Alterations (§ 833), III. Defects of Vision (§ 834), IV. Condition of the Pupils (§ 838), V. Ocular Movements (§ 844), VI. Changes in the Fundi (§ 848). The reader should turn to the section dealing with the defect to which the patient's symptoms appear to belong.

The systematic examination of the eye consists of: Investigating pain if present; noting any superficial alterations; testing defects of vision, including visual fields, acuity and refraction; examining the pupils, the ocular movements, and the fundi.

I. **Pain in the Eyes** is not infrequently absent in ocular affections. Its commonest cause is some error of refraction (ametropia). Eye-strain may give rise to headache, eye-ache or neuralgia. Glaucoma is a serious cause of pain in the eye and neighbourhood. Dental disorders may not only give rise to reflex pain, but also to both functional and organic affections of the eyes. Among subjective sensations other than pain may be noted muscæ volitantes (black specks) and scintillating scotoma (zigzag lines). The former may be normal, but evident to the patient because of eye-strain or poor health, or they may be pathological and due to vitreous opacities. The latter occurs in association with migraine.

ASTHENOPIA, with painful itching of the eyes and lachrymation, is associated with some degree of photophobia and blepharospasm. Often an expression of allergy, there may be a contributory refractive error. When constant, dust and feathers, or otherwise dogs, cats and horses are causal; when seasonal, pollens. (§ 179, III.)

§ **833.** II. **Superficial Alterations** require examination in a good light, and with the help of a binocular loupe.

1. REDNESS of one or both eyes may be *generalised* when it is due to conjunctivitis, iritis or acute glaucoma. *Localised* redness suggests corneal ulceration, episcleritis or a blocked tear duct. (For further details a special text-book should be consulted.)

2. PROPTOSIS (exophthalmos) is an undue prominence of one or both eyeballs and may be detected by standing behind the patient and looking down over the forehead. Apparent exophthalmos may be seen in high myopia.

Unilateral proptosis occurs in (1) Exophthalmic goitre, (2) Exophthalmic ophthalmoplegia (Anterior pituitary excess), (3) Tumour or aneurysm in or invading the orbit, (4) Irritation of the Cervical Sympathetic in the neck or thorax, (5) Orbital cellulitis or periostitis, (6) Cavernous sinus thrombosis, (7) Facial asymmetry, and (8) Nasopharyngeal tumour. Exophthalmos from goitre is usually bilateral: some of the others become so. *Recession* of the eyeballs (Enophthalmos) occurs in paralysis of the cervical sympathetic, the other symptoms of which are pseudo-ptosis, narrowing of the ocular fissure, contraction of the pupil, loss of the cilio-spinal reflex (reflex dilatation of the pupil when the skin of the neck is pinched) and absence of sweating over the face and the forequarter of the corresponding side.

3. THE EYELIDS.—The eyelids are puffy in renal disease, cardiac dropsy, angioneurotic œdema, after violent coughing or vomiting, in eyestrain and arsenical poisoning, insect bites, frontal sinus suppuration, Graves' disease and mongolism. *Ptosis*, or drooping of the upper eyelid, may be partial or complete, and unilateral or bilateral. It may be due to (i.) paralysis of the unstriped muscle of Müller in the upper lid (cervical sympathetic palsy), (ii.) paralysis of the striped levator palpebræ superioris muscle when it is part of an Oculo-motor Nerve palsy due to tabes, leaking intracranial aneurysm, encephalitis lethargica, and mid-brain tumour. (iii.) Ophthalmoplegic migraine, (iv.) Myopathy affecting the face, (v.) Myasthenia Gravis, (vi.) Hysteria, or (vii.) as a Congenital condition. Ptosis is usually accompanied by a compensatory overaction of the frontalis muscle, except in myasthenia gravis (where

the frontalis is also paralysed) and hysteria. Blepharospasm is an *involuntary clonic twitching* of the eyelid. *Inability to close the eyelids*—Lagophthalmos—is due to weakness of the orbicularis oculi and is met with in Bell's (facial) palsy, myopathy and myasthenia gravis.

Lagging Behind of the Upper Eyelids when the patient looks down constitutes Von Graefe's sign in exophthalmic goitre (see § 186). In *Lid Retraction* a band of white sclerotic is seen between the upper lid margin and the iris. In *Proptosis* a white band of sclerotic is also visible between the iris and the margin of the lower lid.

The Slit Lamp is a refinement in the apparatus at our disposal for the minute examination of the living tissues of the eye. It consists of a binocular microscope carried on an arc and working in concert with a source of light shedding an intense beam on the part examined. By its means the minutest alterations in the anterior parts of the eye (including the anterior vitreous) can be examined, and the detailed progress of pathological phenomena noted.

§ 834. III. **Defects of Vision** may consist of (1) defective sense of form or acuteness of vision, (2) alteration in the field of vision, (3) defective sense of colour.

(1) Acuity of Vision implies the estimation of forms of objects. It may be roughly tested by asking the patient to count the number of fingers held up before him. The defect may be so great that he cannot perceive light from darkness. The eyes must be examined separately, as it is often found that defect of one eye has existed a long time without the patient being aware of it. If with defective acuity of vision the external parts of the eye are normal, the media transparent, and the ophthalmoscope reveals no disease, it is probable that the patient suffers from an error of refraction, tobacco amblyopia, or retrobulbar neuritis. Eye-strain is due to protracted overaction of the intrinsic muscles of the eye, and is manifested by headache, eye-ache, blepharitis and styes, blinking (in children), conjunctival hyperæmia or Vth nerve neuralgia.

Errors of Refraction.—For accurately testing the visual sense of form, the patient is asked to read Snellen's types at a given distance and the visual acuity is recorded as a fraction, the numerator being the distance at which the patient is placed from the types, usually 6 (metres), occasionally 20 (feet); and the denominator, the distance at which the healthy emmetrope could read the smallest line which the patient is able to see. This latter figure is printed in small type under each line of letters on the chart and it varies, according to the line, from 5 to 60 (metres). The error of refraction is ascertained (after paralysing the ciliary muscle and iris by homatropin) by placing various lenses in the trial frame before the eyes until it is found which of them completely corrects his error. Convex lenses are indicated by the sign +, concave by the sign —. The defect is measured by the focal length of the lens required to correct his error, and is expressed in diopters, indicated by the sign D. A lens of one diopter has a focal length of 1 metre. Thus, a + 3 D. lens indicates a convex lens with a focal length of $\frac{1}{3}$ metre, being three times as strong as a lens of + 1 D. Retinoscopy is a more accurate method of testing refractive errors (below). In *myopia* (or near sight) the image is formed in front of the retina, and the patient cannot see distant objects clearly. In *hypermetropia* (or far sight) the image is formed on a plane behind the retina, and the patient has to accommodate powerfully for near objects. Both may be due to defective shape of the globe. Concave lenses are used to correct myopia, and convex to correct hypermetropia. In *presbyopia* the rigidity of the lens renders it either difficult or impossible to accommodate for near objects; it generally shows itself at the age of 45. The far vision of presbyopes may be good, though they cannot read or see near objects distinctly without convex glasses. *Astigmatism* is a non-correspondence of the curve in the principal meridians of the cornea, the curvature being similar to that of the bowl of a spoon. In *simple* astigmatism one meridian is myopic or hypermetropic; in

compound astigmatism the error of the two meridians, though of the same kind, differs in degree; in *mixed* astigmatism there is a myopic error in one meridian, and a hypermetropic error in the other meridian; in *irregular astigmatism*, usually the result of scarring, the curves of the cornea vary even in the same meridian. Astigmatism is detected accurately by a skilled examination with retinoscopy, with the ophthalmometer or better still with a crossed cylinder.

In **Retinoscopy** a plane mirror is used. No details of the fundus are visible in this way, but an evenly red field is seen—the red reflex. On tilting the mirror up and down or from side to side the red reflex will be found to move in the direction of the tilting in Hypermetropia and against the tilting in Myopia. In Hypermetropia this movement will be neutralised by a plus sphere of a power one dioptre more than the degree of Hypermetropia present, this difference being due to the fact that one works at a distance of one metre. Therefore, a movement in a patient with a Hypermetropia of 2 dioptres will be neutralised by a + 3 D. lens and an Emmetrope will require a + 1 D. lens to neutralise the movement. The use of a lens of a higher power will produce an artificial Myopia and therefore reverse the movement. No movement and no lens represents a Myopia of 1 dioptre, and neutralisation of movement against will be brought about by a lens 1 dioptre less powerful than the measure of the Myopia present. Similarly, the use of a minus lens of a higher power would produce an artificial Hypermetropia with reversed movement.

In astigmatism the reflex tends to be rectangular in shape and is neutralised by different lenses at opposite axes; the difference in power of the lenses is the measure of the patient's astigmatism.

The movement of the red reflex is accompanied by the movement of a shadow, and the movement of this shadow may be observed instead of following the reflex, but most observers find the latter to be the more satisfactory method.

Opacities in the media may be detected as dark shadows upon the red field. The radiating streaks of commencing cataract or moving opacities in the vitreous may be thus detected, the former ceasing to move when the movement of the eyeball ceases.

(2) The Field of Vision is the extent of the picture presented to the eye at any given moment. It may be roughly tested by instructing the patient to cover one eye and look fixedly at the opposite eye of the examiner at a distance of about 3 feet. You then hold up one finger on each side of you in turn midway between, and bring it gradually towards the centre, asking the patient to say "yes" the moment it comes into his view. By repeating this procedure in different directions you will roughly ascertain in what part of his field the vision is defective. The dimensions of the visual field can be tested accurately only by the perimeter (below). *Scotoma* is a word used to indicate a spot of blindness or imperfect vision within an otherwise healthy field—*e.g.*, a central scotoma is a blind spot in the middle of the visual field.

The Perimeter.—One eye is covered with a shade and the patient places his chin on the chin-rest. He must be educated to keep his eye steadily fixed on the spot opposite, while the operator, by turning a handle, moves the test object along the arc of the perimeter from periphery to centre. The position in which the patient can first see the test object (while looking fixedly all the time at the central spot) is then marked on the chart by an automatic pricker. With apathetic patients this is a tedious operation, and without due care erroneous results may easily be obtained. The perception of colours in the peripheral field varies normally in extent with the different colours. Thus, from without inwards they are seen in the following order: white, blue, yellow, red, green. For the purpose of detecting minute central and paracentral scotomata, such as are met with in retrobulbar neuritis and pituitary enlargements, the ordinary perimeter is not sufficiently accurate. The *Bjerrum's screen*, with small test objects in skilled and patient hands and with reliable patients, gives very satisfactory results, which enable the examiner to obtain valuable diagnostic indications otherwise unobtainable; it is of particular value in determining any increase in the size of the blind spot, or in demonstrating the arcuate scotomata of chronic glaucoma.

(3) COLOUR VISION may be tested by matching coloured wools, or by the Edridge Green lamp or by Ishihara test cards.

Colour Blindness (achromatopsia) is a symptom in some diseases of the retina and in optic atrophy. Red-green blindness as a congenital or familial deficiency is common: yellow-blue blindness is rare. In tobacco blindness and in some other forms of retrobulbar neuritis, anything from minute scotomata for red and green to complete colour blindness may occur (§ 836).

§ **835. The Causes of Defective Vision** without very obvious ocular changes may be considered under 1. UNILATERAL BLINDNESS, 2. BILATERAL BLINDNESS, 3. DEFECTS OF THE VISUAL FIELDS, and 4. NIGHT BLINDNESS. The defective vision due to errors of refraction has already been dealt with.

AMBLYOPIA is diminished vision, AMAUROSIS total loss of vision, sometimes without discoverable changes in the fundi or error of refraction. It is obvious that amblyopia may be due either to some functional disturbance of the visual apparatus, or to some gross lesion of the brain or paths of vision.

1. UNILATERAL BLINDNESS may be sudden or gradual. (*a*) *Sudden unilateral blindness* occurs in (i.) retrobulbar neuritis, (ii.) thrombosis of the central retinal vein, or embolism or spasm of the central retinal artery, (iii.) sudden intra-ocular hæmorrhage, (iv.) detachment of the retina or (v.) hysteria. (vi.) A patient may suddenly discover blindness in one eye which has long been defective. See retrobulbar neuritis, § 851.

(*b*) *Gradual unilateral blindness* may result from (i.) Cataract, (ii.) Chronic glaucoma, (iii.) Retrobulbar neuritis, (iv.) Tumour of or pressure on the optic nerve, (v.) Optic atrophy in tabes dorsalis or disseminated sclerosis, (vi.) Local disease of the choroid or retina, *e.g.*, Choroido-retinitis, (vii.) Amblyopia ex anopsia (usually in history of squint).

§ **836.** 2. BILATERAL BLINDNESS may be sudden or gradual.

(*a*) *Sudden bilateral blindness* may occur in: (i.) Exposure to blinding sunlight, snow, or electric light, (ii.) Hysteria, (iii.) Sudden and copious hæmorrhage from the stomach, bowels or uterus, (iv.) Uræmia, (v.) Diabetes, (vi.) Insulin hypoglycæmia, or from (vii.) Local Trauma, *e.g.*, gunshot wounds to the calcarine cortex and occipital poles.

(*b*) *Gradual bilateral blindness* may be due to (i.) Cataract, (ii.) Chronic glaucoma, (iii.) Tobacco Amblyopia, (iv.) Toxic Amblyopia, (v.) Pituitary Tumour or Suprasellar cyst, (vi.) Local disease of the Retina and Optic Nerve, *e.g.*, Primary Optic Atrophy as in tabes, or Consecutive Atrophy, following papillœdema or papillitis. (vii.) Leber's Hereditary Optic Atrophy (§ 852).

Tobacco Amblyopia occurs sometimes in hard smokers of over three or four ounces per week, or in debilitated persons and in women from a much smaller quantity. The patient first complains of defective vision in bright light; he sees better at dusk than at noon and mistakes silver for copper coins. The defect is slowly progressive, becoming most marked in the *central* field, and there is a central colour scotoma, especially for red and green. At first there may be no changes in the fundi, then the discs become slightly congested in the earlier stages, and pale and atrophied, especially on the temporal side, in the later. Defective vision is the earliest symptom to attract the patient's notice. It arises from a chronic retrobulbar neuritis. The pupil reaction, both in chronic and acute retrobulbar neuritis, is characteristic. The pupil contracts normally to light, but does not remain contracted under the same light stimulus and dilates slightly after a second or so. The local application of acetyl-choline by iontophoresis is recommended.

Toxic Amblyopia is exemplified in uræmia and diabetes. It may also be produced by large doses of quinine, bisulphide of carbon in india-rubber manufacture, iodoform, dinitrobenzol and arsenical preparations like tryparsamide. Little in the way of treatment can be done for such cases, unless they are seen early, when removal of the cause and functional rest to the structures involved may lead to recovery.

§ 837. 3. Defects in the Visual Fields.

Central Scotoma, or a blind patch in one or both visual fields, may occur from (1) Retrobulbar Neuritis (see § 851), (2) Toxic Amblyopia (see § 836), (3) Early Optic Atrophy (and Leber's Disease), (4) Retinal hæmorrhage, (5) Central opacities in the lens or cornea. As a temporary phenomenon it occurs in (6) Migraine.

Hemianopia has been considered in § 676, to which the reader is referred. The following types are recognised:

(*a*) *Homonymous Hemianopia* or loss of the corresponding halves of the visual fields, is a hemiplegia of the visual fields and results from a lesion in the contralateral visual part of the internal capsule or the occipital lobe. A right homonymous hemianopia means abolition of the right halves of the visual fields. Apart from its occurrence in migraine, it is due to a gross central lesion situated in some part of the

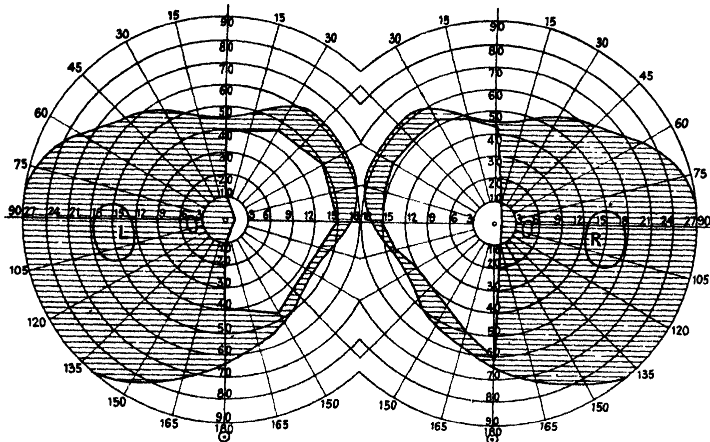


Fig. 189*a*.—Visual Fields showing Bitemporal Hemianopia. Case of Acromegaly due to pituitary adenoma in a woman aged 30 years; two years' duration.

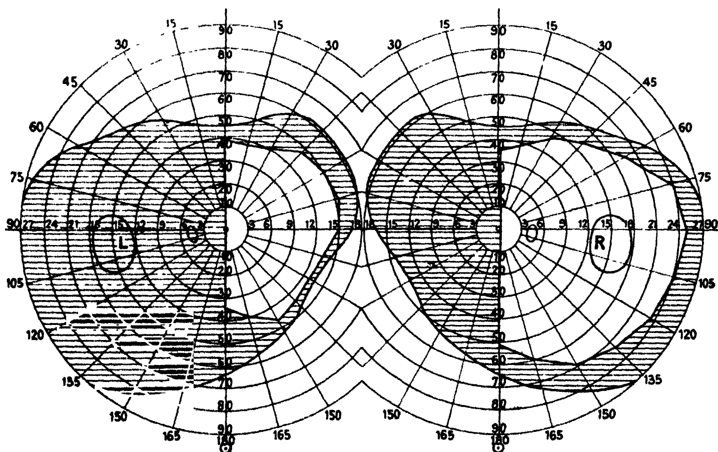


Fig. 189*b*.—Visual Fields showing Left Homonymous Hemianopia. Case of large glioma of the right temporal lobe involving the optic radiation.

visual path behind the chiasma, (i.) in the optic tract, or (ii.) behind the corpora quadrigemina, *i.e.*, the hinder end of the internal capsule, (iii.) the optic radiation in the temporal or occipital lobe, or (iv.) in the calcarine cortex of the occipital lobe. By employing Wernicke's test (see below) the first may be excluded. For the rest, the precise position and character of the lesion can only be diagnosed by the accompanying symptoms. (*b*) *Quadrantic Hemianopia* is a loss of quadrants of corresponding sides of both visual fields. It results from (i.) a lesion deep in the temporal lobe, involving the "temporal knee" of the optic radiation, (ii.) a lesion of the calcarine cortex, (iii.) a lesion either above or below the optic chiasma. Lesions involving the *upper* fibres of the calcarine cortex, optic radiations or optic chiasma, produce blindness of the *inferior* quadrants of the visual fields and *vice versa*. (*c*) *Bitemporal Hemianopia* or loss of the temporal halves of both visual fields arises from a lesion in the neighbourhood of the optic chiasma, *e.g.*, Pituitary tumour in Acromegaly, distended infundibulum in Hydrocephalus or basal meningitis. (*d*) *Nasal Hemianopia* or loss of the nasal halves of both visual fields is of theoretical interest and would result from bilateral lesions on the outer margins of the optic chiasma or optic tracts, *e.g.*, pressure from aneurysmal dilatation of both internal carotid arteries. (*e*) *Altitudinal Hemianopia* or loss of the upper or lower halves of the visual fields might conceivably eventuate from (i.) optic neuritis, (ii.) a bilateral lesion involving the upper or lower lips of the calcarine cortex on the two sides, *e.g.*, from a tumour situated mesially between the occipital poles, (iii.) disseminated sclerosis, (iv.) a suprasellar cyst, or (v.) (temporarily) in migraine.

Causes of Hemianopia.—Hemianopia may result from: (1) Cerebral Tumour, (2) Pituitary Tumour, (3) Hydrocephalus, (4) Cerebral thrombosis, embolism or hæmorrhage, (5) Pyæmic Cerebral Abscess, (6) Syphilitic encephalitis or meningitis, (7) Tuberculomata, (8) Aneurysms.

Wernicke's hemianopic pupillary reflex helps you to determine the seat of a lesion in hemianopia. In hemianopia due to lesions in the optic radiations or occipital cortex, a beam of light thrown directly on the blind half of the retina by the concave mirror of the ophthalmoscope, produces contraction of the pupils, as the pupillary light reflex arc is intact (see Fig. 164). In hemianopia, due to lesions of the optic tract or anterior to this, pupillary contraction will fail to occur.

4. Night Blindness (*nyctalopia*) is defective vision in dim lights. It is a feature of (i.) retinitis pigmentosa, (ii.) syphilitic retinitis, or it may be without fundus changes, (iii.) as a congenital deficiency, or (iv.) as hysterical night-blindness. Concentric contraction of the visual field is met with and, finally, complete blindness. Acute night-blindness may attack those with defective nutrition who have been exposed to a very strong sun or artificial light, and in those cases the prognosis is good. Night vision is of particular importance for night-flying. Bishop Harman has elaborated a disc-spotting test for the dark adapted eye: improvement of function is brought about by breathing oxygen and in some cases by vitamin A administration.

§ 838. IV. **Condition of the Pupils.** The tonic dilator of the pupil is the cervical sympathetic (dilator iridis), the tonic constrictor of the pupil is the oculomotor nerve (ciliary muscle and sphincter iridis). Each of the three muscles may be paralysed separately: paralysis of the last two is known as *ophthalmoplegia interna*. The pupils must be tested with regard to their size, shape, equality, reaction to light, direct and consensual, and to accommodation (see Examination of the Nervous System, § 703).

Abnormal Dilatation of the Pupil (Midriasis) is met with in (1) Neurasthenia, Anxiety Neurosis and other states of Fear, (2) Hyperthyroidism, (3) Anæmia, or from: (4) Midriatic Drugs: Atropine, Homatropine, Belladonna, Cocaine. In nervous disease it may be the result of (5) paralysis of the sphincter pupillæ (oculomotor paralysis from post-diphtheritic, syphilitic or encephalitic paralysis), or (6) irritation of the dilator pupillæ (cervical sympathetic stimulation, *e.g.*, cervical tumour). It is met with in (7) Optic Atrophy, (8) Glaucoma, (9) in deep coma or collapse and in trauma (traumatic midriasis).

ABNORMAL CONTRACTION OF THE PUPIL (Miosis) is met with in (1) Pontine Hæmorrhage; (2) Tabes dorsalis and G.P.I.; (3) Disease of the Cervical portion of the spinal cord, *e.g.*, Syringomyelia; (4) Iritis; (5) Foreign body in the eye, or from: (6) Miotic Drugs: Eserine, Pilocarpine, Opium; (7) Congenital microcoria; (8) Old age.

SLIGHT INEQUALITY OF THE PUPILS may be observed in health, especially as the result of a refractive error. The above causes may operate.

IRREGULARITY OF OUTLINE OF THE PUPILS.—This may be due to (1) old iritic adhesions, or may result from (2) previous iridectomy or trauma. Deviations from the circular shape occur in (3) Neurosyphilis, *e.g.*, tabes, G.P.I., or (4) Glaucoma —in which the pupil may appear twisted. (5) Coloboma iridis is a congenital deficiency of the iris, usually in the lower part, generally on both sides. The defect may be continued backwards, involving the choroid below and even the optic nerve, while the vision is always more or less impaired.

EX-CENTRIC PUPILS (Ectopia pupillæ) may be (1) Congenital or observed after (2) iritis, or (3) in mid-brain lesions, *e.g.*, syphilis, encephalitis, neoplasms.

PUPILLARY REFLEX TO LIGHT.—In testing the light reflex, *both* eyes should be covered for half a minute, and each uncovered in turn, opposite a bright light which makes the pupils contract. The (*a*) direct and (*b*) consensual reactions should be tested (see § 703). In a good light the iris can sometimes be observed to contract and dilate rhythmically, a phenomenon known as *hippus*. Hippus may be seen in normal eyes or in some cases of chorea.

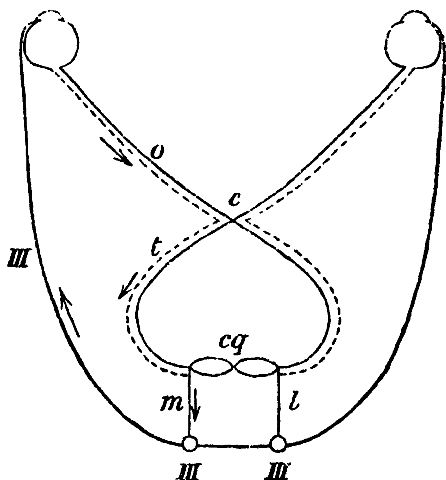


FIG. 190.—Diagram showing REFLEX ARCS concerned in the MOVEMENTS OF THE PUPIL (Horizontal Plane) —*o*, optic nerves; *c*, optic chiasma; *t*, optic tract; *cq*, corpora quadrigemina; *III*, third nerves and nuclei; *m* and *l*, Meynert's fibres communicating between the third nuclei and the corpora quadrigemina.

The *pupillary light reflex* depends on the integrity of the retina and the following tracts (see Fig. 190): the optic nerve (*o*), the chiasma (*c*), the optic tract (*t*), the superior corpora quadrigemina (*cq*). These last-named nuclei (*cq*) are connected by means of the colliculo-ocular (Meynert's) fibres (*m*, *l*), with the nuclei of the third nerves (III) situated in the floor of the aqueduct of Sylvius. The fibres of the third nerve, through the long or short ciliary branches, conduct impulses to the sphincter iridis, causing pupillary contraction.

§ 839. Loss of the PUPILLARY REFLEX TO LIGHT (light iridoplegia) may be produced by a lesion situated anywhere in these afferent or efferent tracts. Loss of direct reflex to light, together with preservation of brisk pupillary contraction on convergence-accommodation constitutes the *Argyll-Robertson phenomenon*. Argyll-Robertson pupils may be large or small and the phenomenon may be unilateral or bilateral. In addition, such pupils may be (i.) ex-centric in position, (ii.) irregular in outline, (iii.) unequal in size.

Causes of Argyll-Robertson Pupils: Clinically: the Argyll-Robertson pupil is found in syphilis affecting the vicinity of the aqueductus Sylvii and the superior colliculi. Apart from the syphilitic lesion, Argyll-Robertson pupils have been observed and reported as a clinical rarity in the following conditions: non-specific encephalitis (*e.g.*, encephalitis lethargica), mid-brain tumours, syringobulbia, in traumatic lesions of the mid-brain, in alcoholic polyneuritis and diabetes mellitus.

The *anatomical* basis of the Argyll-Robertson phenomenon is obscure. The condition may be met with in: (1) lesions of both optic nerves, (2) lesions of both optic tracts, and (3) central lesions of the colliculo-ocular fibres which pass to the anterior end of the third nucleus in the mid-brain. (If one is to insist on small pupils as described by Argyll-Robertson the third situation is the only one possible.) The nerve centre for the pupillary light reflex is the oculomotor nucleus, but recent work has shown that there is no lesion of this nucleus in tabes.

MYOTONIC PUPILS are pupils which react only to very strong light, and on convergence-accommodation may contract slowly, remaining contracted for some seconds, before very slowly dilating again: in many cases only one eye is affected. The condition may be met with in association with absence of tendon reflexes, and may simulate tabes dorsalis, if the true condition of the pupils is not recognised (Adie).

CONVERGENCE—ACCOMMODATION REACTION (Near Reflex).—When looking at a near object the eyes are converged, the ciliary muscles contract (causing increased convexity of the lens) and the pupils narrow. These ciliary and pupillary reactions constitute accommodation. Loss of pupillary contraction on accommodation occurs in: (1) Post-Encephalitis Lethargica, (2) Diphtheria (with palatal paralysis or loss of tendon reflexes), (3) Poisoning with atropine or belladonna, (4) lesions of the oculomotor nerve, (5) Deficient convergence is commonly seen after head injuries, in association with defects of memory and of concentration.

§ 840. **Iritis**, inflammation of the iris, is manifested by (1) immobility, change of colour, loss of pattern, and exudation; (2) pain (which may be absent in serous iritis) dimness of vision and watery discharge; (3) adhesion between the iris and anterior capsule, revealed under atropine; (4) circumcorneal injection, indicating hyperæmia of the ciliary vessels. Care must be taken not to mistake this disease for conjunctivitis or glaucoma, because the treatment suitable for either will make iritis worse. In conjunctivitis the injection is most marked away from the cornea, while in iritis the injection is most marked at the cornea, *i.e.*, pericorneally.

Causes of iritis: Focal sepsis, tuberculous, gonorrhœal, syphilitic, diabetic and other toxæmias may cause it. Syphilitic iritis is usually non-recurrent; the other forms are liable to relapse. Chill, bright light and injury may be determining causes.

Treatment consists in regular applications of atropine drops (1 per cent.), heat, and leeches to the temple. If attended by much pain, aspirin (gr. 15) generally gives complete relief in fifteen to twenty minutes. For chronic iritis dionin drops (4 per cent.) twice daily, along with atropine, may be tried. In recurring iritis, not associated with cyclitis (keratitis punctata), iridectomy in the quiescent stage is a good treatment. If complete annular synechiæ form, iridectomy should be performed, to prevent secondary glaucoma. The use of tuberculin should not be forgotten.

§ 841. **Ophthalmia Neonatorum** has always been a frequent cause of blindness: though the number of notifications changes little, resultant blindness has been greatly reduced. The causal organism is the gonococcus in more than 60 per cent. of cases, and next in frequency is the streptococcus. *Treatment.* Penicillin drops (5,000 units per c.c.) frequently applied, have replaced all other forms of treatment, when the organism is penicillin sensitive. Otherwise the sulphonamides should be used in doses of ½ G. six-hourly for five days.

§ 842. **Cataract.**—The following points are of interest: *Lamellar cataract* is a post-natal condition associated with and due to rickets; the enamel of the margins of the following permanent teeth of both jaws

is defective—both incisors, the canines and the first molars, *i.e.*, the enamel which is being laid down between the sixth and twenty-fourth month of life. The malady is therefore preventable. It also occurs with parathyroid tetany. *Senile cataract* may also be preventable, but the cause is unknown. Sodium iodide drops or ointment are supposed to limit the rate of progress of cataract and even cause its disappearance in early cases. The urine should be examined for sugar and before operation all carious teeth should be removed, the tonsils and sinuses examined, and the lachrymal sac, if infected, should be removed.

§ 843. **Cervical Sympathetic Lesions** may be due to (*a*) Paralytic, or (*b*) Irritative Lesions.

(*a*) *Paralytic lesions* produce (i.) small pupil, (ii.) loss of cilio-spinal reflex, (iii.) enophthalmos, (iv.) narrowing of the ocular fissure, with ptosis, and (v.) absence of sweating on the corresponding side of the face and forequarter.

(*b*) *Irritative lesions* produce (i.) dilated pupil, (ii.) brisk cilio-spinal reflex, (iii.) exophthalmos, (iv.) widening of the ocular fissure, with lid retraction, and (v.) increased sweating on the corresponding side of the face and forequarter.

The *Causes* of paralytic or irritative lesions of the Cervical Sympathetic are: (1) Intrathoracic aneurysm, Neoplasm or Enlarged Mediastinal glands (Lymphadenoma, etc.); (2) Apical pleural fibrosis in Pulmonary Tuberculosis, and Apical Pneumonia; (3) Tumours in the neck; (4) After operation for neck glands; (5) Cervical Rib; (6) High Brachial Plexus lesions, *e.g.*, trauma, secondary deposits; (7) Disease or injury of the cervical cord from implication of the bulbo-spinal sympathetic fibres, *e.g.*, syringomyelia, spinal tumours, fracture-dislocations; (8) Mid-brain lesions; (9) Myasthenia gravis; (10) Exophthalmic goitre; (11) Congenital Sympathetic lesions (see Fig. 173).

§ **844. V. Ocular Movements.**—The external muscles of the eyeball (as distinct from the internal or involuntary muscles of the eye) are six in number, and they are supplied by three cranial nerves: External rectus (VI. nerve); superior oblique (IV. nerve); internal, superior, and inferior recti and inferior oblique are supplied by the III. nerve (which also, it will be remembered, supplies the levator palpebræ, the sphincter fibres of the iris, and the ciliary muscle). It follows therefore that:

Complete paralysis of the third nerve (oculomotor) is attended by	Ptosis; external strabismus; pupil dilatation and immobility; loss of accommodation; inability to move eyeball inwards or upwards, and only imperfectly downwards; slight protrusion of the eyeball: crossed diplopia.
Paralysis of the sixth nerve (abducens) is attended by	Internal strabismus; inability to move eye outwards; homonymous diplopia.
Paralysis of the fourth nerve (trochlear) is attended by	Slight deviation of cornea upwards; homonymous diplopia on looking downwards.

Defects in the ocular muscles are revealed (1) by defective movements of the eyeball towards the side of the paralysed muscle; (2) by false orientation; (3) by squint in pronounced cases; (4) by diplopia (double vision); (5) by vertigo; and (6) by the head being tilted towards the side of the paralysed muscle.

The more recent the paralysis the more marked are the above symptoms, while in old paralyses the symptoms are more mixed.

Deficient movement of the eyeball—(Fig. 191)—*indicates paralysis of*

outwards	external rectus—sixth nerve.
inwards	internal rectus—third nerve.
upwards	*superior* rectus, *inferior* oblique } third nerve.
downwards	*inferior* rectus—third nerve. *superior* oblique—fourth nerve.

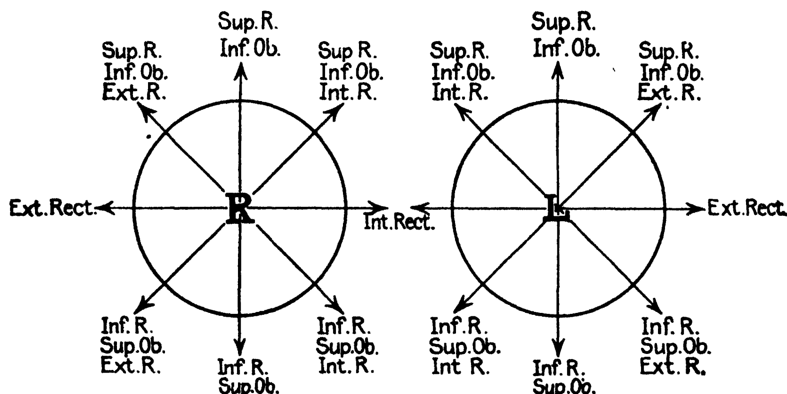


FIG. 191.—MACGILLIVRAY'S DIAGRAM showing muscles concerned in ocular movements. Lateral movements—one muscle. Vertical movements- two muscles. Oblique movements—three muscles; *e.g.*, Elevation and adduction—the resultant of the forces on each side, *i.e.* Elevation (Sup. Rect. and Inf. Ob.) and adduction (Int. Rect.).

Method of Detecting the Affected Eye and Paralysed Muscle.—Place a red glass before the patient's left eye, and hold a lighted candle before him in a dark room, on a level with his eyes at 2 yards distance. Suppose that it is found that the red image overlaps, or is a little to the left of the white image, and both images are on the same level, to determine which muscle is affected the candle must be moved to the right and to the left, and we must notice *in which direction the distance between the images becomes increased.* On moving the candle to the right the image approaches till only one candle is seen, and on moving to the left, the distance between the true and false images increases. Bearing in mind the rule that the weakened muscle is on the same side as the direction in which diplopia increases, it is evident that either the left external rectus or the right internal rectus (which turn the eyes to the left) is affected. Ask then on which side the red image appears. If on the left of the white image, homonymous diplopia is present; therefore the left *external* rectus is the paralysed muscle. If, however, the red image is to the right of the white image, crossed diplopia is present; therefore the right *internal* rectus is the paralysed muscle. Paralysis of the superior or inferior rectus, and of the superior or inferior oblique, gives rise to vertical diplopia. The former causes crossed diplopia, the latter homonymous diplopia. Loss of motion upwards is due to paralysis of the third nerve; loss of motion downwards may be due to paralysis of the inferior rectus (third nerve) or the superior oblique (fourth nerve). Werner's diagrams (Fig. 192) simplify the detection of the affected muscle in vertical diplopia. The black lines in the diagrams represent the true images, the dotted lines the false images. The dotted lines extend above and below the black lines, indicating that the false images are higher and lower than the true images. The names of the muscles in the upper and lower part of the diagrams indicate that the diplopia is caused by upward and downward movements respectively

of the eyes when these muscles are affected. Thus, for example, in paralysis of the right inferior rectus an analysis of the diagram shows that (1) the diplopia occurs with downward movements of the eyes; (2) the diplopia is crossed, the false being to the left of the true image; (3) the false image has its upper part inclined towards the

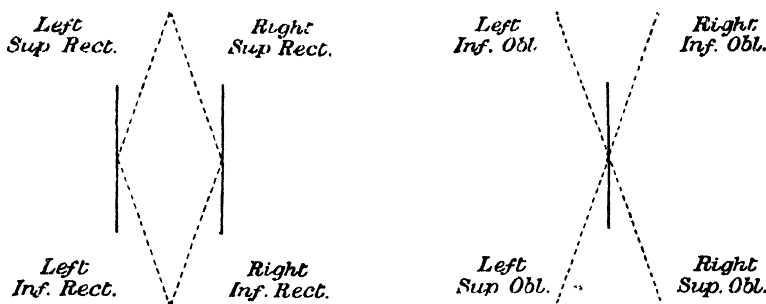


FIG. 192.—WERNER'S DIAGRAMS for detecting which is the affected muscle in cases of diplopia.

true image; and (4) the false image lies lower than the true one. With the oblique muscles it must be remembered that the superior oblique moves the eye downwards, and therefore the false image due to the paralysis of the superior oblique appears on moving the eye downwards. By remembering the diagrams it is comparatively easy to diagnose the paralysed muscle causing a diplopia.

§ 845. **Squint** or strabismus is a want of parallelism between the visual axes when looking at a distant object. It is called *convergent* when one eyeball looks inwards and *divergent* when one eyeball looks outwards. In *children* it is chiefly associated with some error of refraction—hypermetropia (internal strabismus, the commonest in children), or myopia (external strabismus). In many cases the patient or a parent is left-handed: the refractive error may be small and a psychological factor may be of great importance. In adults or children, squint of recent acute origin is due to definite paralysis of an ocular nerve.

Diplopia or double vision is, in many cases, due to weakness of one or more ocular muscles, but may be due to any condition altering the visual axes, *e.g.*, unequal proptosis, fracture of the orbit. Erroneous projection—*i.e.*, error in judging the position of objects—and vertigo (due to the same cause) are invariably associated with diplopia.

Clinical Investigation of Squint.—There are three steps. (1) Diagnosis of the Type of Squint, (2) Diagnosis of the Affected Muscle, (3) Diagnosis of the Position of the Lesion and its cause.

(1) DIAGNOSIS OF THE TYPE OF SQUINT.—Squints are of three kinds: (A) Concomitant, (B) Paralytic, and (C) Alternating.

(A) *Concomitant Squint* is met with most frequently in children. (i.) It comes on slowly. (ii.) Each eye, when the other is covered, moves perfectly in all directions, there being no paralysis, but when examined together, the squint is present in all positions of the eyeball. (iii.) Spontaneous diplopia is absent. (iv.) The affected eye follows the sound eye

with equal defect in all directions (hence the name "concomitant"), so that the defect of parallelism is the same in all directions. It is due, in about 90 per cent. of cases, to hypermetropia or other error of refraction, or to a defect in the fusion faculty, and when these are remedied, particularly with the aid of orthoptic exercises, in childhood the squint may disappear.

(B) *Paralytic Squint* is met with in children or adults. (i.) It usually appears suddenly. (ii.) It is always accompanied by double vision. (iii.) There is limitation of movement of the globe corresponding to the direction of traction of the paralysed muscle. Paralytic squints are due to ocular paralyses resulting from intracranial or other serious mischief.

(c) *Alternating Squints* are nearly always associated with left-handedness in the patient or family, and sometimes with stammering.

(2) Diagnosis of the Affected Muscle.—In *Paralytic Squint* the affected muscles *can usually be recognised by simply testing the external ocular movements as described* in § 703.

In cases of slighter weakness, and in *Concomitant Squint,* tell the patient to look at an object straight in front of him, that being the normal position of the eyes at rest, and fix some object. The eye with which he fixes is the normal eye. The deviation of the affected eye from the middle line is known as the "primary deviation." Now partially cover the sound eye and let him fix with the affected eye. The sound eye will now be found to deviate ("secondary deviation"). In concomitant squint the primary and secondary deviations are equal, but in paralytic squint, the secondary exceeds the primary. The patient unconsciously turns his face *towards the side of the weak or paralysed muscle.* To detect which is the affected muscle, hold a pencil vertically in front of the patient and move it rapidly to the right, to the left, and in various directions, and ask him whether he can see two pencils in any of these directions. *The weakened muscle is on the same side as the direction in which the double vision appears.* Diplopia may be homonymous or crossed. In simple or homonymous diplopia the false image lies on the same side as the affected eye; in crossed diplopia the false image lies on the side opposite to the affected eye. Paralysis of the external rectus, or obliques, causes homonymous diplopia; paralysis of the internal rectus, or superior or inferior recti, causes crossed diplopia.

Monocular Diplopia is recognised by the persistence of diplopia when one eye is closed. It is met with in (i.) Hysteria and in (ii.) Malingering. (iii.) It may arise from defects of the media.

Treatment—Orthoptic training, special exercises for promoting fusion and increasing amplitude, are becoming routine treatment.

(3) Diagnosis of the Position of the Lesion and its Cause.—This is of importance in Paralytic Squint.

(a) *There is Diplopia, squint, and defective movement of* one eye *towards the direction of traction of the affected muscle.* The lesion is in the peripheral nerve (III, IV or VI).

The *Causes* of this type of Diplopia are:

I. Rheumatism.
II. Arterio-sclerosis.
III. Syphilis.
IV. Meningitis.
V. Trauma.
VI. Cerebral Tumour or Aneurysm.
VII. Subarachnoid Hæmorrhage.
VIII. Polyneuritis.
IX. Herpes Ophthalmicus.
X. Gradinego's Syndrome.
XI. Cavernous Sinus Thrombosis.
XII. Syndrome of the Sphenoidal Fissure.

I. RHEUMATISM produces a sudden oculomotor paralysis, usually of the external rectus muscle. There is severe pain in the face and in the eye itself with paralysis of one or more ocular muscles. The condition is analogous to Bell's palsy.

II. ARTERIO-SCLEROSIS.—Isolated external rectus palsy in the aged may be due to pressure on the sixth nerve in its long intracranial course by a distorted atheromatous artery.

III. SYPHILIS.—A chronic syphilitic lepto-meningitis may cause isolated ocular palsies in tabes and other forms of neuro-syphilis.

IV. MENINGITIS of tuberculous or other origin (see § 726) may cause diplopia.

V. TRAUMA.—Blows without fracture, or fractures involving the anterior or middle fossa of the skull may cause damage to the oculomotor nerves and cause diplopia. In other cases, the displacement of the orbit or damage to its muscles resulting from the fracture is the cause of the diplopia. Diplopia may follow spinal anæsthesia and frontal sinus operation.

VI. INTRACRANIAL TUMOUR OR ANEURYSM.—Tumours of the base of the skull, pituitary growths, or internal carotid aneurysm, may cause oculomotor paralysis. Small berry aneurysms on the Circle of Willis may cause unilateral headache and oculo-motor palsy (ophthalmoplegic migraine). In increased intracranial pressure from any cause whatsoever (*e.g.*, Hydrocephalus) the sixth nerve may be damaged in its long intracranial course by pressure from above, with resultant external rectus palsy.

VII. SUBARACHNOID HÆMORRHAGE may be massive with unconsciousness, and later the patient may develop a paralytic squint, with hæmorrhage near the optic disc, and field defects.

VIII. POLYNEURITIS.—The polyneuritis of alcoholism, diphtheria, lead, etc. (see § 793), may be accompanied by external ocular palsies.

IX. HERPES OPHTHALMICUS may be followed by external ocular palsies, pupillary abnormalities, or even by optic atrophy.

X. GRADINEGO'S SYNDROME.—In association with acute suppurative mastoiditis in children, an external ocular palsy develops (usually the external rectus muscle) with pain referred to the distribution of the trigeminal nerve on the face. Other cranial nerves, *e.g.*, facial, hypoglossal, may be affected on the same side. The syndrome is believed to be due to localised meningitis or granulations at the tip of the petrous temporal bone. The symptoms clear up completely some weeks after surgical treatment of the mastoid infection.

XI. CAVERNOUS SINUS THROMBOSIS (see § 738).—In pyæmic conditions, with rigors and high fever, sudden unilateral proptosis develops with œdema, external ocular paralysis of the nerves running in the cavernous sinus (see Fig. 169), blindness and retinal hæmorrhage. The symptoms usually become bilateral within a few hours or days.

XII. SYNDROME OF THE SPHENOIDAL FISSURE.—There is acute supra-orbital pain, and pain in the eye, followed by signs of paralysis of the oculomotor, trochlear or abducens nerves. There may be sensory impairment over the supra-orbital nerve. The condition is supposed to be due to periostitis of the bones of the sphenoidal fissure and clears up in some months, after treatment with iodides and salicylate. Syphilis or orbital neoplasm may cause similar signs.

(*b*) *There is Diplopia, squint, and defective conjugate movement of* BOTH EYES *to the right, left, upwards or downwards*, or *in all directions* (ophthalmoplegia externa). The lesion is IN THE MID-BRAIN and is either NUCLEAR or SUPRA-NUCLEAR.

There may be associated pupillary abnormalities. The functions of the oculomotor nuclei in the mid-brain from before backwards are : (1) Pupillary reflex to light, (2) Convergence-Accommodation mechanism, (3) Upward, (4) Downward, and (5) Lateral movement of both eyes. The

symptoms of "crossed paralysis" resulting from lesions in the mid-brain are described in § 670.

The Causes of *Conjugate Ocular Paralyses* are:

I. Mid-brain Tumours.
II. Mid-brain Vascular Lesions.
III. Myasthenia Gravis.
IV. Encephalitis Lethargica.
V. Neurosyphilis.
VI. Diphtheritic Polyneuritis.
VII. Chronic Bulbar Palsy (motor neurone disease).
VIII. Botulism.

I. MID-BRAIN TUMOURS.—Tumour of the crus, pons or pineal body, produces loss of conjugate upward movement of the eyes with pupillary abnormalities, and progressive headache and vomiting. The symptoms are slowly progressive.

II. MID-BRAIN VASCULAR LESIONS.—Thrombosis, embolism, or hæmorrhage, may produce conjugate ocular paralysis of sudden onset usually with hemiplegia.

III. MYASTHENIA GRAVIS shows diplopia, ptosis, and loss of conjugate movement of the eyes. The paralyses may appear only in fatigue or may be permanent; their variability is characteristic. Other signs of myasthenia (see § 808) may be present. The pupils usually escape, but the light reflex may be lost.

IV. ENCEPHALITIS LETHARGICA.—The eye-signs of encephalitis lethargica are described in § 698. Diplopia is common at the onset of the disease, internal and external ophthalmoplegias may be present in an acute attack or afterwards. Residual paralysis of convergence-accommodation is characteristic of post-encephalitis.

OCULO-GYRIC CRISES (tonic eye-fits) occur in patients with post-encephalitis-lethargica. The patients' eyes suddenly become fixed in a position of conjugate deviation (usually upwards). They can be brought to the resting position by an effort of will, but only for a few seconds. They remain tonically deviated for minutes or hours until the spasm relaxes.

V. NEUROSYPHILIS, especially tabes dorsalis, may cause conjugate ocular palsies.

VI. DIPHTHERITIC POLYNEURITIS occasionally produces diplopia. The paralysis of convergence-accommodation is more characteristic.

VII. CHRONIC BULBAR PALSY (see § 747) is rarely associated with conjugate ocular paralysis.

§ **846.** VIII. **Botulism** is a very rare severe disease following the consumption of artificially preserved food infected with the *B. botulinus*. It occurs in small localised epidemics. The first symptoms, headache and giddiness, occur twelve to twenty four hours after infection. Then come double vision, failure of accommodation and ptosis, with external and internal ophthalmoplegias. Signs of gastro-intestinal irritation are usually absent and the temperature is subnormal. Later, there is dysphagia and dysarthria and general severe muscular asthenia, but sensory changes are absent and the mind is clear. The disease is fatal in over 50 per cent. of cases; death may occur from respiratory failure.

Diagnosis.—The disease bears a superficial resemblance to acute encephalitis lethargica; the history of infection in other consumers of the same tinned food, the absence of lethargy and the subnormal temperature, make the diagnosis clear.

Treatment.—Wash out the stomach and colon repeatedly and keep the bowels open with saline aperients. Alcohol should be given early. An anti-toxic serum is available and may prevent extension of the disease if given before all the exotoxin is fixed in the nervous system.

(c) There is CONJUGATE DEVIATION OF THE HEAD AND EYES. *The lesion is in the* INTERNAL CAPSULE *or above this.*

Following a cerebral thrombosis, embolism, or hæmorrhage, the patient loses the power of turning his eyes to the contralateral side and the eyes are pulled over to the side of the lesion by the unopposed action of the

antagonists. Irritative lesions of the second frontal convolution cause conjugate deviation of the head and eyes to the contralateral side.

Treatment of Squint.—In concomitant squint the visual refractive error is corrected, and afterwards attempts are made to develop binocular vision. In paralytic squint, to relieve diplopia, the false image should be excluded by covering the affected eye; but the cause must always be sought for and treated.

Skew-deviation of the eyes is a transient state of loss of parallelism of the visual axes, so that they cross, one eye looking down and in, and the other up and out. It occurs in acute lesions of the cerebellum or pons.

§ 847. **Nystagmus** is a rapid involuntary oscillation of the eyeballs, usually from side to side (lateral nystagmus), occasionally in a vertical direction (vertical nystagmus), or in a circular direction (rotatory nystagmus). Both eyes are usually involved, though each eye should be separately examined. The movements may be constantly present, but slighter degrees can only be brought out by causing the patient to follow your finger or a bright object to the extreme left or right. Very slight nystagmus can be discovered by direct ophthalmoscopic examination, when the image of the fundus, becoming magnified about fifteen diameters, shows the slightest movements of the eyeball. The symptom is notably present in labyrinthine disorders, disseminated sclerosis, cerebellar tumour, and Friedreich's disease, and in tumour involving the corpora quadrigemina, or one side of the pons. So-called CONGENITAL NYSTAGMUS is generally produced by any condition, such as ophthalmia neonatorum, which prevents the child using his eyes during the first few weeks of infancy when the co-ordination of the extrinsic muscles must be acquired; or in albinos, where, for want of pigmentation, the retina never receives definite images and cannot therefore acquire the power of fixation and muscular co-ordination. OCCUPATIONAL NYSTAGMUS is met with in miners, compositors, iron-founders, and those who work at close quarters, or in conditions of strain with deficient light.

Miner's Nystagmus.—Four stages are described: Latent, subacute, acute and neurasthenic. The latent form is found after leaving work and is elicited on rotation. The subacute is found at work; there are headache, dazzling, giddiness, eye movements (nystagmus), and a defect in visual acuity by day and poor vision at night (night blindness). In the acute stage all the above symptoms are aggravated and complicated by photophobia and spasm of the lids; the fourth stage is similar to the acute but general nervous symptoms develop. Bad illumination and poor ventilation have been blamed, but the causes are largely psychological, *i.e.*, hysterical symptoms superimposed on congenital or acquired instabilities of the ocular mechanisms.

§ 848. VI. **Changes in the Fundi.**—The reflecting **ophthalmoscope** is now rarely seen, having been replaced by the electric ophthalmoscope. To examine the eye, commence with a + 12 lens in the aperture and examine with its aid the cornea, aqueous and lens; by rotating the battery of lenses the power is gradually reduced, any opacities in the vitreous can be noted and when the fundus comes into focus the number of the lens in the aperture should be observed. This gives a rough measure of the refraction of the eye. Estimation of visual acuity without a knowledge of the patient's refraction is bound to mislead.

The *optic disc* should be examined as to its shape, its borders, its colour, its vessels, and its level. Normally the disc is circular or slightly oval, with a clearly defined

border, especially at the outer edge. It appears oval in astigmatic eyes. The *colour* of the disc is a rosy vermilion, but paler than the rest of the fundus. The vessels curve from the centre, and then lie flat. Arteries and veins go together, but the arteries are narrower (two-thirds) than the veins, a trifle paler, and have a broader, more continuous light stripe running along the centre. Normally the arteries do not pulsate, but the veins may do so. Pulsation in the arteries may indicate (i.) increased intraocular tension or (ii.) aortic regurgitation. The *level* of the disc is important, but a little difficult to gauge. If when using the ophthalmoscope, the surrounding *retina* can be seen clearly without the aid of any lens placed in the mirror hole, but the *disc* cannot be seen clearly without the aid of the lens, it must be at a different level. If a weak − lens is necessary to see the disc under these circumstances, then the disc must evidently be behind the retinal level (cupping). If, on the other hand, a weak + lens is necessary, then the disc is on a level anterior to the retina (swelling). One can gauge the amount of swelling or cupping in this way, for roughly each 3 D. = 1 mm. of swelling or cupping. Thus, supposing it is necessary to use 1 D. to focus the retinal vessels precisely, and 4 D. to focus the disc, then there must be 3 D. or 1 mm. swelling. This is an accurate method of measuring, provided the observer is able to *relax his own accommodation thoroughly.*

§ **849. Papillœdema** is œdema of the optic nerve at its entrance into the globe, and is evidenced, in a typically marked form, by blurring of the edges, swelling, and increased vascularity of the disc (Plate VI.4). The arteries become narrower, and the veins are enlarged and tortuous, the vessels curving over the œdematous edge. The vessels, moreover, may appear broken here and there, as they are hidden by the œdema. In the early stage the disc has simply a fluffy look, and then the upper and lower edges only are blurred. These œdematous changes may gradually subside, or go on to "consecutive" atrophy (Plate VII.6). It should be remembered that the *acuity of vision may be undisturbed,* even when there is considerable papillœdema, though the visual field is usually diminished in some degree. Disturbance of vision is generally more marked as the acute stage subsides. In papillœdema with preservation of vision the pupils react to light. *Bilateral papillœdema* is very strongly suggestive of intracranial disease, and especially (i.) intracranial tumour, in which it is present at some time in about 85 per cent. of the cases. It is especially common in cerebellar tumour. It is rare in cerebral hæmorrhage and embolism. (ii.) Increase of pressure from other causes—*e.g.*, subarachnoid hæmorrhage or hypertensive encephalopathy. It is uncommon in acute meningitis. Syphilis may produce papillœdema. *Unilateral papillœdema* may indicate disease in the orbit or behind the orbital fissure, *e.g.*, from orbital tumour, exophthalmic ophthalmoplegia, aneurysm of the internal carotid, infections.

§ **850. Optic Neuritis** or **Papillitis** may develop at any part of the optic nerve and is usually associated with loss of acuity of vision and central field defects. It is visible ophthalmoscopically only when the optic papilla is involved. Such cases are called intra-ocular neuritis, or because of changes in the papilla, papillitis. Distinguish from these the cases in which the inflammation is located in the optic nerve farther back (retrobulbar neuritis). Since in retrobulbar neuritis the focus of inflammation cannot be seen, its diagnosis is inferred from other symptoms. Various toxic conditions of the blood may cause Papillitis, chief among which is renal disease, giving rise to a special form (see § 853 and Plate VI.3). Papillitis occurs in intracranial inflammations, *e.g.*, syphilis, abscess. Plumbism, anæmia and disseminated sclerosis are occasional causes, as are toxic encephalomyelitis and the encephalomyelitis of acute specific fevers.

§ **851. Retrobulbar Neuritis** is an inflammation of the optic nerve behind the nerve head. The symptoms are: (1) *Sudden* loss of vision in one eye, with (2) aching in the orbit and tenderness of the eyeball, (3) central scotoma, and (4) defective vision for red and green. (5) The pupil tends to be dilated and reacts poorly to direct light, but reacts normally to consensual illumination; it reacts normally on

PLATE V.

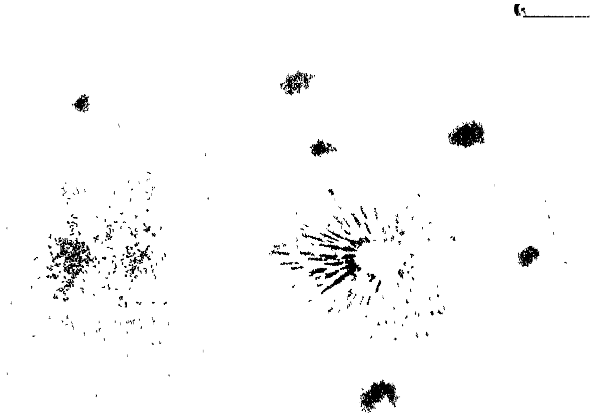


1. NORMAL.



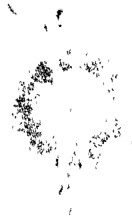
2. ARTERIO-SCLEROSIS.

Arteries tortuous and irregularly narrowed, with light reflex marked, and loss of transparency. Veins kinked and their course deviated where crossed by arteries.



3 CHRONIC NEPHRITIS.
Later stage.

Disc— Swollen and margin blurred. Macula -Radiating lines of white dots resembling the Star of India. Fundus --Flame-shaped hæmorrhages disposed radially to the disc, White woolly patches.



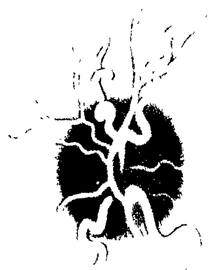
4. PAPILLŒDEMA.

Mushroom-like swellings of disc with blurring of margins due to lymphatic stasis engorgements of veins. Vessels bent by swelling and partly covered up. Hæmorrhages variable No loss of visual acuity in early stages.

PLATE VII



5. PRIMARY OPTIC ATROPHY.
Clear cut blue white disc with fine vessels



6. SECONDARY OPTIC ATROPHY.
White disc blurred by deposition of fibrous tissue which may extend along the vessels, giving them a fibrous sheath. Tortuosity of vessels near disc.

accommodation. The symptoms may be transient and last only a few weeks, although a permanent scotoma and atrophy may follow. The commonest *cause* is (1) disseminated sclerosis. The condition may rarely occur as a complication of (2) suppuration in the sphenoidal or ethmoidal air sinuses. Retrobulbar Neuritis, coming on gradually, may be due to (3) neuromyelitis optica, (4) tobacco or alcoholic poisoning (especially wood alcohol), (5) syphilis, (6) diabetes and nutritional disorders, (7) Leber's hereditary optic atrophy (§ 852).

§ **852. Optic Atrophy** (Plate VII).—*Primary* Atrophy is characterised by porcelain-like-pallor of the disc and sharply defined outline. The best example is tabetic atrophy, but atrophy in disseminated sclerosis and that following various forms of retrobulbar neuritis are included, though the atrophy is not strictly primary. Optic atrophy may be secondary to retinal disease such as retinitis pigmentosa or other forms of extensive choroido-retinal disease. In these cases the disc is a waxy yellow colour. When atrophy follows papillœdema from any cause, or papillitis, it is termed *secondary* or *consecutive*; the disc is white, opaque, the edges are blurred and the vessels sheathed in fibrous tissue to an extent varying with the severity of the primary lesion.

Leber's Hereditary Optic Atrophy is a hereditary type of optic atrophy affecting males and females but transmitted through the females alone. It commences as a retrobulbar neuritis, soon after puberty, with central scotoma and slight swelling of the disc. The atrophy and visual failure are rarely complete; often useful vision is retained. The malady is probably analogous to Cerebro-macular Degeneration (see § 763).

§ **853. Retinitis.** Most affections of the retina are described as retinitis, though few of them are true inflammations. Œdema of the retina, whatever the cause, involves loss of transparency as is seen *par excellence* in embolism of the central artery; it is also a marked feature of severe albuminuric retinitis. Other important features of retinal disease are hæmorrhages, exudates of various kinds, aggregations of pigment (superficial to the retinal vessels) and changes in the vessels themselves. Retinitis is symptomatic of disease, and nephritis is the most important cause. The retina is essentially a vascular structure and presents direct evidence of high blood pressure and arterio-sclerosis; this leads eventually to arterio-sclerotic retinitis. It also shows numerous manifestations of general arterio-vascular disease and its complications, as for example, thrombosis of a retinal vein. Retinitis occurs also in syphilis, diabetes, leukæmia and in septic states of the teeth, tonsils and other parts. The picture depends on the stage at which the examination takes place, on the age of the patient and on the condition of the blood vessels at the time of onset of the retinal disease. Retinitis causes no pain but a patient may complain of entoptic flashes or intolerance of light.

Detachment of Retina.—In practically all cases a hole is found in the retina. The method of treatment involves the production of an adhesive choroiditis around the hole by means of coagulation or perforating diathermy, electrolysis or a combination of these coupled with evacuation of the subretinal fluid. Detachment complicating inflammatory disease is not suitable for such treatment: solid detachment due to an underlying new growth may be accompanied by a fluid detachment.

Albuminuric Retinitis (Plate VI) is really a neuro-retinitis, consisting of three elements. (i.) Papillitis (see above); (ii.) hæmorrhages into the retina, usually flame-shaped and most plentiful towards the disc; and (iii.) fine white spots near the macula, and large woolly patches on the retina. One or other of these is sometimes wanting, but in its typical form this kind of retinitis is sufficiently distinctive to diagnose renal disease without examining the urine. It may occur in any form of renal affection, but is frequently associated with chronic nephritis. Albuminuric retinitis is of very grave significance, the patient seldom surviving more than six months after the diagnosis is made; albuminuric retinitis of pregnancy is an exception to this, complete recovery often following parturition, normal or induced. **Hæmorrhages** into the retina and choroid are met with as dark red patches. They accompany any severe retinitis papillitis or papillœdema, particularly albuminuric retinitis, diabetes

and hypertension with arterial degeneration: they are also met with in pernicious anæmia, leukæmia, pyæmia, malaria, scurvy, and other purpuric and toxic conditions. **Embolism of the Central Artery of the Retina** occurs most frequently in the course of cardiac disease, with vascular sclerosis, especially in disease of the aortic valves, in auricular fibrillation and ulcerative endocarditis and in pregnancy where the embolus may be a fragment of a chorionic villus. An embolus may be arrested in a branch of the central artery or in the main trunk, causing immediate loss of part or the whole of a visual field in one eye. On examination, the retinal vessels are found empty, the retina opaque, and a peculiar round, cherry-red spot is seen in the macular region. The disc is pale. Thrombosis of the central vein similarly causes sudden but incomplete blindness. **Retinitis Pigmentosa** is characterised by narrowing of the lumen of the retinal and choroidal vessels, secondary atrophy of the optic nerve with a waxy looking disc and the deposition of aggregations of pigment, particularly in the periphery, which resemble bone-corpuscles. Night-blindness is the chief symptom; the disease begins in childhood.

§ **854. Choroiditis Disseminata** (usually bilateral, though sometimes limited to one eye) is frequently an evidence of acquired syphilis (in which case it arises three months to three years after infection): or of congenital syphilis. On examination, discrete, white, atrophic patches, with irregular black edges, are found scattered over the fundus, *most marked at the periphery.* This condition has to be differentiated from the slow type of choroido-retinitis. **Acute tuberculous lesions** may be found as yellowish ill defined spots in the choroid in tuberculous meningitis. **Atrophic Patches in the Choroid,** with retinal involvement, occur in the secondary changes of progressive myopia: the macular region is apt to suffer.

§ **855. Glaucoma** is a term applied to many conditions of the eyeball having one common feature, increased tension. There are three main varieties: (1) primary, (2) secondary, (3) infantile. (1) For *primary glaucoma* there are many theories but none generally accepted: whatever the cause, secretion of aqueous into the eye exceeds excretion from it, there is a tendency for dilatation of the pupil and, in the later stages, the filtration angle is more or less closed by adhesion of the peripheral part of the iris to it. The old operation of iridectomy is replaced by the modern operation of trephining the corneo-scleral junction with iridectomy: some prefer iridenclesis, iridodialysis or a flap sclerotomy. The alternative to operation, which should always be given a trial in mild cases, is the instillation of pilocarpine or eserine drops, the latter being stronger, in the lowest concentration compatible with the attainment of normal tension and stationary fields of vision.

Chronic glaucoma is characterised by reduction of the field, particularly a nasal "bite" and enlargement of the blind spot with cupping, increasing pallor and, finally, atrophy of the disc, the atrophy being an ascending one from the moribund ganglion cells. Emotional disturbances may play a part in the etiology.

Acute primary glaucoma may come on suddenly or complicate a chronic case: extreme pain, rapid loss of sight and vomiting are found, and the eye may be lost in twenty-four hours. Owing to the abdominal symptoms, such cases have been operated on as appendicitis. Treatment is extremely urgent—eserine repeatedly instilled, fomentations, leeches and purgation. Operation—a large iridectomy—should be performed as soon as possible.

(2) *Secondary glaucoma* sometimes complicates iritis and cyclitis, and is due either to complete posterior ring synechiæ damming up the fluid in the posterior chamber with iris bombé and an idle excreting mechanism, or to the increased protein content and viscosity of the aqueous fluid interfering with the proper functioning of the filtration apparatus in the Spaces of Fontana and Canal of Schlemm. Operation in these cases, where necessary, is iridectomy or multiple perforations of the bombé iris with a Grafe knife. Secondary glaucoma may follow herpes ophthalmicus and thrombosis of the central veins.

(3) *Infantile glaucoma* or *buphthalmus*, fortunately rare, is due to faulty development of the angle of the anterior chamber with poor excretion and increased tension:

the globe enlarges as the pressure increases. Trephining offers the only hope, but the prognosis is bad.

§ 856. **The Trigeminal Nerve.**—The *motor* and *sensory* nuclei of this, the largest of the cranial nerves, have been described in § 683. The motor and sensory roots leave the ventro-lateral surface of the pons in a sheath of dura mater called the cavum Meckelii. In this cave, on the tip of the petrous portion of the temporal bone, lies the trilobed Gasserian ganglion. The ganglion divides into three nerves—(1) The Ophthalmic division, which enters the orbit; (2) The Maxillary division, which leaves the skull by the foramen rotundum; and (3) The Mandibular division, which is joined by the motor root and leaves the skull by the foramen ovale. The first two divisions are purely sensory, the third is a mixed nerve.

(1) *The Ophthalmic division* supplies the eyeball and conjunctiva (corneal reflex), the skin of the forehead and scalp up to the centre of the vertex, a median cutaneous strip on the nose, the meninges and the mucous membrane of the upper nasal cavity and lachrymal glands (Fig. 186).

(2) *The Maxillary division* supplies the skin of the face on the lateral aspect of the nose, on the cheek, from the upper lip to the lower eyelid inclusive and, laterally, as far as the pinna, the upper jaw and its teeth, pharynx and tonsil, and the lower part of the nasal cavity.

(3) *The Mandibular division* supplies the skin of the posterior aspect of the temple, the upper part of the pinna and side of the face (not the angle of the jaw, C1), the tongue, lower cheek and gums and the Eustachian tube. The motor fibres supply the masseter, temporal, pterygoids, mylohyoid, anterior belly of digastric, tensor palati and tensor tympani muscles.

The methods of examination are described in § 703.

Symptoms.—Lesions of the fifth nerve produce sensory loss, diminution or loss of corneal and conjunctival reflexes on the side of the lesion, and paræsthesiæ over the anatomical distribution of the nerve and, if the motor root is involved, paralysis of the muscles of mastication. Slowly progressive lesions are painless and produce, as their earliest sign, diminution of the corneal reflex. Wasting of the masseter and hollowing of the temporal fossa often precede loss of power in the muscles affected.

Etiology.—In its intracranial course the nerve may be involved by (1) Extra-cerebellar tumours, (2) Secondary growths at the base of the skull, (3) Meningitis at the tip of the petrous bone in chronic suppurative middle-ear disease, or (4) Basal Syphilitic Meningitis. In Tabes Dorsalis a " butterfly " zone of analgesia is common over the nose and cheeks (masque tabétique), while in Syringomyelia, involvement of the spinal root leads to a zone of analgesia on the periphery of the face.

Herpes Ophthalmicus is an inflammation of the Gasserian ganglion, due to the virus of herpes zoster (§ 826). It is a disease of maturity, but may occur in young adults. Vesiculation and pain occur over the distribution of the ophthalmic division only. Keratitis and córneal ulceration may be present, and vesicles occur also in the upper nasal cavity. The vesiculation in the forehead may be followed by permanent scarring and often by intractable supra-orbital neuralgia and sensory impairment. The trophic corneal lesions may cause blindness. External ocular palsies, pupillary abnormalities, even optic atrophy, may accompany the condition and there may be associated iritis.

§ 857. **Progressive Facial Hemiatrophy.**—This rare condition is characterised by atrophy of the skin and its appendages, subcutaneous tissues, muscles, bones and cartilage, within the territory of the trigeminal nerve. The eye may be involved.

The malady comes on in early life and is commoner in females. There is no sensory loss or paralysis.

TRIGEMINAL NEURALGIA (see § 822).

§ 858. **The Facial Nerve.**—Of all the nerves of the body the facial is the most frequently affected by paralysis. It is peculiar in having a long tortuous course through a bony canal, the aqueductus Fallopii. During the onset of slow paralysis, or during slow recovery, clonic or fibrillary twitches may be observed in the paralysed muscles: these are seen in the case of no other nerve in the body.

Motor and *sensory* roots are described. The *motor root* arises from a nucleus in the lower part of the pons and the fibres bend round the sixth nucleus before emerging from the brain-stem at the junction of pons and medulla. The nerve passes forwards

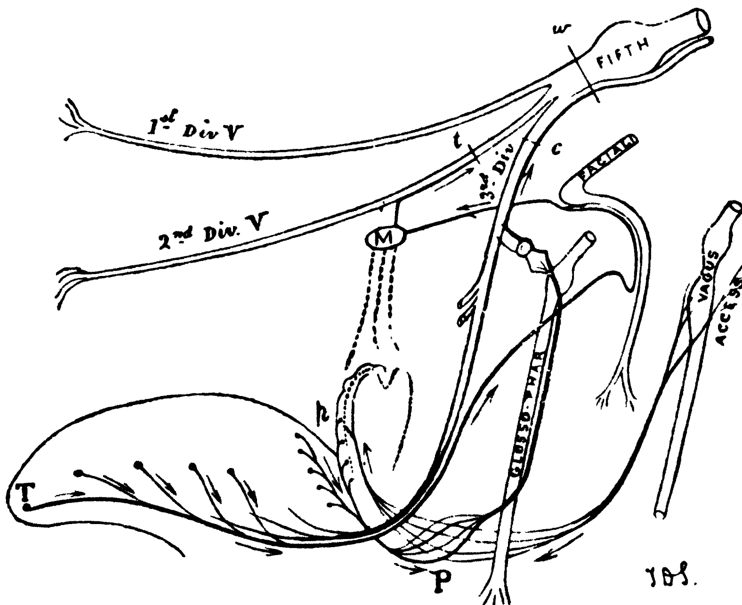


FIG. 193.—SENSE OF TASTE and NERVE-SUPPLY OF PALATE.—Diagram to show how taste impressions reach the fifth nerve ⟶, and the motor-supply of the palate ←-- . T, branches of the chorda tympani conveying taste from the tip and sides of the tongue, and running with the lingual branch of the fifth, then through the facial to Meckel's ganglion (M), and thence to the second division of the fifth. *p*, soft palate from which, and from the dorsum of the tongue, taste fibres pass through the pharyngeal plexus (P) to join the glosso-pharyngeal, and thence through the otic ganglion (*o*) to the third division of the fifth. The palate, it will be seen, is supplied by the accessory portion of the spinal accessory through the vagus.

and outwards with the auditory nerve to enter the internal auditory meatus, where it is joined by the sensory root (n. intermedius). In the aqueduct of Fallopius, the nerve pursues a curved course. To it is attached the geniculate ganglion of the n. intermedius. The nerve within the petrous temporal bone gives off a nerve to the stapedius muscle and runs down behind the tympanum. A quarter of an inch above its emergence from the stylo-mastoid foramen, it gives off the chorda tympani to join the lingual. On emerging from the stylo-mastoid foramen the nerve passes forwards in the substance of the parotid gland to supply all the facial muscles of expression and the platysma. The *sensory root* (n. intermedius of Wrisberg) is really a separate nerve with its own ganglion (geniculate ganglion) functionally distinct from the facial. From the geniculate ganglion, fibres run centrally to a nucleus

in the pons (n. gustatorius) and peripherally with the facial, then into the chorda tympani with which they are distributed to the anterior two-thirds of the tongue, supplying this with taste fibres (Fig. 193).

The METHODS OF EXAMINATION are described in § 703.

Symptoms.—Supranuclear affections of the facial nerve produce weakness of the lower face only (see § 683). These are described under Hemiplegia (§ 752). The facial nerve may be affected in four situations:

(1) *After its exit from the stylo-mastoid foramen*, lesions produce paralysis of voluntary and emotional movements of the facial muscles, with loss of tone and diminished reaction to faradic stimulation. This may occur from toxic or infective neuritis, injuries, or tumours of the parotid.

(2) *Within the aqueductus Fallopii* a lesion produces similar effects, but accompanied by loss of taste on the anterior two-thirds of the tongue on the affected side, from involvement of the taste fibres in the geniculate ganglion. This may follow toxic or infective neuritis, injury, meningitis from caries of the petrous bone in suppurative otitis media, or pressure from bony or cholesteatomatous growths in the middle ear. Such palsies are often slow in onset and accompanied by facial hemispasm.

(3) *Between the pons and the internal auditory meatus* lesions cause purely motor symptoms, and from the proximity of the auditory nerve tinnitus or deafness. The common lesion is an extra-cerebellar acoustic neurofibroma with occipital pain (§ 814).

(4) *Within the pons* lesions again cause purely motor symptoms. There is usually an associated diplopia and ipsilateral external rectus palsy, from paralysis of the neighbouring sixth nucleus, and extensor plantar responses may be present from pyramidal involvement.

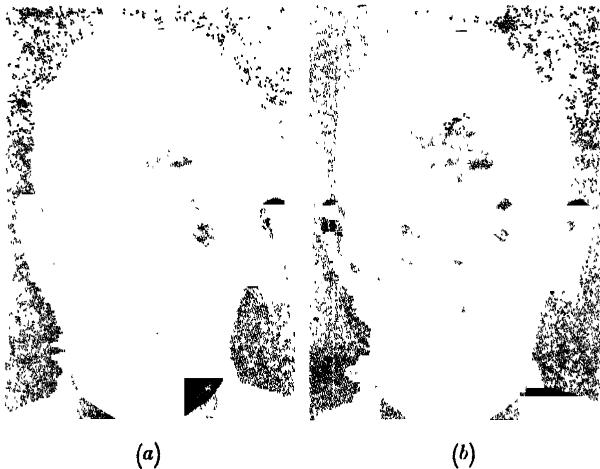
§ **859. Bell's Palsy.**—The onset is rapid and there is commonly a history of exposure to cold. Pain and muscular tenderness below and behind the ear, and even swelling in the neighbourhood of the parotid, may precede the palsy and persist for three or four days. The patient notices stiffness of the affected side of his face, he cannot close his eye properly, and tears trickle from the flaccid lower lid. Articulation is indistinct at first and fluids are spilled in drinking. In severe cases, there is complete immobility of the upper and lower face on the affected side and no voluntary or emotional movement is possible. The muscles are toneless, and, especially in old people, epiphora occurs from paralysis of the tensor tarsi. In severe cases, there is nearly always loss of taste on the lateral edge of the anterior two-thirds of the tongue. When the patient attempts to close his eyes, the globe on the affected side turns upwards and outwards more than on the normal side (Negro's sign).

Secondary Contracture occurs in the paralysed muscles, after some months, in severe cases. The unsightly distortion thus produced leads to narrowing of the ocular fissure and exaggeration of the naso-labial fold, and drawing up of the angle of the mouth, so that the healthy side appears the weaker. In other cases *distortion of facial expression* occurs from misdirection of the regenerating fibres so that corresponding nerve bundles

do not supply the same muscles on the two sides of the face. Both defects are permanent.

Diagnosis.—Peripheral facial paralysis may occur in poliomyelitis, diphtheria, tetanus and encephalitis lethargica. When the paralysis is due to petrous temporal disease, deafness, otorrhœa and perforation of the tympanum will co-exist. Lesions within the skull (acoustic neurofibroma) are accompanied by deafness, cerebellar ataxia and diminished corneal reflex from trigeminal involvement. Lesions in the pons cause associated diplopia, hemiplegia, hemianæsthesia or hemiataxia.

Prognosis.—Most cases clear up in a few weeks or a few months. The *electrical reactions* of the paralysed muscles should not be tested until a fortnight has elapsed from the onset of the paralysis: by this time all



(a) (b)

FIG. 194.—CASE OF LEFT-SIDED FACIAL PALSY.—(a) At rest. Observe the facial asymmetry with widened ocular fissure and drooping of the angle of the mouth on the left side. (b) On attempting to shut the eyes the patient merely rolls the eyeball upwards and outwards on the affected side so that the cornea passes under cover of the upper lid.

degenerating fibres will be destroyed and the results will give valuable information. If at the end of four weeks the muscles do not react to faradism, recovery is likely to be incomplete and long delayed (§ 709). In almost all cases, however, recovery occurs, and this is true also in traumatic cases and obstetric cases resulting from the pressure of forceps during delivery. Where taste is intact, recovery may be expected within three months. Profound paralysis and flaccidity of muscles at the onset is of bad prognostic origin. If after three to six months there is little recovery, secondary contracture or distortion of facial expression are likely to occur in varying degree.

Etiology.—Bell's palsy is believed to be due to an infection allied to that of herpes zoster. Other cases of facial palsy are due to otitic and other infections or to new growths.

Treatment.—The patient should be given a peroxide mouth-wash to

use after meals to get rid of food particles which collect between the cheek and the gum. When the lids cannot be closed, in order to prevent exposure conjunctivitis, the eye is bathed night and morning with boric lotion. The patient should be instructed to wipe his eye from below upwards, so as to lessen the danger of epiphora. Voluntary re-educative exercises should be practised for ten minutes by the clock, night and morning, in front of a mirror. The patient attempts to screw up his eyes, wrinkle his forehead, show his teeth, blow out his cheeks, whistle, etc. A piece of copper wire, covered with rubber tubing, can be bent over one ear to hook up the angle of the mouth on the flaccid drooping side. This should be worn at night. Facial massage is of use, radiant heat in skilled hands is of value. Iodides or salicylates may be given internally. Cases due to mastoid or middle ear disease rarely show any recovery and, after a year, an attempt may be made to anastomose the peripheral cut end of the facial to the proximal cut end of the hypoglossal or spinal accessory nerve. Duel and Ballance suggest decompression of the nerve in the aqueduct. In intractable cases the deformity may be considerably lessened by a plastic operation in which an attempt is made to support the paralysed muscles by fascial slings or strips inserted subcutaneously.

Bilateral Facial Palsy.—It may occur in Bell's palsy, with basal gummata or secondary deposits, in diphtheritic and alcoholic polyneuritis and in *uveoparotitic paralysis* (see § 9). Bilateral facial weakness may occur in Myasthenia gravis and in the Myopathies.

FACIAL HEMISPASM (see § 774).

FACIAL TICS (see § 772).

Geniculate Herpes.—Herpes Zoster (§ 826) may attack the geniculate ganglion, producing vesiculation in the external auditory meatus, hard palate or tonsillar fossa, with severe pain in the ear, and associated facial paralysis. Such cases may be followed by tinnitus or vertigo, which is transient, like the facial paralysis.

The **Auditory** and **Vestibular Nerves.**

§ **860. The Eighth Nerve** consists of two separate nerves which are distinct peripherally and have separate central connections. (*a*) The *Auditory* (*Cochlear*) *Nerve*, concerned with hearing, and (*b*) the *Vestibular Nerve*, concerned with equilibrium.

(*a*) *The Auditory* (*Cochlear*) *Nerve* is described in § 675. It is concerned solely with hearing. Lesions of the auditory nerve produce deafness and tinnitus.

(*b*) *The Vestibular Nerve* originates in the ganglion of Scarpa, which lies at the outer end of the internal auditory canal. Peripherally this ganglion sends filaments to (1) the semicircular canals, and (2) the otolith organs (utricle and saccule). The semicircular canals are stimulated by movements, the otolith organs by alteration in position of the head. The central connections are described in § 680. This nerve is concerned with the mechanism of equilibrium. Lesions of the vestibular nerve or semicircular canals produce Vertigo (see § 692).

CLINICAL INVESTIGATION.—*Hearing* can be tested by the voice, the watch, and the tuning-fork—the two former by air-conduction, the last by bone-conduction and air-conduction; with the audiometer more exact testing and the recording of hearing is now possible. Wax in the external meatus must be first removed, and if necessary the ear should be syringed with warm water, after the wax has been softened by warm oil, or bicarbonate of soda solution (2 teaspoonfuls to the pint). To test

the *acuteness* of *hearing* by *air conduction*, stand behind your patient, close one of his ears with one of your hands, and place a watch in the other hand outside the range of his hearing, then approximate it slowly, asking the patient to say directly he hears the tick, and then estimate the distance. Examine the other ear in the same way. Ascertain on yourself what is the normal distance at which that particular watch should be heard, and supposing this is 60 inches, and the patient hears with the left ear at a distance of 5 inches, and with the right at 60 inches, then the acuteness of his hearing in the left ear is represented by the fraction $\frac{5}{60}$. The speaking and the

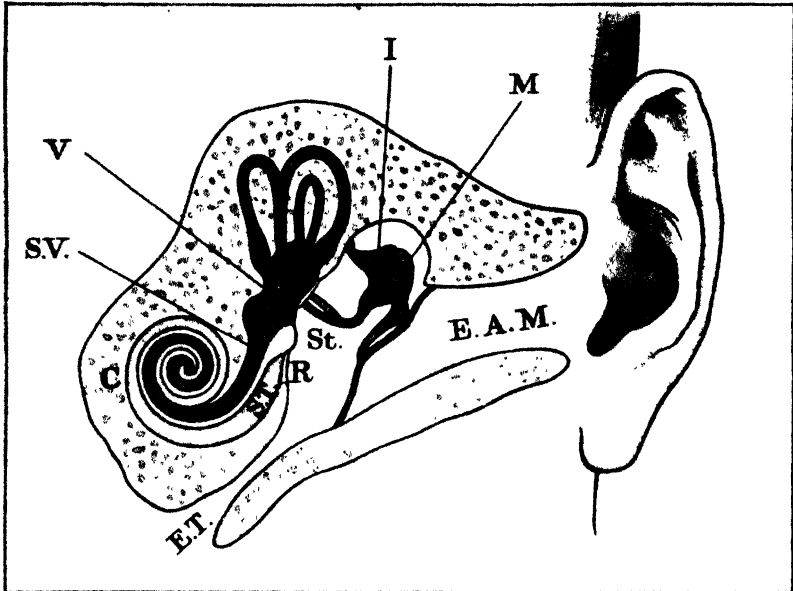


FIG. 195.—AUDITORY APPARATUS (diagrammatic representation) of the left side seen from the front, the internal parts being magnified two-fold.

E.A.M., External auditory meatus, separated by tympanic membrane from tympanum in which R. is situated.
M., on head of Malleus.
I., Incus fixed to wall by its short process, and articulating with the stapes by its long process.
St., Stapes, the foot of which fits into the fenestra ovalis.
V., Vestibule consisting of saccule (below) and utricle (above). Into the latter open the three semicircular canals, superior, posterior, and external (or horizontal). The vestibule leads on to the scala vestibuli (S.V.) of the cochlea (C.).
R., Fenestra rotunda leading from the scala tympani (S.T.) to tympanum.
E.T., Eustachian tube.

whispering voice are very useful for testing hearing. Each ear is tested separately, the other for the time being closed by the patient's finger. Various words are used; the observer gradually approaches until the patient can repeat them without mistakes, and the distance is noted. To test by *bone-conduction* (*Schwabach's test*), a tuning-fork in vibration is placed with its stem on the patient's mastoid process, and when he ceases to hear it, it is applied to the observer's mastoid. If the observer still hears the sound, there is said to be diminution of bone-conduction. On the other hand conduction may be lengthened. A vibrating tuning-fork is held on the observer's mastoid till it ceases to be heard; it is then transferred to the patient. If the patient still hears the sound, bone-conduction is longer than normal (assuming that the

observer's hearing is normal). A watch applied to the mastoid may be used in the same way to give an idea of bone-conduction.

Weber's Test.—To ascertain whether an impairment of hearing is due to *nerve deafness* or to *obstructive deafness*, test the per-osseus hearing by placing a watch or a vibrating tuning-fork on the centre of the patient's forehead. If the deafness is due to disease of the auditory nerve it will be heard not in the affected ear, but in the good ear. This indicates nerve deafness. If the deafness is due to obstructive ear disease the sound will be heard *on the defective side.*

Rinne's Test.—In a normal person a tuning-fork (256 double vibrations per second) placed on the mastoid bone until no longer heard in that situation, can still be heard by him if removed and held opposite the meatus (Rinne's test positive), hearing by air conduction being more prolonged than by bone-conduction ; it indicates an absence of any considerable middle-ear disease. When the middle ear or conducting apparatus is definitely diseased the tuning-fork cannot be heard opposite the meatus after it has ceased to be heard when held on the mastoid (Rinne's test negative). If Rinne's test is positive in a deaf ear there is probably nerve deafness present.

Galton's Whistle and the *monochord* are used for testing the upward limit of audition of a patient. Diminution of audition for highly-pitched notes occurs in old age and in nerve deafness. The introduction of the *audiometer* enables hearing to be accurately tested and recorded. *Paracusis Willisii,* "hearing best in a noise," is a characteristic of bilateral middle-ear disease, especially oto-sclerosis, and is usually associated with fixation of the stapes. Such patients can hear conversation better in a train or omnibus than otherwise. In boiler-makers' and some other forms of nerve deafness the converse is true.

(*b*) *Vestibular Nerve.*—The condition of the vestibular system can be ascertained by endeavouring to produce nystagmus by syringing with cold water and hot water, or by rotating in a rotating chair. Normally, syringing with cold water (30° C.) induces a nystagmus to the opposite side in about forty seconds; while hot water (42° C.) produces an opposite effect. Cold air may be blown in by Dundas Grant's apparatus. Rotation (ten times in twenty seconds) produces a nystagmus towards the side from which the patient is rotated if the semicircular canals and vestibular nerve are healthy.

Inspection of the Ear.—Note should be made of any discharge and its character (see below), any pain or tenderness over the mastoid (see below), any eczema of the meatus, etc. To examine the *meatus* (which should be done first without a speculum) the auricle should be pulled gently upwards and backwards, the tragus being pulled forwards by making traction with the thumb on the skin in front of it. If a speculum is to be used, the auricle should be held between the middle and ring fingers of the left hand (for the patient's right ear), the speculum being inserted with the right hand inwards and slightly downwards and forwards. The speculum can then be held between the thumb and forefinger of the left hand. It facilitates examination to have a mirror on one's forehead as in laryngoscopy (§ 164) to reflect the light from a lamp at the side of the patient's head. Cerumen is of dark colour and soft consistence. The membrana tympani may present indrawing (due to blocking of the Eustachian tube), congestion, thickening, or loss of lustre, atrophic areas, or perforations.

The Nose, Throat and Naso-Pharynx should be next examined. Note (1) the activity of the palatal muscles, (2) the Eustachian tubes and back of the nose by posterior rhinoscopy, and (3) the patency of each nostril, (4) the presence of pus or any other abnormality in the nose, (5) the condition of the tonsils.

The Patency of the Eustachian Tube is usually tested by inflation of the middle ear by *Politzer's* method. The nozzle of the rubber bag is inserted into one nostril, and both nostrils are then held close between the thumb and finger of the operator. The patient is then directed to swallow or to say "hic," and at the same moment the air from the rubber bag is forced into the nose. Deglutition raises the palate and opens the Eustachian tube, and the air, having no other outlet, is forced into it. A

tube connecting the ear of the patient with that of the operator will enable the latter to hear an audible " pick " if the middle ear is inflated, and this will reveal the patency of the Eustachian tube. A second point to note is the effect which inflation has upon the symptoms—deafness and tinnitus. The hearing is temporarily improved in middle-ear or Eustachian disease, and is unaltered in otosclerosis and nerve deafness. In *Valsalva's* method of inflation the patient pinches his nostrils firmly, and makes an expiration as if to blow his nose, but without allowing the air to issue. The *Eustachian catheter* is also used to inflate the ear for diagnosis or treatment. It is not a difficult operation, but requires a little practice. Pass it tip downward very gently along the floor of the nose to the edge of the hard palate, the patient being directed to breathe through the nose so that the soft palate may droop. Immediately the tip of the catheter has reached the edge of the hard palate turn it upwards and outwards, and it will enter the Eustachian orifice. It may be aided by the patient swallowing at the same time. The nozzle of Politzer's bag may now be carefully introduced into the catheter, and inflation performed as before.

§ 861. **Causes of Deafness.**—Two kinds of deafness are recognisable: **nerve deafness,** due to lesions of the auditory nerve or internal ear, and **obstructive deafness,** due to some disease in the middle ear or auditory passages.

DIAGNOSIS OF NERVE DEAFNESS AND OBSTRUCTIVE DEAFNESS.

Nerve Deafness.	*Obstructive Deafness.*
Diminution of air and bone-conduction. Positive Rinne; Weber to good ear.	Loss of air-conduction only, with negative Rinne, and Weber to deaf ear.
Loss of hearing for very high-pitched tones (except in hysterical deafness).	Better hearing for high than for low tones.
Decreased hearing in midst of noise.	Better hearing in midst of noise.
Hearing for conversation relatively better than for watch.	Hearing for conversation relatively worse than for watch.

I. **Nerve Deafness** may be of (*a*) Sudden or (*b*) Gradual onset.

(*a*) Nerve Deafness of *sudden onset* may be due to:

I. Ménière's Disease.
II. Acute Labyrinthitis.
III. Syphilis.
IV. Concussion of the Labyrinth.
V. Hysterical Deafness.
VI. Lesions of the Central Nervous System.
VII. Small Hæmorrhages or Thromboses in the Labyrinth.

I. MÉNIÈRE'S DISEASE (see § 692).

II. ACUTE LABYRINTHITIS (see § 692) may result from acute or chronic otitis media and is usually suppurative. In mumps, influenza and meningococcal meningitis as a rule no suppuration occurs. Although the above are the commonest causes, the labyrinth may be destroyed and nerve deafness result from any acute infection.

III. SYPHILIS, ACQUIRED or CONGENITAL.—A syphilitic neuritis of the cochlear nerve, or syphilitic neuro-labyrinthitis may occur within a few weeks after infection. This form may be bilateral (Table XXXIX) and is more sudden in its onset than nerve deafness due to syphilitic lepto- or pachymeningitis, which may be unilateral or bilateral.

IV. CONCUSSION OF THE LABYRINTH may result from head injuries, explosions and loud noises, forcible syringing of the ear or fracture of the petrous temporal bone. In fractures of the skull the auditory nerve may be torn or bruised. The facial nerve is often paralysed at the same time.

V. HYSTERICAL DEAFNESS may be sudden in onset, following an emotional shock. (i.) Blinking of the eyes occurs if sudden loud noises are made near the patient, and (ii.) even if he appears stone-deaf it may be possible to waken him from sleep by simply calling his name. (iii.) Galvanic currents to the mastoid produce vertigo.

VI. Lesions of the CENTRAL NERVOUS SYSTEM may (rarely) cause deafness.

VII. Sudden complete nerve deafness sometimes occurs presumably from some VASCULAR CHANGE (thrombosis or hæmorrhage). In leukæmia hæmorrhage or *leukæmic deposits* may be responsible (§ 543).

(*b*) Nerve deafness of *gradual onset* may be due to:

I. Extra-Cerebellar tumour.
II. Syphilitic Pachymeningitis.
III. Congenital Syphilis.
IV. Meningitis.
V. Occupational Deafness.
VI. Drugs or tobacco.
VII. Toxic Causes.
VIII. Senile Changes.

I. An ACOUSTIC NEURINOMA (Acoustic Neurofibroma, § 829) is an important cause of chronic deafness in middle life. Other symptoms gradually appear: giddiness, tinnitus, headache, vomiting, etc., and the corneal reflex on the same side as the deafness may be found to be diminished, or absent, or there is slight facial weakness.

II. SYPHILITIC PACHYMENINGITIS in the lateral region of the posterior crania fossa causes gradual nerve deafness.

III. CONGENITAL SYPHILIS is an important cause of chronic nerve deafness (§ 552).

IV. MENINGITIS.—Local meningeal changes in the lateral recess may cause gradual nerve deafness and occur (1) in connection with suppurative middle ear disease (circumscribed serous meningitis), or (2) after cerebro-spinal meningitis (see § 503).

V. OCCUPATIONAL DEAFNESS may occur from repeated loud noises of machinery or guns.

VI. POISONING with Quinine, Salicylates, Paraphenylenediamine or Tobacco, may cause transient nerve deafness.

VII. TOXIC NERVE DEAFNESS may arise from circulating bacterial toxins, (i.) dental sepsis; (ii.) intestinal sepsis; (iii.) nasal, sinus, tonsillar or other infection.

VIII. In OLD AGE a certain amount of nerve deafness is very common.

II. **Obstructive Deafness** or deafness due to disease of the middle ear or auditory passages may be (*a*) acute or (*b*) chronic.

(*a*) ACUTE OBSTRUCTIVE DEAFNESS may be due to:

I. Impaction of wax.
II. Impaction of a foreign body in the meatus.
III. Acute Eustachian catarrh.
IV. Acute otitis media with inflammation of the tympanum.

(*b*) CHRONIC OBSTRUCTIVE DEAFNESS OF SOME STANDING:

(*a*) *Without a History of Previous Discharge.*

1. If the deafness dates from an acute naso-pharyngeal catarrh, tinnitus is not constant, and on inspection, the tympanic membrane is indrawn, opaque, and thickened, and inflation by Politzer's bag gives some relief; the disease is due to *Eustachian obstruction* and the presence of adhesions in the middle ear.

2. If the deafness has an insidious onset, tinnitus is a prominent symptom, inflation gives no relief, and on inspection the tympanic membrane is practically normal, with a pink sheen in its posterior part; the disease is probably *otosclerosis*. Hearing by bone conduction is longer than normal and the Rinne test negative.

(*b*) *With a History of Previous Purulent Discharge.*—There are probably perforations or cicatrices resulting from suppurative inflammation of the middle ear, and inspection of the drum may confirm this.

Combined Obstructive and Nerve Deafness.—With this combination it is sometimes difficult to make out the exact state of matters. Such cases may be grouped into those with and those without discharge.

(*a*) If there is a *history or presence of discharge*, a suppurative otitis media spreading to the labyrinth is probably in operation. In these circumstances we get signs of nerve deafness gradually supervening on those of obstructive deafness.

(*b*) If there is *no discharge, past or present*, the most usual conditions are: 1. Disease

of the cochlea supervening on an old chronic catarrh or sclerosis of the middle ear. 2. If the history of nerve deafness precedes the obstructive deafness, the middle-ear catarrh has supervened on the nerve deafness. 3. In advanced cases of otosclerosis there is usually marked loss of hearing for high notes as well as for low notes—in fact, the deafness is mixed middle ear and nerve deafness.

Pain in the Ear may be due to : 1. Otalgia, when there is no sign of local disease or defective hearing, and a reflex cause such as a carious tooth or diseased tonsil is present. 2. Disease of the external meatus, such as furuncle or eczema. 3. Disease of the middle ear, when there is deafness, some pyrexia, and examination reveals congestion and bulging of the membrane.

PAIN IN THE MASTOID REGION may be due to : 1. Mastoiditis ; this is accompanied by redness, swelling and tenderness, deep throbbing and constitutional disturbance. It may follow acute or chronic suppuration. 2. A furuncle on the posterior wall of the meatus may give rise to a swelling behind the auricle. 3. A tender post-auricular gland may be found with local sepsis or pediculi capitis. 4. Mastoid neuralgia, which sometimes follows old mastoid disease.

Pain more or less GENERALISED OVER THE HEAD, accompanied by PYREXIA, may be associated with the following diseases of the ear :

(*a*) *Acute Diseases.*—1. Acute middle-ear suppuration, which is relieved by outlet of pus ; 2, acute meningitis. 3. If the temperature is continuously high, it may be due to retention of pus, extradural abscess, or meningitis. 4. If the temperature oscillates, there may be sinus thrombosis and pyæmia (§§ 738, 515). 5. If the temperature after an initial rise is normal or subnormal, and there are headache, slow pulse, and delayed cerebration, suspect abscess of the temporo-sphenoidal lobe or cerebellum (§ 737). 6. Labyrinthitis (nystagmus, giddiness, vomiting and pyrexia).

Discharge from the Ear.—A STICKY OOZING may be due to eczema of the meatus, boils or condylomata. A HÆMORRHAGIC discharge may be due to vascular granulations or erosion of blood-vessels occurring with middle-ear disease. In acute otitis media due to the hæmolytic streptococcus there are often hæmorrhagic blebs on the drum or meatal wall and the discharge at first may be blood-stained. An offensive SANIOUS discharge, with fungating granulations, acute radiating neuralgia, and enlargement of the neighbouring glands, is characteristic of malignant disease of the ear.

A PURULENT discharge (*a*) *which is or has been copious*, and associated with deafness from the beginning of the symptoms, is due to acute or chronic suppuration of the middle ear. When associated with chronic suppuration, its chronicity may be due to the presence of polypus, granulations, or cholesteatoma, caries of the malleus, incus, or temporal bone, disease of the mastoid, antrum or naso-pharynx, or to constitutional causes, such as diabetes mellitus, tubercle, or syphilis.

(*b*) A PURULENT discharge *which is not, and never has been, copious*, and deafness, which, if present, did not supervene till some time after the onset of symptoms, may be due to external disease of the ear, acute or chronic. On the other hand, with chronic mastoiditis, there is not necessarily a copious discharge and deafness may be slight.

The *Prognosis* and *Treatment* of these various symptoms depend mainly on the cause in operation. To deal with them individually would be beyond the scope of this work. Nerve deafness is not very amenable to treatment. Any toxic or other cause in operation should, if possible, be removed. Obstructive deafness is, in a large proportion of cases, due to Eustachian catarrh, and treatment should relieve the deafness. A certain amount of good may be done in chronic catarrh by regular inflations, which the patient can be taught to do himself, and regular inhalation of various remedies such as tr. benzoin co. The nose and throat must be searched for abnormalities or infections, and they must be cleared up. Acute middle-ear disease requires prompt measures. Hot fomentations and incision of the tympanic membrane may be necessary. Warm drops of glycerine with 5 to 10 per cent. carbolic acid should be used. The sulphonamides by mouth and penicillin injections are most useful. Mastoiditis and other intracranial symptoms demand surgical interference. It is found that if the discharge of acute otitis media does not stop in about four weeks

with adequate treatment (if adenoids, nasal sepsis, etc., have been attended to), the mastoid is usually infected. X-rays are useful in demonstrating this. Operation is therefore necessary in these cases. It is most important to prevent the case becoming chronic. In chronic suppurative otitis media treatment should be by drops such as spirit or argyrol 10 per cent. Zinc ionisation and insufflation of boracic acid powder containing iodine 1 per cent. may be used. In many cases, however, a conservative or radical mastoid operation will be necessary. In cases of deafness an aid to hearing may be prescribed; lip reading is of great help to the very deaf. This short outline will be found useful in many cases to indicate the direction in which further investigations should follow.

§ 862. **Tinnitus,** or subjective noises in the ear, is an extremely common symptom. The noise may be described as hissing, whistling, singing, buzzing "like a bell," roaring, and may be pulsating, continuous or intermittent. If the noise is referred by the patient definitely to the ear, the condition is true tinnitus and is due to disease of the (1) external auditory canal, (2) middle ear, (3) cochlear apparatus, or (4) cochlear nerve. If the noise is not definitely referred to the ear the condition is "head noises" and the cause probably psychical. Hallucinations of sound, the hearing of imaginary voices, is usually associated with mental illness.

The commonest *causes* of true tinnitus are: (1) Otosclerosis, (2) Nerve deafness, (3) Temporary Eustachian obstruction in coryza, (4) Anæmia (relieved by lying down), (5) Wax in the ear, (6) Drugs, *e.g.*, salicylates, quinine, (7) Prolonged loud noise affecting boiler-makers and those who work with electric drills.

Pulsating tinnitus (1) checked by compression of the carotid artery is due to arteriolar dilatation within the middle or external ear; (2) checked by compression of the vertebral arteries in the suboccipital triangle—a similar dilatation within the internal ear; (3) audible on auscultating the head—probably intracranial aneurysm.

Treatment.—The cause must be determined by a systematic examination of the patient generally and the auditory apparatus. "Head noises" are common in the silent watches of the night in neurasthenia and anxiety states, when the patient fears they will lead to insanity. True tinnitus is usually an intractable symptom, but it may be relieved by administering potassium iodide, hydrobromic acid or luminal.

Vertigo and its causes are dealt with in § 692.

§ 863. The **Glossopharyngeal-Vagus-Accessorius Nerves,** see § 683.

The methods of examination are described in § 703.

Lesions of the **glossopharyngeal nerve** produce (1) sensory loss over the upper part of the pharynx, (2) loss of taste on the posterior third of the tongue. Lesions of the **vagus nerve** produce (1) unilateral paralysis of the soft palate and pharynx, with a "curtain-movement" of the posterior pharyngeal wall to the sound side on swallowing, (2) laryngeal palsies. Lesions of the **spinal portion of the accessory nerve** produce sterno-mastoid and trapezius paralysis. The winging of the scapula produced is greater than that seen in serratus magnus paralysis, and the scapula lies further from the vertebral spines and is more rotated. This nerve may be affected by toxic neuritis or by injuries in its course in the posterior triangle. The three nerves are commonly affected together, as they arise together in the medulla and leave the skull together through the jugular foramen.

Medullary Lesions of the Glossopharyngeal-Vagus-Accessorius are due to focal thrombosis, syringobulbia, diphtheria, or chronic bulbar paralysis (motor neurone disease). The symptoms produced are unilateral paralysis of the palate, pharyngeal muscles and larynx (Avellis' Syndrome). In Syringobulbia and vascular lesions, crossed anæsthesia of the dissociated type is commonly present (Fig. 162).

Lesions at the Base of the Skull are due to secondary malignant deposits, fractures or syphilitic meningitis. Unilateral paralysis of pharynx, palate, larynx, sterno-mastoid and trapezius and hypoglossal (Hughlings Jackson's Syndrome) may result.

Lesions in the Neck result from injuries, tuberculous, lymphadenomatous, or malignant glands and aneurysms. Unilateral paralysis of the vocal cord and palate results.

The palate and pharynx escape if the lesion is below the point of origin of the pharyngeal branches. Transient palsies of the soft palate and laryngeal muscles occur in Myasthenia Gravis (§ 808). Paralysis and wasting of the sterno-mastoids is often marked in Myotonia Atrophica. Wasting of the trapezii and winging of the scapulæ may occur in Myopathy.

§ **864.** The **Hypoglossal Nerve** leaves the skull through the anterior condylar foramen and runs forwards above the hyoid bone and resting upon the hypoglossus muscle, to the under surface of the tongue. It is a purely motor nerve and supplies all the muscles of the tongue, intrinsic and extrinsic. The methods of examination are described in § 703.

The *symptoms* of a unilateral hypoglossal palsy are atrophic paralysis of one side of the tongue with loss of faradic excitability. The affected side of the organ shrinks and the tongue cannot be pushed into the contralateral cheek. There is little defect of articulation except at first, but the patient soon learns to overcome it.

Etiology.—Bilateral nuclear or supranuclear lesions occur in Chronic Bulbar Palsy (§§ 746, 747) with spasticity, or atrophy and fibrillation of the tongue. Spastic paralysis of the tongue also occurs in double hemiplegia (§ 746). Transient paralysis of the tongue occurs in Myasthenia Gravis. Unilateral hypoglossal palsies occur from motor neurone disease, medullary tumours, syringobulbia and gummatous meningitis. The nerve may be injured in the neck or under the tongue during operations upon the neck or throat.

CHAPTER XX

PSYCHOLOGICAL DISORDERS

THE forms of illness considered in this section are related essentially to the behaviour of the individual. They represent failure to make a satisfactory adaptation to life, environment and the other members of his social group. All students in general medicine have been impressed by the incidence of psychological factors in physical illnesses and how they influence their duration. Here we are concerned only with those conditions in which the psychological symptoms predominate. These can be studied in common with all other forms of illness, and failure to do so results, inevitably, in a lack of understanding of the factors involved. The major forms of mental illness have acquired for the student a sense of mystery, aggravated by the conflicting views and theories of the various schools of thought. The less severe forms include neurotic, emotional and personality disorders: individuals vary in their types of reaction to various forms of stress. Therefore many factors, constitutional, environmental, physical and psycho-biological may be involved; the significance of each can be evaluated only in relation to the other factors.

§ 875. **Psychopathology.** In order to understand the nature of psychological symptoms one must have some knowledge of *mental mechanisms*. The following account is a brief summary, involving the minimum of theory. Mental health depends upon the maintenance of a state of equilibrium between the conscious and the unconscious. Normally, these work in harmony and the individual adapts himself to his environment. The experience and stress of everyday life, with its struggles between desires and their gratification, within limits that allow a satisfactory adjustment of the personality, inevitably give rise to a state of mental *conflict*. Conflict is then a condition of normal mentation which is maintained by the formulation of a satisfactory solution. Difference of opinion has arisen regarding the nature of the conflicting forces. Freud originally considered they had a sexual basis, but it is obvious that this limitation is erroneous. Conflicts are accompanied by emotional tension and are the cause of much waste of mental energy. Most are solved successfully on the conscious level, are finally disposed of and give rise to no symptoms. Unfortunately, for various reasons, inherent or acquired, many patients are unable to deal with their conflicts in this manner, and devise various methods to maintain the state of equilibrium above-mentioned. These methods are common, invariably uniform in pattern, and are described as *mental mechanisms.*

Suppression and *repression* are the most common methods. Suppression is a conscious effort to forget what is unpleasant and is always accompanied by a focusing of the attention on something else. Repression is an attempt to expel from consciousness the factors that offend; they are transferred to the level of the unconscious. There they continue to exist and seek expression. Each repressed thought with its emotional component constitutes what we speak of as a *complex*. Invariably the personality elaborates methods on the conscious level to prevent expression of the repressed ideas; consequently exaggerated personality traits become prominent. Prudishness, for instance, results from the repression of normal sexual impulses; prejudices are developed by the individual to prevent the expression of repressed ideas. The establishment of such mechanisms need not disturb the mental equilibrium,

but certain types of interaction of these forces give rise to various nervous symptoms: persistent anxiety, fatiguability, hysterical or obsessional manifestations.

In all cases the energy remains latent and expression cannot be allowed an outlet unless in disguised form. This is achieved by *sublimation*. By this mechanism the energy is directed into some other activity combined invariably with an altruistic motive, such as religion, art and music. Another method of obtaining expression is by *rationalisation*. This is an unconscious mechanism and leads to the elaboration of various explanations in defence of beliefs or actions which are really the result of unrecognised motives. An attempt is made to justify such beliefs or actions by reasoning and great emotion may be displayed in their defence. The tendency to rationalise with regard to politics, religion, alcohol, tobacco are examples in everyday life. *Compensatory* mechanisms occur psychologically as well as physiologically: the aggressive, boisterous manner unconsciously adopted to compensate for excessive shyness is very familiar.

All mental life is influenced by *symbolisation*, often to such a degree that we fail to appreciate its significance. Music and art are symbolic representations of the feelings and ideals of the various artists. Similarly much of the play activity of children is symbolic. One method whereby repressed complexes achieve expression is when they are symbolised and submitted in disguised form. Many symptoms can be thus explained. Many dreams are symbolic, but the interpretation of such symbolisation is often exceedingly difficult.

Another form of defence mechanism is known as *projection*. Here the complex is regarded by the personality as no longer belonging to itself but as the product of some other real or fictitious person. By this method the activities of others are interpreted by the motives that determine the patient's own conduct. To explain their behaviour they project on others their own motives. This is the basis of many delusional ideas. The chronic alcoholic readily develops delusions regarding his wife and family and may attribute to them his own behaviour. Sometimes the desires are projected into imaginary persons. The aim of this, as of other mechanisms, is to make life more pleasant and avoid tension.

The method of repression may be replaced by that of *dissociation*. The mind becomes disintegrated, portions are separated off and function independently. On this basis are explained somnambulism and fugues. In the case of the psychotic, delusional ideas may be dissociated; this explains how patients with gross delusional ideas of their high status in life will perform most menial tasks.

The elaboration of excessive *phantasy* formation constitutes a common mental mechanism. Some degree of phantasy or imagination is common in normal life. Such elaborations in health remain subject to conscious criticism. It is also frequently seen in childhood with the elaboration of imaginary playmates. In certain forms of mental illness, particularly schizophrenia, it assumes an objective reality and thus repressed complexes attain an imaginary fulfilment.

The above-described mental mechanisms are substitutes for a logical adjustment, and may tend to produce deviation from reality; this is particularly evident in the pernicious mechanism of phantasy. The greater the degree to which they are developed the more profound are the symptoms, and the more serious the outcome.

§ 876. General Factors. Effects of constitution and environment are not readily separated; their influence varies according to the balance of the other factors and may vary at different periods in the life history. Similarly there is a constant inter-reaction between physical factors and the nervous mechanism. It is important to appreciate that mental ill-health is the sum total of a number of factors, and not the result of any one alone. The occurrence of mental illness in members of the same family has drawn attention to the influence of heredity. There is evidence that, as in certain forms of physical illness, there is probably inherited a diminished resistance to the development of certain types of mental illness. Of these the manic-depressive variety shows the greatest family incidence; the schizophrenic to a lesser degree. Mendelian researches suggest that the former is due to a dominant gene and schizophrenia to a recessive.

Psychotic illnesses are rare before adolescence, when physiological changes are marked; conflicts between instinctive desires and social standards then become much more active and demand fresh adaptations. At this age the incidence is high and remains so for some years. Similarly there is an exacerbation at the involutional period, when further endocrine changes occur, with marked emotional components. The association of mental illness and pregnancy led formerly to the classification and description of puerperal psychoses. It is now generally accepted that such a physiological event acts by reducing the general resistance, and that a mental illness developing at this time is determined by the previous personality and the other factors already discussed.

Apart from toxic-infective conditions, physical factors in themselves are rarely direct causes of mental illness. Exceptions are found with disorders resulting from trauma, which produces direct injury to the brain, and those due to vascular changes, generally of an arterio-sclerotic nature. Acute general infections may give rise to delirium followed, as in the case of influenza, by lassitude and depression. Such an illness may also release other symptoms, of a schizoid or cyclothymic nature, depending on the personality and make-up of the individual. Divergent views exist as to the rôle of focal sepsis and the frequency with which it is responsible for the development of mental symptoms. That such arises in certain cases, and must be dealt with, is agreed, but unless there is clinical evidence of toxæmia, improvement may not follow surgical treatment. The rôle of alcohol in the production of mental illness is limited: the acute alcoholic psychoses and Korsakoff's psychosis are the direct results of alcohol. A detailed investigation of the history and personality of the individual will generally show chronic indulgence in alcohol to be symptomatic rather than causative of a mental illness.

PART A. *SYMPTOMATOLOGY*

It is often alleged that symptoms of mental illness are much less clearly defined than those of bodily illness. Such symptoms represent the reactions of the individuals as an integrated whole, not of single mental processes. Consequently in their interpretation attention must be paid to all factors in order to appreciate fully the setting in which the symptoms have appeared. The fact that symptoms may have a symbolic meaning tends to confuse the student, but no matter how disguised, these symptoms are always germane to the mental state.

The more important symptoms may be considered as they involve predominantly behaviour, consciousness, emotion, thought-processes, sensation, memory and personality.

1. Behaviour . Overactivity, stereotypy, retardation, negativism, morbid impulses.
2. Consciousness . Confusion, dream states, delirium, disorientation, stupor.
3. Emotion . . Elation, euphoria, depression, anxiety, apathy, emotional instability.
4. Thought . . Delusions, obsessions, ideas of reference.
5. Perception . Hallucinations, illusions.
6. Memory . . Amnesia, hypermnesia, paramnesia.
7. Personality . Depersonalisation, transformation of personality, dissociation, fugues, multiple personality.

§ **877. Disorders of Behaviour.** *Overactivity* is characteristic of all forms of excitement, mania, schizophrenia and general paralysis. The activity is invariably purposeful, but not necessarily productive. In states of mania there is a concomitant talkativeness; distraction by external stimuli is profoundly exaggerated in the form of *flights of ideas*. Rhyming and punning are frequent accompaniments. In schizophrenic behaviour repetition of movement or speech may occur over a prolonged period and is known as *stereotypy*. Repetitive movements cause *mannerisms* to develop in the form of gestures and the embellishment of various ordinary movements. *Perseveration* should be distinguished from stereotypy: even against his own will there is an inability in the former to avoid repeating an action or a word which the individual has just used. Perseveration is a frequent symptom in organic brain disease. *Retardation* of psycho-motor activity is seen typically in melancholia. It may be accompanied by *negativism*—a refusal on the part of the patient to co-operate, even to the extent of doing the opposite of what he is told to do. Profound degrees of retardation may merge into a state of *stupor*: then *flexibilitas cerea* may develop. It is so called because of the wax-like position of the limbs which may be maintained in the most uncomfortable position for prolonged periods. *Morbid impulses* are the result of an irresistible urge to carry out some action. Failure to do so is accompanied by tension and restlessness; its performance is usually devoid of reflection or of any consideration for the interests of themselves or others. Among such impulsive forms of behaviour are pyromania—impulse to set on fire; kleptomania—impulse to steal; dipsomania—impulse to drink to excess; these are variations of obsessional behaviour.

§ **878. Disorders of Consciousness.** Consciousness implies ability to be aware of ourselves and our environment, involving the ability to synthesise and integrate as well as evaluate new experiences. It is dependent, in part, on attention. If the power of attention is impaired there develops *confusion*, perplexity and bewilderment. Such is found in the most acute stages of mental illness and in the toxic-infective group of psychoses. *Dream states* and *twilight states* are usually of psychogenic origin and last for varying periods of time. The disturbance of consciousness may be sufficient to cause the individual to lose knowledge of his surroundings, as in epilepsy and hysteria. In hysteria fugues or dissociated states may follow. *Delirium* may be psychogenic but is most frequently of toxic origin. All degrees of clouding of consciousness may develop, together with vivid hallucinations, visual and auditory. Invariably there is marked *disorientation*, indicating an inability of the individual to appreciate his position in respect of time, or place, or his relationship to other persons. Profound degrees of delirium are usually followed by a complete amnesia for the acute periods. In *stupor* there is an absence of all spontaneous activity or of any response to stimulation. It may develop in benign melancholia, the catatonic form of schizophrenia, certain toxic states, epilepsy and hysteria. In the psychogenic variety, rapid

emergence from stupor to activity may be seen, once the precipitating factors have ceased.

§ 879. **Disorders of Emotion.** Emotional disturbances are assessed by the intensity, duration and degree of harmony between the mood and the content of thought. *Exaltation* is an exaggerated degree of elation, typical of mania when it is accompanied by psychomotor overactivity. *Euphoria* implies, in addition, an abnormal feeling of wellbeing. Feelings of *depression*, characterised by hopelessness or despair, may be of all degrees: the less severe are apparent in the neuroses, whereas in melancholia the degree may be profound. When depression develops as the result of obvious external factors it is termed reactive. *Anxiety* implies more than fear. The latter ceases when the danger passes, whereas in anxiety the danger is usually described as being within. *Apathy* indicates a lack of either pleasure or sorrow. On the other hand the response may be apparent but may show a marked deviation from that normally seen, *e.g.*, news that would cause sorrow to the average person may be the occasion for elation and laughter. This inappropriateness of affect is typically found in schizophrenia. *Emotional instability* is characterised by rapid variations in the affective state without any apparent cause; it occurs in organic conditions.

§ 880. **Disorders of Thought.** A *delusion* is a false belief, quite impervious to argument or reason, and one that would not be shared by persons of the same race, education and status in life. Various psychological mechanisms have been elaborated in an effort to explain such ideas. In certain cases the significance of the delusional idea and its method of elaboration are obvious; in many they remain a matter of speculation. Hallucinations may frequently form the basis of subsequent delusional ideas. They are classified for descriptive purposes as delusions of unworthiness, self-reproach, poverty—depressive ideas; of grandeur, mania, G.P.I.; of persecution—paranoid. When the delusions are based around a central theme from which deductions have been logically made to form a coherent organisation of ideas, the delusional system is said to be systematised. *Insight* is the degree of conscious appreciation which the patient has of his abnormal symptoms and the nature of his illness, and is measured by the extent to which he is prepared to discuss these symptoms and recognises them as abnormal. An *obsession* is an idea that obtrudes into the mind, the individual being perfectly aware of its absurdity and control over his will. It differs from a delusion in that the nature of the idea is recognised as absurd, and the patient endeavours to rid himself of it. *Ideas of reference* are misinterpretations; the patient becomes convinced that various happenings or things recorded in the press relate to himself. When he believes that some force or other agency directs his activities so that he may perform certain acts even against his will, such are described as *passivity feelings*—commonly found in schizophrenia. *Nihilistic* ideas are associated with a marked disturbance of affect and

take the form of beliefs that the individual has no body, that he is dead or that the world has ceased to be.

§ **881. Disorders of Perception.** *Hallucinations* are sensory perceptions without any external cause. This contrasts with *illusions*, in which real perceptions are misinterpreted. Illusions are experienced by all at some time or other, and are common when there is clouding of consciousness. Hallucinations are frequent in the early stages of mental illness and are particularly common in toxic-infective conditions. Although physical factors may be present, the content is often influenced by previous psychological experiences. Hallucinations without any disturbance of consciousness—in other words, occurring in a clear setting—are of more serious import: in organic conditions hallucinations may show a nocturnal frequency. Hallucinations affect all senses, those of hearing are by far the most common. They may consist of indeterminate noises, but more often consist of words spoken by voices which may or may not be recognisable. In schizophrenia these voices may be said to arise from within and may be attributed to one of the internal organs. Hallucinations of sight are less frequent and usually occur with some clouding of consciousness; they often accompany toxic-infective illnesses, particularly delirium tremens, in which the hallucinatory experiences are very vivid and accompanied by intense fear. Hallucinations of smell are found in more chronic types of illness—chiefly schizophrenia; they are usually unpleasant and are associated with a sense of guilt. Hallucinations of taste are uncommon and are often associated with those of smell. Illusions of taste are more frequently experienced. Hallucinations of touch—haptic or tactile—occur in toxic-infective conditions, especially delirium tremens and cocaine addiction.

§ **882. Disorders of Memory.** Loss of memory is known as *amnesia* (§§ 716, 888). This may be for limited periods, not necessarily always sharply defined, and is then accompanied by some disorder of consciousness. It is a common result of head injury, a seizure or a hysterical attack. Amnesias connected with events the recollection of which is unpleasant, are frequent in hysteria. Anterograde amnesia denotes a loss of memory for recent events—retrograde that for remote events. An anterograde amnesia not accompanied by any disorder of consciousness is usually indicative of an organic syndrome. Generalised loss of memory is found in secondary dementia.

Paramnesia indicates a falsification of memory. In certain illnesses, particularly Korsakoff's psychosis, the memory deficiencies are filled in by the patient without any relation to fact; this is *confabulation*. *Hypermnesia* denotes an abnormally acute memory. Incidents with a strong emotional colouring may be recalled with greater ease. It is seen in hypomania, in paranoid states, and in certain prodigies, and may be seen even in gross mental defect.

§ **883. Disorders of Personality.** With *depersonalisation* the patient ceases to believe in his own existence: not only do external things appear

unreal or strange—*derealisation*—but there is a subjective feeling that he has lost his own reality. He does not believe that he is someone else—*transformation of personality*—but rather he has ceased to identify his own personality. *Splitting of the personality* is seen in schizophrenia. There is a disintegration of the personality coupled with independent activities of their functions so that grotesque incongruities of thought and action become possible. *Dissociation* is a mental mechanism to avoid conflict whereby a group of mental processes may be separated from the stream of consciousness and so functions on its own. Examples of dissociation are somnambulism, automatic writing and fugues. In *fugues* the secondary personality may generate such activity as takes the individual varying distances from his usual habitat. More complete degrees of dissociation give rise to *double* or *multiple personality*, in which the patient assumes a new disposition and character which may alternate with his normal recognisable self. The personalities then produced display, as it were, a complete ignorance of each other.

PART B. CLINICAL INVESTIGATION

As in the investigation of other bodily systems it is imperative to adopt some scheme of examination. To obtain an adequate understanding of the patient's mental make-up we largely depend on our ability to get the patient to talk of his problems ; this must be encouraged and directed by judicious stimulation and questioning. The personal factor enters into the examination to a greater degree than in the investigation of other systems. Whether the patient is co-operative or not, make it a constant rule to interview the relatives or friends in order to obtain an independent account of the patient's illness. Keep an open mind on the problem, and remember that statements of relatives need not necessarily be correct.

The close interrelationship between mental and bodily illnesses makes obvious the necessity for a detailed physical investigation.

Collect all data in an orderly manner and in chronological sequence even though the interpretation may be difficult.

§ 884. The first requirement is a clear understanding of the patient's *symptoms* and the development of these from the earliest time that any abnormality was noticed. Find out how far the present condition differs from his previous character and conduct : behaviour which indicates madness in one person is only eccentricity in another. Ascertain, where indicated, any peculiarities of behaviour, over-activity, violence, depression, suicidal preoccupations, excessive phantasy formation, etc. Investigate any *etiological factors*, domestic, business or financial difficulties, etc., so that you may judge whether or not such are adequate to cause the symptoms. Details of the *family history* must be obtained, particularly as to the incidence of alcoholism, drug addiction, a family tendency to eccentric behaviour or peculiar personalities, and other evidence of mental illness. The *previous medical history* and any earlier mental breakdown must be noted : obtain details of the previous personal history, *i.e.* early development, the presence of neurotic traits, school career, work record and habits. The *environment*, particularly during early life, should be enquired into especially with regard to (*a*) parental over-anxiety for the patient's health in early childhood,

(*b*) lack of harmony in the family, quarrels, separation of parents, (*c*) faulty education or upbringing, spoiling, temper outbursts, (*d*) ethical and religious training, sexual development in adolescent and adult life.

Finally enquire into the patient's *previous personality*: Was he of (1) the syntonic type, popular, a good mixer and sociable; (2) the schizophrenic type, reserved, shy, given to day-dreaming; or (3) the paranoid, inclined to be suspicious and see hidden meanings in things? Endeavour to get an impression of the patient as a whole, so that you may form an opinion as to his endowments and make-up. All such points are of enormous importance in evaluating the prognosis of a mental illness.

The success or failure of a psychiatric examination is often dependent on the physician's mode of approach. Never be in a hurry: any careless, inept, insistent or rapid questioning will only confuse and silence the patient and defeat your object. Endeavour to gain his confidence. To overcome this reserve, let him talk about things in general, then of his thoughts and feelings and finally of any hallucinations or delusions. Allow the patient to give his own account of his symptoms and their development. In making a mental examination, the following scheme is suggested as a guide; from the results thus obtained you are able to make any necessary judgments.

(1) Record your observations on the *patient's reaction* to the interview. Does he look ill? Pay attention to his facial expression and to his dress. Is there over-activity or retardation? If retarded in his movements, does he resist passive movements; is there negativism; flexibilitas cerea; are mannerisms evident? (2) Consider next the *patient's conversation*. Is the enunciation clear; is there evidence of slurring, as in G.P.I.? Note the stream of mental activity; are questions answered promptly and relevantly? Is there excessive volubility; jumping from one topic to another (flight of ideas); are there self-invented words (neologisms) or there may be diminished volubility, with evidence of retardation of the mental processes. In stupor, the patient may refuse to speak or answer questions—mutism. (3) From the patient's appearance, one may get an impression as to the *mood or affective state*. If it is constant, is it appropriate to the emotional state? Carefully note any discrepancy or fluctuation in the mood, such as a smile in a situation which should produce a depressive response. Augment observation by direct enquiry—"How do you feel? How are your spirits?" etc. (4) Much may be obtained from the *content* of the patient's conversation. He is therefore encouraged to talk freely. General enquiries should be made to determine his reaction to those with whom he comes in contact. Are delusional ideas present? What is the patient's reaction to his environment? Is he suspicious? Does he think that he is treated badly or in any special way? Is he persecuted?—paranoid. Are there passivity feelings, that people can influence him to do things? Does he feel that his thoughts can be read? Can he read those of others?—schizophrenic. Are there grandiose ideas—of enormous power or wealth—mania, G.P.I.? Are there depressive ideas such as those of self-reproach, that he has done great wrong, committed the unpardonable sin, that there is no hope, that life is not worth living, found in melancholia? Is there evidence of illusions or of hallucinations? The type, degree of vividness, time of occurrence and content must be noted as also the patient's impression of these. Ascertain if there is obsessional preoccupation either in thought or action. How far do such control activity: do they emanate from within the mind or from an outside force? In regard to delusions, illusions or hallucinations, estimate how far such perversions of the mind influence or are likely to influence the acts or conduct of the individual.

Ascertain if the patient is correctly orientated as to time, place or person. Determine any memory defects, both as regards recent and remote events. What is his attitude towards such—does he fill in the gaps by confabulation (Korsakoff's)? What is his level of general information; is it adequate, considering his social class; is there evidence of deterioration from the former level? Finally, does the patient regard his condition as an illness for which treatment is desired? Has he any in-

sight? This is of value in assessing the prognosis and in recommending treatment. The essential facts in the illness can now be summarised and an opinion formed as to diagnosis, prognosis and treatment.

PART C. DIAGNOSIS, PROGNOSIS AND TREATMENT OF MENTAL ILLNESSES

§ 885. Classification. PSYCHONEUROSES and PSYCHOSES in their characteristic forms may appear to be distinct entities, easily differentiated from one another. However, in some cases the line of demarcation is not always so clearly defined, and the one may merge into the other. The essential difference is the degree to which the personality is involved: in *psychoneurotic illnesses* the personality remains intact, there is ability to differentiate between reality and subjective experiences and the patient retains insight into the nature of his illness and is anxious to get well. The *psychotic patient*, on the other hand, shows distortion and disintegration of his personality, he loses his sense of reality, frequently lives in a world of phantasy, and his beliefs are not amenable to reasoned explanation.

The resultant forms of illness are not to be regarded as disease entities, but rather as *types of reaction* to various forms of stress. These may appear in several forms :—

GROUP I.—COMMON MINOR MENTAL ILLNESSES.

I. Anxiety Neurosis .. .. § 886
II. Neurasthenia .. .. § 887
III. Hysteria .. .. § 888
IV. Traumatic Hysteria .. .. § 889
V. Obsessive-Compulsive Neurosis .. .. § 890
VI. If there is Abnormal Behaviour without gross Psychotic Symptoms, turn to .. .. § 891

GROUP II.—MAJOR MENTAL ILLNESSES.

I. If there is Excitement or Exaltation, turn to .. .. § 892
II. If there is Depression or Retardation, turn to .. .. § 894
III. If there are Hallucinations or Bizarre Delusional Ideas accompanying the affective changes, turn to .. .. .. § 897
IV. If the dominant system of ideas is that of Persecution, turn to § 898
V. If there is Addiction to Alcohol, Drugs, or there is Delirium associated with fever or other physical disorder, turn to § 899
VI. If Organic features are prominent, turn to .. .. .. § 902

IN CHILDREN.

If the patient is a Child, turn to .. .. § 907
If abnormal Nervous or Mental Symptoms appear in early life unaccompanied by mental retardation, turn to .. .. § 907 B
If there is Mental Retardation or Mental Defect in a child, or if the condition has persisted from childhood to adult life, turn to .. § 907 C

GROUP I. COMMON MINOR MENTAL ILLNESSES

Psychoneurotic illnesses comprise mental disturbances (anxiety, fugues, phobias) or physical symptoms (paralyses, anæsthesiæ)—for which no organic cause can be discovered. They are in general terms the result of mental conflict, dealt with by repression. The conflicting forces can often be traced to instincts of self-preservation, gregarious or herd instincts, to reproductive instincts, or to ethical teachings implanted in childhood. The various methods have been discussed already by which such repressed factors obtain expression and produce different types of neurotic symptoms.

The patient complains of loss of interest, inability to concentrate and various fears. The condition is ANXIETY NEUROSIS.

§ **886.** I. **Anxiety Neurosis** (Synonym : Psychoneurotic Anxiety State) is the commonest form of Psychoneurosis. Many cases, diagnosed Neurasthenia, are really examples of Anxiety Neurosis.

Symptoms.—(1) Anxiety is the chief symptom, and this, acting through the autonomic nervous system, produces all kinds of visceral symptoms. Fears of bodily or mental illness, of suicide or death, are common. Acute panics, lasting minutes or hours, characterised by paroxysmal terror and emotional distress, occur. Between these panics the patient's emotional tone is one of anxiety. He fears to meet people, fears to walk in the traffic, and may sit in the house all day. Sleeplessness, arising from his anxiety, adds to the patient's distress ; when sleep occurs, nightmares are often present. (2) Visceral symptoms, visceral reactions secondary to the panics and prevailing anxiety, are almost constantly present. Besides the physical concomitants of fear (viz., dry mouth, shaking, tachycardia, frequency of micturition) all kinds of gastro-intestinal symptoms may arise—vomiting, nausea, diarrhœa, water-brash, constipation, etc. Seminal emissions, blurring of vision, twitchings of muscles also occur. To these morbid fears, which are of infinite variety, various names have been given—fear of open spaces (agoraphobia), fear of closed rooms and buildings (claustrophobia). These are not different diseases but are part of an anxiety state, varying according to the patient's history. Fear of dirt, of knives and scissors, of medicines, of travelling in tubes, lifts, omnibuses, etc., are also met with. The patient cannot explain why he dreads these things, but realises their absurdity.

Diagnosis.—Anxiety as a symptom is common in *obsessional neurosis, melancholia* and *schizophrenia*. The presence of the other characteristic features of these conditions simplifies the diagnosis. Anxiety neurosis results when the reaction produced is out of proportion to the cause, real or fictitious. It is the commonest form of psychoneurosis.

Etiology.—(i) Hereditary, constitutional, and environmental factors. (ii) The patient is usually emotionally unstable ; prolonged anxiety becomes a habit. (iii) A sexual basis was propounded by Freudians to

explain such reactions, but there is no doubt that anxiety neuroses may develop from mental conflicts arising from many sources, of which the chief are domestic, financial, sexual, and business worries.

The *Prognosis* is better than in any other form of psychoneurosis. Apart from psychological treatment much will depend on how modifiable the other etiological factors may be.

The patient complains of tiredness, lack of concentration, irritability, feelings of pressure in the head, and other subjective symptoms for which you can find no organic cause. The disease is NEURASTHENIA.

§ 887. II. **Neurasthenia** (Synonym: Chronic Nervous Exhaustion), as a clinical entity is rare, and the term is one of the most abused in medicine. Mainly characterised by fatigue, some degree of anxiety is also usually present, and the relative importance of each has to be assessed. *Symptoms.*—(1) The chief symptom is tiredness, a ready mental fatigue and physical prostration without discoverable lesion. The tiredness of the neurasthenic differs from ordinary exhaustion in that it is not relieved by a night's rest, and it is not always precipitated by factors which cause exhaustion in the normal individual. (2) There is fatigue of cortical inhibition and control, manifesting itself by abnormal irritability over trifles, lack of emotional control and motor restlessness. All kinds of visceral sensations, normally inhibited by cortical control from reaching the sensorium, become manifest and the patient is conscious of the beating of his heart, peristaltic movements of his intestines, etc. (3) The patient becomes egocentric and introspective, the whole of his consciousness filled with his abnormal physical sensations. So full is he of his illness that he cannot devote his attention to outside affairs and he suffers from lack of concentration, which he wrongly attributes to mental failure. (4) There is a peculiar kind of headache, which is described as a pressure rather than a pain, or an aching tightness radiating from the frontal region into the back of the neck or the spine. At other times the feeling in the head is described as a " woolliness " or " cloudiness." The discomfort is never actual pain, but is described as something worse and less bearable than pain. (5) Sleeplessness adds to the patient's misery. (6) Symptoms referable to the autonomic nervous system and endocrine glands occur, tachycardia, tremor, vaso-motor instability and loss of weight. The blood-pressure is invariably lowered in severe cases, with giddiness, coldness and clamminess of the extremities and tachycardia, pallor or syncope on suddenly assuming the erect position. (7) The menstrual periods in women may be irregular. All kinds of disturbances in the sexual sphere occur and may dominate the clinical picture. Sexual impotence, premature ejaculations, spermatorrhœa, all occur in men. The urine usually contains a heavy deposit of phosphates and is alkaline. Phosphates may be deposited in the bladder and appear in the urine at the end of micturition, the patient mistaking the phosphate deposit for seminal fluid and imagining that his virility is draining away.

Diagnosis from the other psychoneuroses is generally not difficult. Fatigue may be a hysterical conversion symptom. It is more difficult to differentiate between neurasthenia and a depressive illness. Fatigue is prominent in both, but there is more affective disturbance in depression; the previous history and familial incidence often aid diagnosis. Early schizophrenia and neurasthenia are not always easy to distinguish.

Etiology.—Psychological factors are the primary cause. Neurasthenia should not be accepted as the diagnosis until adequate mental phenomena have been elicited to account for the symptoms. Opinions vary as to whether the constitutional factors present are contributory or secondary to prolonged emotional conflict. Overwork is not causative; lack of occupation and of emotional outlet may be predisposing causes.

Treatment of Anxiety Neurosis and Neurasthenia.—It is essential to appreciate the psychological origin. (i) At the outset obtain a detailed account of the patient's illness and of the development of his symptoms; and at the same time his own ideas on his illness. (ii) So often he fears physical disease that the next step is a *complete physical examination*: this must be thorough, so that when negative you can reassure the patient with confidence that there is no physical basis for his symptoms. (iii) It is important to explain that recovery takes place in irregular fashion, good days being followed by bad days, until eventually the bad days disappear. By this time the confidence of the patient should have been obtained, an essential factor for progress. (iv) Next, try to trace the cause of the anxiety; for this a knowledge of mental mechanisms is generally necessary. Nevertheless by patient enquiry and encouragement each symptom can usually be traced to its origin. Advise as to the effect on the general health of an emotional upset and demonstrate how the symptoms have developed during such periods. Make it clear that you do not consider the symptoms are imaginary, but that they originate from genuine causes. For example, the exhaustion which often accompanies these psychoneuroses can be explained in terms of anxiety or emotional perturbation, the anxious preoccupation preventing the patient from using his available store of energy. Sexual impotence may be traced to false ideas of physical damage resulting from past habits of masturbation, rendered more distressing by associated feelings of remorse. (v) If environmental factors operate largely in the etiology, or if the symptoms are severe, it may be necessary to remove the patient for a time from the atmosphere of his conflicts. If isolation is practised, it should be complete, the patient being allowed to see no one but his doctor and nurse, to write and receive no letters. In slighter cases it is sufficient to re-educate the patient to face up to his difficulties and make whatever adjustments are possible.

Sleeplessness.—It is essential to ensure adequate sleep. Sleeplessness is to be treated on the lines laid down in § 697. It is better that the patient, by using an effective hypnotic, should secure sufficient sleep, than that he should go on having nights filled with wakeful, anxious preoccupa-

tion. *Occupational therapy.*—If the patient is unable to concentrate on reading, the periods of rest prescribed may be usefully employed in the practice of rug-making or some handicraft. Occupational therapy of this kind helps to restore confidence (§ 905). *Diet.*—If the patient is physically below par, careful feeding with extra amounts of cream and milk foods, and small doses of malt and cod-liver oil are indicated. *Massage.* —Daily massage and exercises will build up the general muscular tonus. For stronger patients exercise in the fresh air, short of fatigue, will be helpful. *Drugs.*—In nearly all cases the administration of such a mixture as sodium bromide gr. 7, liquor arsenicalis ♏ 1, tinct. belladonnæ ♏ 6, aq. aurantii ad fl. oz. ½, thrice daily after meals, will help to stabilise the emotions of the patient. Alcohol is seldom advisable and tobacco should be limited. Strychnine, in even small doses, nearly always makes the patient worse by increasing general hypersensitiveness. *Change of Environment.*—When the patient has recovered sufficiently, a holiday in a dry, warm climate may be prescribed. He should not be rendered anxious by needless restrictions and warnings.

To summarise, the methods of treatment are: (1) *Explanation* and analysis of the symptoms, (2) *Persuasion*, by all kinds of encouragement and reassurance, and, lastly, (3) *Re-education*, by general methods, until the patient's adaptations to his environment are normal.

There are physical signs of loss of function but without signs of organic disease. The disease is HYSTERIA.

§ **888.** III. **Hysteria.**—The results of a hysterical type of reaction to a distressing situation are extremely varied but present a clear clinical picture. The hysterical reaction is a manifestation of a childish need for dependence on others, and the hysterical individual solves her problem usually by the production of some physical symptom, with unconscious desire that it will remove her from the scene of her difficulties. She is, however, entirely unaware how her symptoms have arisen. The temporary solution of the problem brings her a characteristic composure and indifference (" la belle indifférence " of Janet) and a measure of mental relief, wrongly attributed by her friends to fortitude or resignation. Such patients are commonly extremely egocentric and selfish; there may be an outward show of great emotion, but there is probably very little real emotional content.

Clinically, the disease shows characteristic physical and mental symptoms:

(1) PHYSICAL SYMPTOMS, characteristically produced by suggestion, curable by suggestion and without objective evidence of structural nervous disease. The mimicry of organic nervous disease may be very close but is never complete, and there is usually some paradoxical phenomenon present, revealing the true cause of the symptom, *e.g.*, hoarseness may be present in speaking, yet the cough is clear.

(2) Mental Symptoms such as amnesia, delirium, twilight-states, stupor, fugues, somnambulism, and self-mutilation.

It should never be forgotten that hysteria and organic disease may co-exist in the same patient. This is especially true of the traumatic cases.

1. Physical Symptoms.

(*a*) Motor Symptoms.—When *Rigidity* is present it is proportional to the force used to overcome it, on attempted passive movement. The associated contraction of the antagonistic muscles can be palpated when the patient attempts voluntary movement. *Flaccid hysterical palsies* are commoner than rigidity. When the leg is affected it trails behind the patient, who hops on the sound limb with the dorsum of the affected foot dragging on the floor behind him. All kinds of bizarre *hysterical gaits* are met with, which may be associated with remarkable spinal curvatures occasionally simulating hip-joint disease. In these conditions (i.) there is never any alteration, either quantitative or qualitative, in the deep or cutaneous reflexes, although the plantar response may be absent at the toes, (ii.) the muscles react normally to electrical stimulation, and (iii.) there is no wasting, apart from that occasioned by disuse.

Hysterical tremors and tics vary in degree and are increased by attention. Hysterical tremor may closely simulate the tremor of paralysis agitans or disseminated sclerosis, but other features of these diseases are absent. Paroxysms of violent tremor may pass over into hysterical fits. Slighter forms accompany paralysis and rigidity of hysterical origin. Hysterical blepharospasm is fairly common.

In *Hysterical Aphonia* the patient whispers but can phonate normally on coughing. On laryngoscopic examination the cords are not sufficiently approximated to produce a sound.

In *Mutism* there is complete abolition of speech, although the organs concerned are used normally for clearing the throat, coughing and mastication.

Hysterical recurrent cough is fairly common.

(*b*) Sensory Symptoms.—Sensory loss affects only *cutaneous sensibility* and is usually complete. Sense of position (which is outside the patient's knowledge) is unaffected, as evidenced by accurate co-ordination of movement. Sensory loss may be of the " stocking " or " glove " distribution in the limbs. The upper level of a hysterical analgesia has the following characters: (i.) It is horizontal. (ii.) It varies from time to time on successive examinations. (iii.) The transition from analgesia to normal skin is abrupt, without any area of impaired sensibility or hyperæsthesia, as in organic disease. In hysterical hemianæsthesia the corneal reflexes remain intact and the sensory loss ceases abruptly at the mid-line in front; whereas in organic disease it is continued for a short distance across the mid-line in front. In hysteria there is often affection of all the spinal senses on the side of the hemianæsthesia. For Janet's " Yes-No " test, see § 706.

Hysterical blindness is often sudden in onset and may be complete or

incomplete. In the complete form the patient commonly avoids obstacles placed in his path. In the incomplete forms perimeter charts show an unequal constriction of the visual fields in the two eyes (hysterical amblyopia) or a spiral "fatigue" field. Diplopia can be produced by pressure displacement of one eye. The pupils and optic discs are always normal. *Hysterical deafness* can be recognised by the fact that the patient can be wakened from sleep if called by name, but when awakened he cannot hear. *Hysterical anosmia* is common in plumbers after gas explosions, and includes loss of sensation to ammonia vapour, which is a function of the fifth, not the first nerve.

(c) VISCERAL SYMPTOMS.—Digestive disturbances are not infrequent and to the relatives most alarming. *Hysterical globus* is the sensation of a lump in the throat interfering with swallowing. *Hysterical ærophagy* may lead to enormous abdominal distension with simulation of pregnancy or ovarian cyst. *Hysterical vomiting* is seldom accompanied by any profound loss of weight: there is no nausea and the expulsion of certain foods from the stomach will not prevent the retention immediately afterwards of others more palatable. In a few cases nothing is retained and progressive emaciation results. In diagnosing these conditions great care must be taken to exclude the presence of organic disease. The chronic abdominal pains of hysterical women are often the cause of repeated laparotomies, for "adhesions," "fixation," etc. The patient, unaware of the true cause of her malady, often invites operation with amazing eagerness.

Anorexia Nervosa is characterised by loss of appetite or a persistent refusal of food. It is a more serious condition, found more frequently in young women, and progressive emaciation may develop even to the point of death. The refusal of food is at first involuntary but soon all desire for food is lost. The condition should be distinguished from the anorexia seen in melancholia and from the cachexia of endocrine origin.

The term *Effort Syndrome* describes a clinical picture that may develop in a variety of nervous conditions. The symptoms comprise breathlessness, giddiness, palpitation, tachycardia, sweating, pain and exhaustion, and these develop during or are aggravated by exercise (§ 34). They occur most frequently with anxiety or with varying degrees of depression, but they may be superadded to a hysterical or a psychopathic personality. The physical methods of treatment, graduated exercises, occupational therapy, etc., will obviously be augmented by psychological treatment of the primary disability.

(d) CUTANEOUS SYMPTOMS.—Blueness, coldness, variable œdema, are all met with in hysterically paralysed limbs. Dermographism is common. Anomalous skin eruptions produced by the patient by rubbing, corrosives, or by burning (§ 608).

(2) MENTAL SYMPTOMS.

Hysterical amnesias are frequently for limited periods of time and follow a marked emotional disturbance. Similarly *delirious states* follow an emotional upset; the degree of confusion is variable and the flow of talk is of a nonsensical type. The *twilight state* (Ganser syndrome) occurs essentially among prisoners and represents an attempt to appear irresponsible; the purposeful nature is obvious. *Stupor* may be maintained for prolonged periods but terminates rapidly once the precipitating factors have ceased to exist. *Amnesias, fugues,* and *somnambulism* are evidence of dissociation. The dissociated portion of the personality functions independently and for the time being controls the patient's activities. *Self-mutilation* is met with and suicidal threats and attempts may be made; the latter are invariably dramatic.

(e) HYSTERICAL FITS never occur between definite hours of the day, as do epileptic fits, and never during sleep. They take place in the presence of an audience, there is never any incontinence or tongue-biting, and the patient never injures herself, although others may be injured. The eyes are usually screwed up and the hands

clenched, and the movements are "purposive." Hysterical fits may follow upon true epileptic fits. They may be accompanied by outbursts of unrestrained laughing and crying.

Etiology.—(i.) Constitutional factors are important. (ii.) Hysteria often occurs in families with a history of schizophrenic, alcoholic or other psychopathic disorders. (iii.) It is more common in adolescence and at the menopause, and (iv.) in females than males. (v.) It occurs in a hysterical type of personality, characterised by egotism and an attitude of posing or make-believe. Such patients show a marked emotional susceptibility and are readily influenced by and imitate those who appeal to them. (vi.) Mental stress is the exciting factor; the symptom achieves some aim or desire (often not consciously appreciated by the patient) and thus allows a maintenance of self-respect otherwise impossible.

Prognosis.—Individual symptoms are usually easy to cure but the liability to recurrence is great. The prognosis depends on how far the personality can be modified and the etiological factors readjusted.

Treatment.—Treatment directed merely to the relief of local symptoms is inadequate, though by suggestion, persuasion, and re-education it may be temporarily effective; somatic symptoms respond readily to suggestion. It is not, however, enough to remove the symptoms; efforts must be made to elicit their mechanism; until this is discovered and treated no permanent benefit is derived. The employment of all psychotherapeutic methods (§ 905) may be necessary at some time or another to achieve this end. The chief obstacle to effective treatment is the mental indifference of the patient; this prevents an appreciation of the causes which have led to the development of the symptoms. Environmental factors must be considered and readjusted as far as possible. The thought of return to the arena of conflict is, in some cases, insupportable, *e.g.*, the childless woman will not return to her drunken husband. In such cases, you must do what you can to make the patient's environment more tolerable. In all cases, she should be encouraged to do some kind of useful work.

§ **889.** IV. **Traumatic Hysteria** (Synonym: Traumatic Neurasthenia). The symptoms of this neurosis must be carefully distinguished from those of *Unresolved Cerebral Contusion* (see § 696. VII).

Immediately following upon an accident, which may be trivial, or more usually some days or weeks after the accident, the patient develops hysterical symptoms. The condition is particularly likely to occur in injuries to the head or spine. There is always an additional element of anxiety in the symptoms, due to the uncertainty in the patient's mind as to the outcome of litigation and the possibility of his return to work. It occurs in workmen, especially those engaged in dangerous occupations and who work at a height from the ground. Such cases often come into Court in connection with the Workmen's Compensation Act.

Symptoms.—Mentally, the patient becomes extremely introspective, with a profound conviction of the seriousness of his complaint. He becomes sleepless and irritable, and his anxieties are increased by his

uncertainty of his ultimate recovery and return to work, and the financial straits into which his illness throws him. Physically, all kinds of hysterical phenomena are met with, tremors, paralysis, spasms, including convergent ocular spasm, hysterical gaits and attitudes, and hysterical affections of the special senses and cutaneous sensibility. There is commonly loss of weight, tachycardia and low blood-pressure. Pains of all kinds are complained of, and these may indeed be due to associated organic disease, *e.g.*, Spondylitis. The distinction from malingering is often difficult. In malingering there is conscious attempt to deceive others for gain; unlike the hysteric, the patient is not deceived as to the mechanism of his symptoms.

Treatment.—A period of rest in bed for two weeks, with a full neurological and X-ray examination, will prepare the way for cure. If no organic disease is found the patient should be informed of this and his symptoms should be tactfully explained to him. Hypnotics and sedatives may be necessary to secure the sleep essential in treating such cases. Explanation should be combined with reassurance and re-education. Graduated gymnastic exercises, physical work, ladder-climbing, weight-lifting, may all assist in the cure. The symptoms, however, do not usually entirely clear up until legal proceedings are finally settled, and the patient has proved or failed to prove his case against those he deems responsible for his illness. Even after such a settlement, symptoms occasionally persist, owing to the anxiety occasioned by his illness.

The patient is dominated by some thought or action recognised as senseless and accompanied by a feeling that it must be resisted. The condition is OBSESSIVE COMPULSIVE NEUROSIS.

§ 890. V. **Obsessive Compulsive Neurosis** (Syn. Psychasthenia). Almost everyone has experienced at some stage in life obsessions or compulsions of a mild degree. They are frequently present in minor degree in childhood and are rarely to be taken seriously then. The condition is an attempt to ward off unknown evil by a process akin to magic. When present in later life they are of much greater significance and may be so severe that the greater part of the patient's life is spent in attending to these symptoms. The condition is characterised by: (1) *Anxious preoccupation with some obsessive idea.* The patient may ask himself over and over again some religious or metaphysical question, *e.g.*, "What was the beginning of everything?" Some are disturbed by the reiteration of certain words or phrases in their mind. These are frequently symbolic and may be so severe as to exclude other interests. (2) *Morbid compulsions.* It is easy to appreciate how ritualistic actions may develop as a means of combating the fears associated with the obsessive ideas, *e.g.*, a fear of dirt and contamination may necessitate constant washing and scrubbing of the skin to ensure cleanliness. Patients may feel compelled to touch objects, to do things in a certain order, to steal something intrinsically worthless, which they do not need (kleptomania), to commit homicidal acts or sexual assaults, to take alcohol (dipsomania) or drugs. There is, sometimes, only fear that these imperative acts will be committed, and the act is never actually carried out.

Prognosis.—When the onset is insidious and there is no marked affective disturbance the prognosis is poor. The course of the illness is invariably prolonged. It has been frequently stated that it is not uncommon for obsessional-compulsive states to be followed by a psychosis. This seems doubtful. If the obsessional symptoms are

part of a depressive illness and accompanied by the other characteristic features of an affective disorder the prognosis is reasonably good.

Treatment.—The hereditary basis is marked. When the morbid impulse or obsessional idea is part of a depressive illness, the treatment is as for melancholia. The more severe forms of insidious onset are not readily amenable to psychotherapy, which is necessarily prolonged.

The patient exhibits ANOMALIES OF BEHAVIOUR, *characterised by* INADEQUATE SOCIAL ADAPTATION, ANTAGONISM TO OTHERS, *or by* PATHOLOGICAL SEXUAL IMPULSES WITHOUT *gross* PSYCHOTIC SYMPTOMS *or intellectual impairment. The condition is* PSYCHOPATHIC PERSONALITY.

§ **891.** VI. **Psychopathic Personality.**—*Symptoms* often appear in early life, generally in the form of excessive emotional displays, temper tantrums, pathological lying, and antisocial behaviour of a criminal kind, stealing, wilful destruction, etc. (§ 907). As puberty normally requires numerous fresh adaptations between instinctive desires and social standards, at this time the expression of a psychopathic personality invariably becomes more pronounced. Antisocial actions become more frequent, more acute, and are generally of an episodic nature and quite uncontrollable: they are devoid of reflection or any consideration for feelings of themselves or others, and their execution produces no sense of remorse. These abnormal acts generally occur in a setting of clear consciousness with no other abnormal psychotic manifestations, so that it is extremely difficult to exercise control. The intellectual level is invariably within normal limits.

Etiology.—The chief etiological factors seen are the effects of heredity and environment. A similar picture may be seen associated with organic diseases of the brain, *e.g.*, epilepsy, encephalitis lethargica.

Prognosis.—The prognosis is serious, particularly if treatment is not instituted before adult life.

Treatment.—This as it applies to problems in childhood is described in § 907. At this stage prevention offers the greatest measure of success. Ascertain the factors that determine or aggravate the outbursts. If environmental factors are prominent, effect a change in this direction. Where antisocial acts of conduct are serious, admission to hospital may be necessary. The provision of colonies for the supervision and treatment of these patients has been repeatedly advocated. Once adult life is reached the results of psychotherapeutic treatment are disappointing.

GROUP II. THE MAJOR MENTAL ILLNESSES

§ **892.** I. STATES OF EXCITEMENT and EXALTATION. Acute mental excitement may be a **transient** phenomenon in the following conditions, where it is clearly traceable to some bodily disorder. DELIRIUM (§§ 469 and 901); ALCOHOLIC and DRUG INTOXICATION (§§ 899 and 900); HEAD INJURIES (§ 716); THYROTOXIC CRISES (§ 186); EPILEPSY ("Equivalents") (§ 721).

Continuous mental excitement is found in:—MANIA (§ 893) where the mental condition is the only, or at any rate, the principal symptom; AGITATED MELANCHOLIA (§ 895), where the agitation is accompanied by severe depression; ACUTE TOXIC CONFUSIONAL PSYCHOSIS, where hallucinations and delusions are prominent features of the excited state, together with symptoms of profound exhaustion; SCHIZOPHRENIA (§ 897), when it is accompanied by delusional ideas frequently grotesque in nature; and in GENERAL PARALYSIS (§ 902), when accompanying physical signs are apparent. When a patient is confused and delirious, investigate for drug intoxication, such as that following the cumulative effects of a sedative, especially bromides, taken for weeks or months; URÆMIA may produce similar symptoms (§ 372).

The patient is continuously excited, restless, garrulous, showing flights of ideas, or lacking in control. The disease is MANIA.

§ **893. Mania** is classified as "Simple," "Acute," and "Chronic."

Simple Mania.—In this mild form of excitement one of the principal features is a loss of self-control. This is accompanied by an exaggerated sense of well-being. The patient is talkative, restless, and optimistic. Often he is interfering and irascible. There is a general failure of judgment and instability of purpose.

Acute Mania may supervene suddenly—(1) during convalescence from exhausting diseases (as previously mentioned); (2) in the course of other diseases of the nervous system—*e.g.*, G.P.I.; (3) in the course of some other form of mental illness. Its onset is usually rapid, tongue-tremor being often met with in the early stage. The stage of excitement is soon reached—loquaciousness, sleeplessness, continual restlessness, incoherence in which delusions and ideas succeed each other with great rapidity, sometimes relating to moral and religious, at other times to intellectual topics. After lasting some weeks or months, recovery (sometimes quite suddenly) ensues; sometimes it is followed by moral or mental obliquity or dementia; it may pass into chronic mania. The temperature is normal throughout. In many cases there is a tendency to relapse and sometimes an alternation with melancholia.

Chronic Mania is suspected when a patient's restless excitement, incoherent conversation, and disordered conduct continue, although sleep and appetite have fully returned and are accompanied by improvement in the physical state. Delusions become more prominent and auditory hallucinations may be a feature. The condition is rarely seen before middle life.

Acute Delirious Mania is an acute maniacal condition which may follow upon one of the other varieties of mania or develop suddenly in a person in apparent health. It is attended by pyrexia, usually running a rapidly fatal course, no lesions being found after death. It is happily rare. The symptoms come on abruptly, and quickly amount to frenzy, accompanied by outbreaks of great violence and refusal of food. The temperature ranges irregularly from 100° to 104° F., and in the course of one to three weeks the disease terminates in great bodily prostration, and often in death. The majority of these cases are nowadays better described as **Acute Toxic Confusional Psychosis.** The toxic nature of the illness is evidenced by the rise in temperature, the vivid numerous and fleeting hallucinations, the intense confusion, and the state of muttering delirium which supervenes. The patient has all the appearance of one suffering from toxæmia, the complexion is pale and muddy with perhaps a malar flush, the mouth and tongue are dirty, and there is sordes on the lips. From acute mania it is known by the fever, the rapid wasting, and more rapid and fatal termination. It resembles some cases of typhoid fever very closely, acute pneumonia and acute meningitis, but their distinguishing signs are absent.

The *treatment of mania* consists mainly of rest in bed, preferably in the open air, and in the administration of food. Narcotics and depressants are invariably required. Paraldehyde is of great value in inducing sleep. Sulphonal has been recommended to allay the excitement, but the danger of methæmoglobinæmia (§ 32) must be remembered and its use over a long period discouraged. In recent years innumerable modifications of the barbituric acid group have been elaborated and are frequently used for continued narcosis. The degree of somnolence maintained is such that the patient can be aroused for meals. The drug most frequently employed is somnifaine 2 c.cs. intramuscularly two or three times daily, after an initial induction of sleep by the hypodermic injection of morphia gr. ¼, hyoscine gr. 1/100. It should be remembered that the threshold between the therapeutic and toxic doses is low and complications may readily ensue. To prevent such, the administration of glucose and insulin has been recommended; the degree of success achieved thereby is, however, not always complete. Hydrotherapy in the form of the continuous bath is extremely valuable. Constant supervision is necessary as there is a danger of collapse. The temperature of the water may vary between 96° and 98° F. The successful management of maniacal patients depends on tactful and patient handling. By such means it is usually possible to ensure adequate nourishment and rarely is tube feeding necessary. The principal indications in the treatment of *acute toxic confusional* psychosis are to control the extreme restlessness by hypnotics, to promote elimination as freely as possible and to maintain the patient's strength. The bowels should be well washed out with a high enema at least twice weekly, and the patient should be given copious fluids, by stomach tube if necessary. The mouth should be kept clean and the general medical and nursing indications are similar to those required in the treatment of any acute toxic and febrile illness.

§ 894. II. MENTAL DEPRESSION and RETARDATION may occur in: (1) MELANCHOLIA, where the signs and symptoms of depression are constant, and are the dominant features of the illness; (2) SCHIZOPHRENIA, where the affective change is inadequate, or bizarre delusional ideas are prominent; and (3) GENERAL PARALYSIS, in which disease the accompanying *physical* signs are the determining features.

The patient is continuously depressed, self-reproachful and hopeless. The disease is MELANCHOLIA.

§ 895. Melancholia is a morbid condition of miserable self-consciousness and self-abnegation without hope. The onset is usually insidious, and commences with extreme self-consciousness, combined with sadness, as indicated by depression, without adequate cause, and the patient is irritable when remonstrated with. He loses interest, finds increasing difficulty in concentration, develops fears of impending calamity which cannot be named and becomes sleepless. Self-reproachful ideas are conspicuous and often refer to minor events that may have occurred years previously. The diagnosis is strengthened by a history of a previous attack, or an intervening period of high spirits and bounding energy. A family incidence of depression puts the diagnosis beyond doubt. Always regard such patients as potential suicides. The degree of affective response differentiates such patients from those suffering from hypochondriasis.

Melancholia may be simple, acute, or chronic. (1) **Simple Melancholia** is characterised by a lack of interest, loss of feeling for others, inability to concentrate as before and a dulling of the mental processes. It consists simply of misery, sleeplessness, self-reproach, and inability to continue at work. This form is common in the overworked or much-worried, and in women at the climacteric. There are no hallucinations, but self-reproachful, depressive ideas are common and characteristic. Suicidal preoccupations are usual in these cases and they constitute a large proportion of the

suicides that occur each year. Adequate precautions, which are sometimes neglected on account of the simplicity of the affection, should not be omitted. Otherwise the prognosis is favourable. (2) **Acute Melancholia.**—The symptoms already mentioned are present but to a more marked degree. There is marked psychomotor retardation and the picture is that of the most profound misery. Delusional ideas are marked and are of a self-accusatory nature, *i.e.* they think they are the most wicked individuals, that they have committed the unpardonable sin, etc. Hypochondriacal ideas are also prominent and refer to alleged dysfunction of bodily organs. Auditory hallucinations may be prominent. Food is frequently refused because of ideas of unworthiness and the general condition deteriorates. Insomnia is marked. (3) In some cases **Melancholic** or **Benign Stupor** is met with. The patients lie in bed speechless, motionless, and are often negativistic. Their habits are faulty; in some retention of urine and fæces has to be specially watched for. Their limbs may be flaccid or in cataleptic rigidity. Although they may appear oblivious to external stimuli they retain as a rule a surprising degree of appreciation of their environment while in this state. They resist external interference, but are not usually violent. It is equally common in both sexes, but is more frequent in the young than in the old. Sometimes it follows a severe and exhausting illness, and sometimes it follows acute mania. (4) **Chronic Melancholia.**—In this condition, the symptoms of depression persist, but they are obviously less acute than in the other forms. Although the patient still has the same despairing attitude towards life, sleep returns, food is taken satisfactorily, and the physical state improves. (5) **Agitated Melancholia.** The characteristic picture is that of severe depression without retardation. The age of onset is usually the involutional period though an earlier incidence is not unknown. Anxiety is marked and is accompanied by extreme motor restlessness. The patient paces up and down wringing his hands and repeating the same despairing remarks over and over again. Auditory hallucinations and delusional ideas are prominent. The absence of retardation increases the degree of suicidal risk. The general physical condition deteriorates because of the difficulty in feeding and the expenditure of much energy.

Recurrent Mania and **Recurrent Melancholia.**—In many instances there is a tendency for a patient to have repeated attacks of mania or melancholia. In such cases it is often remarkable how faithfully reproduced are the general symptoms and the individual peculiarities which have characterised former attacks. For many years it has been recognised that there is a close alliance between mania and melancholia. In the history of almost every attack of depression one finds evidence of an elated phase having been experienced, and similarly, attacks of elation may be preceded or followed by depression. For this reason both mania and melancholia now come under the one classification, namely, **Manic Depressive Psychosis.**

Course and Prognosis.—The melancholic process is longer than the maniacal one. The duration varies considerably but lasts an average of some three to twelve months. However, cases of melancholia may recover even after a very long time—up to fourteen years has been recorded. Relapses are frequent. Heredity is an important factor and the nutrition of the body at the time is another. The danger of exhaustion and intercurrent infection is great in the agitated variety; about 10 per cent. of such terminate fatally in spite of the most careful attention. The presence or absence of an adequate cause, the type of onset, the form of the illness and the absence of other features (arterio-sclerosis, etc.) are the important factors to consider in estimating the prognosis. The slower the advent of the disease, the slower is the recovery. There is a distinct suicidal tendency in all cases of melancholia. The risk is less if the degree of retardation is marked, but is greatly increased in the convalescent stage. Never relax observation because the patient says he will not attempt to injure himself; most attempts to commit suicide are of an impulsive nature.

Etiology.—Hereditary factors are prominent and have already been discussed. There is a distinct constitutional basis in the majority of these patients as characterised by their pyknic build: their bodies are round with an abundance of fat, poor

muscular development, a rather broad face on a short neck and small hands and feet. The predominant disposition may be either pessimism or optimism and unbounded confidence. These characteristics do not explain the etiology; much depends on the degree of stress to which an individual, so endowed, is subjected. An important factor is a general depression of the vital powers from bodily disease, *e.g.*, fevers, heart disease, and in particular, influenza. The illness is more frequent in women than men. The first attack of mania develops invariably before thirty, whereas depression is more frequent at or after middle life.

Treatment.—In the simpler cases, such as those referred to under Simple Melancholia, a few weeks' or months' rest under supervision, with a pleasant companion and complete absence of the conditions under which the disease arose, will generally set the patient right. Prolonged narcosis (§ 893) is frequently very beneficial, particularly in the less acute forms. Occupational therapy (§ 905), especially in the convalescent stages, is of value in creating interest and increasing the degree of self-confidence. Feeding is necessary, and in case of refusal it may be done by means of (*a*) a spoon, pouring the fluid into the cheek beside the teeth, or (*b*) by the nasal or stomach tube. The quantity thus administered should consist of one or two pints of milk; to this should be added one or two eggs, a dessertspoonful of sugar and fruit juice. Where artificial feeding is maintained over long periods the feed should be varied and may include soups and meat juices. Sleep must be secured; strychnine tonics may be prescribed before meals. As constipation is usually present, the bowels require attention. Hydrotherapy in the form of the continuous bath is valuable when agitation is marked. Suicide can only be prevented by adequate and constant supervision. If this cannot be obtained at home, then treatment in hospital should be arranged. Convulsive therapy, electrically induced (§ 897), may affect very materially the course of a manic-depressive illness. It influences both phases, though the response in the depressive phase is more easily achieved. The degree of improvement in agitated melancholia is frequently dramatic. Operative measures—prefrontal leucotomy (§ 897)—have been carried out in certain cases where anxiety and agitation have been marked. Beneficial results have been obtained.

§ **896. Hypochondriasis,** though it cannot accurately be isolated as a clear-cut neurotic or mental disorder, frequently gives rise to such a characteristic clinical picture that a description of the condition is warranted. It is a morbid condition of the nervous system which has features common to both melancholia and to some of the neuroses.

The patient suffers from prolonged emotional disturbance interpreted in terms of general malaise and of particular physical symptoms. Although most hypochondriacs may be shown to suffer originally from a depressed reaction to their environment, it is usually found that they also have some underlying physical disability. They refer all their difficulties to this, and in time become intensely preoccupied with the numerous abnormalities which they discover in their state. Thus, while we must presume that most hypochondriacs are badly adjusted to their environment, we can recognise that most of their symptoms are based in the first instance upon very real somatic disturbances arising from such conditions as chronic dyspepsia, visceroptosis and "floating kidney." Very soon the whole lives of such patients are coloured by their ideas regarding the deficiencies of their internal organs. Many of them go from doctor to doctor reciting their symptoms and gleaning from each a few fresh catchwords about their state. Male hypochondriacs appear to be excessively worried most frequently about their sexual functions and fear impotence. Female hypochondriacs are more usually concerned with peritoneal adhesions and "floating kidney." Many such women contrive to be operated upon frequently in order to be relieved from a condition which is basically due to emotional disturbance and maladjustment.

Diagnosis.—It is impossible to enumerate all the symptoms of hypochondria, and it must be understood that a great number of neurasthenics and true melancholics are intensely hypochondriacal. Nevertheless, it can be recognised that there exists

a definite number of patients whose symptoms appear to be purely those described above.

Etiology.—Hypochondriasis is occasionally seen in women, about the menopause, but more often in men of middle age. It is rare before puberty or before thirty, and generally makes its first appearance between thirty and forty. There is often a neurotic family history, including insanity. Digestive disorder (gastro-intestinal or hepatic) is always present, and may be looked upon as its most frequent cause—a fact of interest in connection with the marked prostration and depression which attend gastric and abdominal disorders. Flatulence and dilatation of the stomach are common.

Treatment is difficult unless one has the time to examine thoroughly the patient's mental state, his environmental circumstances, and to discover the reasons for his failure to react adequately towards these. The dyspepsia should be relieved, the bowels should be carefully regulated and otherwise treated. These means, with regular exercises, change of environment and cheerful society, may break through the vicious mental attitude if they are supplemented by reasonable discussions with the patient on his circumstances and his method of adjusting himself to them.

The patient becomes apathetic, withdraws from outside interests, day-dreams continuously or develops delusional ideas. The disease is SCHIZOPHRENIA.

§ **897.** III. **Schizophrenia** (Syn.: Dementia Præcox). This form of mental illness is characterised by an abnormal emotional reaction, accompanied by varying degrees of apparent deterioration in the personality. It has been reported in early life, but is more frequently met with between the ages of fifteen and thirty-five. See § 907 B for symptoms in adolescence. There is frequently a family history of nervous or mental disorders, and the patient may show one or more stigmata of degeneration, *e.g.*, deformities of the ears. The onset is invariably insidious and no adequate cause can be demonstrated. The previous personality is that of a reserved, unsociable individual of few interests. Four types are found. (1) *Simple:* The characteristic feature is the gradual loss of interest in the environment. As time goes on such patients withdraw further from reality and substitute a world of phantasy, showing itself in indifference and apathy. Delusional ideas and hallucinatory experiences are infrequent and many of these patients lead a simple life outside hospital care. (2) *Hebephrenic:* Here delusions and hallucinations are prominent. The oddity of the delusional ideas—that half of their body has ceased to function, that their blood has gone—and the indifference with which they recite such, are characteristic. Hallucinations are both auditory and visual. Attacks of depression alternating with acute outbursts of excitement occur in the course of the illness. During the latter suicidal or homicidal attacks may be made. Other symptoms frequently seen are echopraxia and echolalia in which the actions or words of bystanders are imitated, although questions may not be replied to. (3) *Katatonic:* The progress of the illness is more rapid in this form. Extreme affections of volition are found and vary from outbursts of excitement to depression and a stage of stupor. In the stuporose state no interest is displayed in anything. The patients sit in one position; if the limbs are placed in an awkward position they will remain there for an indefinite period (flexibilitas cerea). Such patients have to be dressed and undressed and require attention in all respects. They pay no heed to the calls of nature, and may require to be tube fed for long periods. Although indifferent and apparently insensitive they are able to appreciate nevertheless what is going on in the environment. From this state they will pass into one of excitement, possibly without warning. Impulsive attacks, homicidal or suicidal in nature, are frequent and must be guarded against. (4) The *Paranoid* form is characterised by prominent delusional ideas. They are not systematised as in paranoia, are much more bizarre in nature and are accompanied by hallucinatory experiences. It is more common to meet this type after thirty years of age than before.

Etiology.—Hereditary and constitutional factors are again conspicuous. In stature these patients are thin, long-limbed and of poor physique. Their mental make-up is that of a reserved, shy individual with few interests and friends. The symptoms may not become manifest till after some debilitating illness or period of mental stress.

Course and Prognosis.—In the main the outlook is poor. Such patients constitute 40 per cent. of the chronic cases in mental hospitals. Remissions occur of comparative or complete return to health; in others the degree of improvement is less marked. Of the clinical types the katatonic variety is the most favourable. A sudden onset, a previously good personality and an adequate cause, when found, make the prognosis less ominous.

Treatment.—When the illness has developed, then hospital treatment is advisable for the safety of the patient and the community. Excitement is delayed by hydrotherapy and sedative measures. Efforts must be directed to prevent further deterioration in habits. In this respect occupational therapy, properly directed, is of the greatest value. Various special forms of therapy have been advocated at different times, by the induction of fever (malaria, pyrifer), vaccines, polyglandular extracts, and continued narcosis. The variety of such testifies to their inefficacy.

In recent years active therapeutic measures have included (*a*) electrically induced convulsions, (*b*) hypoglycæmia, (*c*) operation—prefrontal leucotomy. The first two require the closest supervision and should be given only by experts. A combination of both methods is sometimes used, the convulsion being induced after a light degree of hypoglycæmic coma has been attained. Convulsive therapy is more effective where the affective disturbance is marked and hypoglycæmia where there are gross delusional ideas. Early treatment gives a much better therapeutic result. *Electrically induced convulsions* have superseded those produced by Cardiazol as being less unpleasant and not accompanied by anxiety: by means of electrodes placed on the head, a small electrical current is passed through the brain (100–150 volts for 0·2 sec.) and a convulsion follows at once, with tonic and clonic phases, the period of unconsciousness lasting a few minutes. These are induced at three-day intervals; improvement is unlikely if it is not manifested after 10 to 15 treatments. Complications that may arise include fractures of bones and dislocations of joints, but the incidence of these has been over-emphasised. They result from muscular spasm and the dangers may be materially diminished if the treatment is combined with curare. In treatment by *Hypoglycæmia,* the aim is to produce a severe reaction by the introduction of insulin. The average dose of insulin to produce coma appears to be 60–80 units, but over 200 units have been necessary in some cases. The coma may be allowed to last for 1½ hours. It is terminated by a nasal feed of 33% glucose, or, if necessary, the giving of intravenous glucose. This form of treatment is given on successive days with one day's rest a week, and the course of treatment consists of 60 coma doses. This method is much more dangerous than the former; in both, the presence of physical disease is a contraindication. (*c*) Prefrontal leucotomy is a surgical procedure. The prefrontal area is exposed and association fibres are severed. The method as at present practised is unscientific but there is no doubt that certain patients are considerably improved.

All the thinking processes of the patient are permeated by an idea of persecution. Such an idea may be logical. The disease is PARANOIA.

§ 898. IV. **Paranoia** is the term used for a variety of mental illness in which the patient's whole mental life is dominated by a delusion—usually one of persecution. Disorder of judgment is the characteristic feature, and in consequence the patient interprets every incident which he observes or takes part in as fresh proof of a plot against him. There are two classes of paranoiacs. In the first, which is of a milder character and rarely needs care in a mental hospital, the patient's own personality does not take any part in the delusion, but he is possessed by some wild theory which

he preaches in and out of season; in the second class, which is a grave form of mental illness, the patient's own personality is all-important, and delusions of persecution are common. This delusion is liable to lead the patient to assassination of some prominent person or even to attempt suicide in order to call attention to his case. Megalomania is apt to develop as the disease progresses. Hypochondriasis, in which the patient's attention is focussed on his health or lack of it, is sometimes a sub-variety of paranoia, but does not lead to any disorder of conduct likely to cause harm to the community. *Folie à deux* is a condition in which one patient, usually a paranoiac, persuades another with whom he or she is very intimate of the reality of the supposed plot against their lives or characters. The second patient, sometimes called the passive element, though mentally ill, is more likely to recover. In true paranoia there is no recovery, although occasionally a remission may occur. When the paranoiac disorder is dominated by active hallucinations the condition is called **Paraphrenia**, and mostly arises in patients past middle life.

V. *The patient is addicted to alcohol or a drug, or the brain is affected by a toxæmia. The disease is* ALCOHOLISM, DRUG HABIT *or* DELIRIUM.

§ **899. Addiction to alcohol,** opium or other drugs, may be symptomatic of an anxiety neurosis, obsessive-compulsive neurosis, psychopathic personality, manic-depressive psychosis or general paralysis. The continual abuse of the drug leads to a gradual deterioration of the personality, although this seldom advances to the stage of certifiable mental disease. Alcoholism or excessive indulgence in alcohol is met with clinically in five forms—(1) Acute alcoholism, (2) Chronic alcoholism, (3) Dipsomania, (4) Delirium Tremens, (5) Korsakoff's Psychosis.

(1) **Acute Alcoholism** is due to an excessive quantity taken in a few hours. It gives rise to mental disturbance, ataxia, and even a temporary flaccid paresis of the limbs. Later, narcosis with a marked lowering of body temperature may develop. From a medical point of view the effects of an acute alcoholic debauch are so transient as to be of no importance except in medico-legal cases. Acute drunkenness must not be confused with diseases causing cerebellar or sensory ataxia (see p. 1006) or Ménière's Disease (see § 692, vertigo). Certain neurotics are abnormally sensitive to small doses of alcohol, and, for months after severe head injuries, even small doses of alcohol may produce intoxication. The stupor of concussion, apoplexy, uræmia, opium poisoning, etc. (§ 716), and the muttering delirium (§ 469) of pneumonia and other diseases, may be mistaken for drunkenness, a serious error which is best avoided *by keeping the patient under observation in bed* and suspending your judgment.

(2) **Chronic Alcoholism** is due to the persistent imbibition of moderate doses of alcohol over a long period. It acts as a poison on the nervous, muscular (voluntary and involuntary) and epithelial elements, and hinders tissue oxidation, thus leading to fatty degeneration.

Symptoms.—(i.) The patient is able to take doses of alcohol which, in the normal person, would produce drunkenness. (ii.) Irritability, even violence, and progressive deterioration of personality appear, especially when the patient is amongst his family circle. (iii.) Carelessness and lack of concentration become evident in the patient's work, and (iv.) he strongly resents any suggestion that these failings are due to alcohol.

(v.) Later, *other mental symptoms* appear, such as alternating depression or excitement, unfounded suspicions or delusions of persecution; the patient ceases to speak the truth and dementia gradually develops. Delirium tremens (see below) supervenes from time to time, and sometimes epileptiform convulsions. (vi.) The physical symptoms are: (*a*) gastric catarrh with anorexia for food and morning vomiting, (*b*) gastric dilatation, (*c*) hepatic cirrhosis, and (*d*) myocardial and arterial degeneration. (*e*) There is often obesity and plethora, or the gastritis or cirrhosis leads to marked loss of flesh. (*f*) Coarse tremors of the hands, tongue and lips, with tremulous dysarthria. (*g*) The kidneys may show interstitial nephritis. (*h*) Polyneuritis may occur (§ 794).

Secret drinking is the term applied to chronic alcoholism occurring in persons who are thought to be "above suspicion." It occurs especially in women about the menopause, who commonly secrete empty bottles in the wardrobe, under the bed, etc.

(3) **Dipsomania** or paroxysmal drinking is frequently symptomatic of a psychopathic personality or of manic-depressive psychosis. It is also found sometimes in epileptics. The condition is often hereditary and may develop in middle life. Between the paroxysms the patient may be quite normal with no desire for alcohol, or even distaste for it. Then comes depression and an uncontrollable craving for alcohol. The attacks may show a definite periodicity.

(4) **Delirium Tremens** arises in chronic alcoholics: (1) after a debauch, (2) with pneumonia or other acute infection, or (3) after sudden withdrawal of alcohol, *e.g.*, when the patient is sent to hospital for treatment of a fracture. The *symptoms* are: (1) restlessness and complete insomnia, (2) terrifying visual hallucinations of animals, especially insects, spiders, rats, snakes (zoöpsia), producing intense fear and impulsive outbursts, (3) occupational delirium with disorientation in space and time, and (4) coarse tremors of the fingers, face and tongue. (5) The temperature is usually slightly raised, and (6) furring of the tongue, dryness of the mouth and anorexia are usual. A first attack will last three to five days, the patient waking to consciousness with amnesia for his delirious period. Second attacks may last two or three weeks.

The *Diagnosis* of delirium tremens is referred to in § 469. The history of alcoholism and the type of hallucinosis are of value but care should be taken not to overlook an acute pneumonia, especially of the apex. The *Prognosis* of delirium tremens is generally favourable if the temperature is not much elevated, and the strength of the patient can be maintained. Second and third attacks are commonly longer in duration and may leave residual mental impairment.

(5) **Korsakoff's Psychosis** occurs most frequently in chronic alcoholics past middle life, but may follow an attack of delirium tremens. It is met with in other toxic conditions also and is frequently seen after a severe head injury. It affects women more frequently than men and is invariably accompanied by neuritis.

Symptoms.—(1) Memory defects are characteristic. There is a gross impairment for recent events; the gaps are made up by confabulations, frequently of a most

plausible nature; (2) disorientation in space and time; (3) auditory and visual hallucinations, (4) moods fluctuate rapidly between one of euphoria and one of anger and irritability, and (5) signs of polyneuritis. The prognosis is frequently not good, some impairment of memory and of the intellectual faculties persists. The duration of the illness extends over months.

The *Treatment* of *acute alcoholism* consists in washing out the stomach or giving an emetic, *e.g.*, apomorphine, grain $\frac{1}{6}$ hypodermically. The collapse is treated with enemata of hot coffee, warm blankets and hot water bottles, care being taken that the patient is not burned before he recovers consciousness. Treatment of the *chronic* types is impossible unless the patient co-operates. A chronic alcoholic habit is rarely abandoned after forty. Institutional treatment, where one can be satisfied that the patient will not obtain alcohol, is the only method of ensuring abstinence. The method of withdrawal may be rapid or delayed. Various forms of treatment have been advocated; none are completely satisfactory. Strychnine and atropine sulphate (hypodermically) $\frac{1}{60}$ grain and $\frac{1}{120}$ grain respectively may be given with cinchona bark (by the mouth) four times daily, until the throat is dry and the pupils dilated. At first, night and day nurses will be necessary. Sleeplessness should never be treated by morphine, which may be cumulative in its effect in chronic alcoholics with hepatic cirrhosis. Paraldehyde ♏ 120–240 per rectum, or chloral gr. 15 and sodium bromide gr. 15, by mouth, may be given safely, with hot packs. Massage and careful feeding improve the general condition. Patients should be encouraged to remain under institutional treatment for some months, during which period the psychological factors must be investigated and treated, so that the patient may endeavour subsequently to face his problems without retreating to alcohol. Regular outdoor exercise and extraneous interests are encouraged. There must be complete abstinence for the rest of the patient's life. The readjustment of any domestic or business difficulties is essential and the question of the patient's return to his former occupation must be carefully considered. If he returns to the circumstances of his previous breakdown recurrence of the habit is probable.

In *delirium tremens*, alcohol may require to be given in the early stages and gradually tapered off. The main objectives in treatment are to improve the physical state, control the restlessness and obtain sleep. Small allowances of liquid nourishment at frequent intervals are necessary to maintain strength. Such patients frequently endeavour to get away from their hallucinatory experiences and in so doing may be a danger to themselves. The closest degree of observation and tactful management are therefore essential and must be insisted upon (two or even three nurses if necessary). Beneficial results have been claimed from frequent lumbar puncture and drainage of the cerebrospinal fluid. Sleep is essential and great variations will be found in the response to sedatives. It may be necessary therefore to change these at intervals. Paraldehyde per rectum, or chloral and bromide, or nembutal four and

a half grains by mouth or hyoscine hydrobromide gr. $\frac{1}{100}$ hypodermically may be tried with hot packs to secure sleep. An attack of *dipsomania* may be averted if premonitory symptoms are present, by injections of apomorphine, grain $\frac{1}{8}$, hypodermically, to produce nausea or vomiting. The gastric catarrh of alcoholism may be treated by tincture of capsicum ♏ 5, sod. bicarb. gr. 10, in half an ounce of infusion of gentian.

§ **900. Morphinism** (Synonyms : Morphia Habit, Morphinomania) and other **drug habits.**—Hypodermically, **morphia** in small doses is a nerve stimulant as well as a hypnotic, and induces a feeling of contentment and well-being ; but in the course of twenty-four hours reaction and craving for more occur, particularly when pain is present, and by degrees the dose has to be increased until in the course of a few months twenty to one hundred times the normal dose is necessary to produce a feeling of satisfaction, and can be easily tolerated. The only *signs* by which the *morphine habitués* can be detected are contracted pupils, pallor of the face, and the frequency with which they withdraw to satisfy their craving —a difference being observed in their depression before and their gaiety and brightness afterwards.

If such a patient is suddenly deprived of the drug, the following *withdrawal symptoms* set in. The pulse, which was previously normal, becomes rapid and of low tension, and the patient prostrate, suffering agonies from tingling in the limbs, sweatings, sneezings, lachrymation, diarrhœa, vomiting, uncontrollable restlessness, faintings, sinkings in the pit of the stomach, extreme wakefulness, and a host of horrible and indescribable somatic sensations resembling extreme neurasthenia.

Consequences of the morphia habit. Enormous doses may be taken by gradual increase. At first the patient is always gay, and has great capacity for mental and bodily endurance. But if the habit be continued, the character gradually becomes altered. The patient alienates his friends by tempers and unreliability ; and, one by one, truth, reverence, and honesty disappear. If there be difficulty in procuring the drug, great craftiness is exhibited. In course of time the mental powers gradually deteriorate, and suicide is not infrequent in those who desire, but are unable, to rid themselves of the thraldom. The body also suffers, and the patients become pale and emaciated. They get careless in the use of their syringe, multiple abscesses form and death may result from septicæmia.

Prognosis.—Since most morphine habitués are psychopathic the prognosis is always serious. It is worst in doctors, dentists, chemists and nurses, who have easy access to the drug. In these, as in all cases, relapse is common, and the permanence of cure depends on continued supervision and the possibility of the patient being able to lead a sheltered life, free from all care. After a cure there may be a tendency towards alcoholism. The morphine habit probably shortens life, and death may occur from over-dosage. A habit of short duration is easier to cure than one of long duration : the actual quantity of morphia taken per diem is of little account. If carcinoma, or some other cause of an incurable and recurrent

pain be present, and especially if the patient be aged, the tendency to relapse is great and it may be impossible to ease the pain in any other way.

Treatment.—(*a*) To break the habit the patient must be willing to place himself in a Home or institution, and in bed. The closest observation and supervision is necessary to ensure that the dose of morphia during the withdrawal period is controlled. Unless this is accomplished, methods of deception will be practised that nullify all efforts. The drug may be withdrawn abruptly, or gradually over 7 to 10 days. The former method is accompanied by intense suffering and should not be practised in elderly or debilitated patients. Various methods of treatment have been devised to alleviate the symptoms. Hyoscine hydrobromide gr. $\frac{1}{100}$ is given hypodermically every hour until delirium supervenes, and after that hyoscine hydrobromide gr. $\frac{1}{200}$ is given hourly, or at such intervals as will maintain delirium for two or more days. Several such deliria may have to be induced before the patient loses the craving. In the intervals between the deliria careful supervision and feeding will be necessary, and care must be taken that no morphine is secreted by the patient or brought into the sick-room. Night and day nurses will be necessary. (*b*) The withdrawal symptoms are treated by moderate doses of sal volatile, strychnine sulphate gr. $\frac{1}{60}$, hypodermically, and *gradually tapering off the morphine.* For the vomiting and diarrhœa give bismuth, and treat sleeplessness with paraldehyde ♏ 120 to 240 per rectum, or chloral and bromide or one of the barbiturates by mouth; prolonged baths are very beneficial. (*c*) When the convalescent stage has been reached psychotherapeutic treatment may be employed, thereby to help the patient achieve a better method of solution of his emotional problems and difficulties. Prolonged supervision and care is essential during which period occupational therapy is valuable. The patient should not return to an occupation or environment where it is easy to obtain the drug.

A **heroin** habit can cause as serious symptoms as a morphia habit.

The **cocaine habit** leads to many of the troubles of the morphia habit, but there is a greater tendency to mental symptoms and deterioration. Morphia and cocaine are often taken together; in such cases the cocaine may, with comparative ease, be first withdrawn. Then the morphia can be reduced as above described.

The **chloral habit** is less common nowadays. It gives rise to gastro-intestinal disturbance, lowered nutrition, pains, skin eruptions, depression, irritability, palpitation, and cardiac weakness. Sudden death may occur from slight increase of the dose.

Sulphonal, phenacetin, antipyrin, and other tar products do not so readily engender a craving, but when habitually used the patient cannot do without them, and in course of time symptoms similar to those of the chloral habit arise.

The psychosis is associated with a general infection; the condition is DELIRIUM.

§ 901. The causes of **delirium** were considered in § 469, and need only be enumerated here.

Clinical Investigation.—The first and most important point in any given case of

delirium or mental excitement to which you may be called for the first time is to ascertain the temperature. Secondly, it is important to make a thorough and complete investigation of all the organs of the body, to ascertain whether there be any local inflammatory disorder, such as pneumonia, with which delirium may be connected, either directly or indirectly. The urine also should be carefully examined for albumen, sugar, or other abnormality. Thirdly, enquiry should be made into the history of the malady and of the patient, especially as regards the consumption of alcohol and sedative drugs, particularly bromides. The latter are cumulative, and patients vary greatly in their degree of susceptibility, more particularly if arterio-sclerotic changes are present. The bromide replaces the chloride ion in the blood plasma and quantitative estimations of the blood bromide can be easily made. In reference to the etiology of delirium, three important *predisposing causes* have to be borne in mind. First, there is a marked predisposition in some nervous people to develop delirium with a slighter cause than would affect others. Secondly, there is a marked hereditary tendency towards the same vulnerability; and thirdly, excessive drinking predisposes to the occurrence of delirium after an injury, operation and many diseases which are not usually so attended.

Febrile.	*Non-Febrile.*
Diseases of the brain — especially meningitis and dementia paralytica.	Delirium tremens.
Acute visceral inflammations—*e.g.*, pneumonia, pericarditis, pyelitis.	Chronic renal disease.
Acute specific fevers.	Post-epileptic delirium.
Delirium tremens (rare cases).	Cardiac failure.
	Drugs—*e.g.*, bromide, medinal, hyoscine.

The clinical form is not dependent on the type of infection and the reaction to the same toxin may vary in different individuals. On the other hand, such physical illness may release a latent mental illness of the schizophrenic or manic-depressive varieties. Hence the variety of symptoms seen after childbirth and the variations in ultimate outcome.

Symptoms.—During fever : (i.) the commonest clinical picture is that of a delirium characterised by confusion, (ii.) disorientation for time and space, (iii.) illusions develop, followed by (iv.) hallucinations of sight and hearing. The hallucinatory experiences are very vivid and arouse great fear and restlessness, (v.) transient delusional ideas of a persecutory nature. *After fever :* (i.) exhaustion and great fatigue, (ii.) varying degree of depression, especially common after influenza, and risk of suicide, (iii.) an amnesia for the acute stages; the greater the degree of confusion the more complete the amnesia. A neurasthenic condition may form the basis of more ominous psychotic illness.

A similar mental picture may be seen following *severe physical stress, pregnancy, parturition* or *severe hæmorrhage.* Mental abnormalities after *parturition* occur most frequently in those who have had a febrile reaction after labour. There is no clinical entity characteristic of this type as was formerly described.

In *cerebral syphilis* the confusional picture is typical of any delirium, and there is a marked loss of memory for recent events. Paroxysmal headaches, sleeplessness and symptoms indicative of transient involvement of the cranial nerves, *e.g.*, squint, ptosis, dimness of vision, are common. Serological investigation will confirm the diagnosis.

The *prognosis* is good if the patient recovers from the physical illness. Those who subsequently develop a schizophrenic illness have displayed abnormal personality changes previous to the physical upset.

Treatment is directed to the primary physical condition; otherwise it is symptomatic, and the objective is to secure adequate rest and nourishment. If the confusional state is due to bromide intoxication the drug should be omitted and the intake of sodium chloride greatly augmented. A period of some weeks may be necessary before the bromide is eliminated.

VI. *There is progressive mental deterioration associated with* ORGANIC DISEASE *or* EPILEPSY. *The disease is* GENERAL PARALYSIS OF THE INSANE, *or* DEMENTIA *due to* ARTERIOSCLEROSIS, ALCOHOLISM (§ 899), PRE-SENILE CHANGES *or* EPILEPSY.

§ **902. General Paralysis of the Insane** (G.P.I. ; Paralytic Dementia) is characterised by progressive muscular weakness and tremor, accompanied by mental symptoms, often of a grandiose character, occurring most frequently in young men, or men in the prime of life. The treponema pallidum can be demonstrated in the cortex of the brain and in the subcortical tissues, at some distance from the blood-vessels. The disease results from the parenchymatous infiltration and destruction caused by this organism.

Symptoms.—Paralysis of the limbs may sometimes exist for many years without mental symptoms (*vide infra*). The characteristic symptoms and signs are changes in the personality, impairment of memory, delusional ideas, tremors, pupillary changes, speech defects, and finally convulsions and generalised weakness. Invariably mental deterioration is the earliest symptom ; in some objective neurological signs, *e.g.*, Argyll-Robertson pupils, optic atrophy, or slurring articulation, are prominent from the first. The course of the illness has been regarded as showing three stages, but these are not always clearly defined, more particularly since the introduction of intensive treatment.

Early Mental Changes.—The earliest clinical manifestations are increased irritability with or without headaches, irrational behaviour and insidious changes of character. The insidious onset often confuses the diagnosis. Impairment of the power of attention develops, so that activities requiring any degree of mental concentration are evaded. Memory begins to fail, particularly for recent events, though this defect may escape recognition unless looked for. Impairment of judgment develops, and with this, conduct becomes more grossly involved. Irresponsible decisions are made and wild speculations may result. Indifference is apparent and the patient becomes careless about his personal appearance. Delusional ideas are prominent in the classical *euphoric variety*. These are of a grandiose type and generally accompanied by much overactivity. The patient believes himself to be all-powerful, of royal descent, or exceedingly wealthy. The last-mentioned idea may lead to his squandering his resources and delay in recognition may leave his family in penury. This type is less frequent than formerly described, and the most usual features now encountered are those of a *simple progressive deterioration*. Sometimes *great depression*, sullenness and loss of energy are predominant.

Various *Physical Changes* accompany, precede or follow the mental symptoms. The most common are: (i.) tremors (fine, small and rhythmical) of the face, also of the hands (giving rise to characteristic writing), and coarse tremors of the tongue (giving rise to a characteristic slurring of the speech) ; (ii.) the pupils in this stage are usually small,

unequal and irregular in outline. They fail to react to light, but react on accommodation (Argyll-Robertson pupil); (iii.) primary optic atrophy is not uncommon; (iv.) the tendon reflexes are invariably increased and the plantar reflexes may be extensor in type. Sometimes symptoms of tabes are present in addition (tabo-paresis).

Serological changes: The blood Wassermann reaction is positive. Examination of the cerebro-spinal fluid shows various pathological changes. The Wassermann reaction is positive in 99 per cent. of cases; the cell count is increased and may be as high as 400 per cu.mm. (lymphocytes). The globulin and total protein are increased and the discoloration of the test tubes in the Colloidal Gold reaction (Table LXI) is characteristic (paretic curve).

Later stages of the illness are characterised by (i.) mental enfeeblement, which replaces the mental changes in the first stage; (ii.) increasing muscular weakness, difficulty in walking any distance, and especially in the act of turning, sometimes combined with giddiness; (iii.) fits (congestive attacks) are almost invariably present at some period of the illness; they vary in character, but are usually syncopal or epileptiform, with or without the loss of consciousness. Sometimes they consist of attacks of numbness of the limbs, or aphasia, or coma. They may occur in the early stages and may constitute the initial symptom. The *final stage* is that of progressive dementia. The speech becomes inarticulate, the paralysis extreme, and may be accompanied by contractures, so that the patient cannot feed himself. His mind undergoes progressive extinction, and there is loss of all its faculties. The urine and fæces are passed involuntarily.

Several varieties may be differentiated:—(1) The Expansive manic variety forms the basis of the above description. It is seen less frequently than formerly reported. G.P.I. should always be considered as the cause of a first attack of excitement in a patient over 30 years of age. (2) The Depressive variety presents a picture difficult to distinguish clinically from melancholia. The impairment of memory, the presence of physical signs and the serological findings differentiate the two conditions. (3) The Simple variety is characterised by childishness, apathy and indifference. Such patients are fatuous and express no gross delusions. The process is a simple progressive deterioration. (4) The Tabo-paretic variety includes those who show physical signs of tabes in addition to mental changes. (5) A Juvenile variety, occurring up to the early twenties, due to congenital syphilis (see § 907c).

Diagnosis.—On account of the great variety of symptoms presented by G.P.I., its diagnosis may be difficult. It is distinguished from (*a*) other forms of *mental disorder*, especially *chronic alcoholic psychosis* and *presenile dementia*, chiefly by the tremor, speech, the pupillary changes, and the spinal fluid findings; (*b*) maladies attended by tremors and other neuro-muscular symptoms, such as *disseminated sclerosis*, *pseudo-bulbar paralysis* and *paralysis agitans*. Chronic *alcoholism* and *polyneuritis* sometimes are difficult to differentiate; they are recognised by examination of the spinal fluid. *Cerebral arteriosclerosis* is associated with retinal changes and often a raised blood pressure. *Lumbar puncture is essential before an absolutely accurate diagnosis of G.P.I. can be arrived at.* The diagnosis from *tabes dorsalis* is not usually difficult.

Etiology.—Adult males, in the very prime of their strength and manhood—that is, between thirty and forty—are the favourite subjects of the disease, but it may occur at any age. It develops 10–12 years after infection, and is generally said to be three or four times more common in men. The disease is a syphilitic inflammation and degeneration of the nerve elements and blood-vessels. The skull and meninges are thickened. The cerebral convolutions are shrunken and the sulci widened, especially in the frontal lobes. Spirochætes are present throughout the brain. The vessels show syphilitic endarteritis and infiltration of the perivascular spaces with lymphocytes and plasma cells. Alcoholic, sexual, and other excesses, anxiety, and mental fatigue may be accessory causes.

Course and Prognosis.—The duration of untreated cases varies from a few months to four years. The earlier the onset of the disease after the primary infection, the more rapidly progressive will it be. Intermissions of comparative or complete return to health are characteristic of the disease. It is rare for the duration of such remissions to exceed two years. Consequently all such patients should be given pyrexial treatment unless there are contra-indications in the physical state. The prognosis in treated cases is dependent on (*a*) the duration of symptoms prior to treatment, (*b*) the form of the illness, (*c*) the age of the patient. It is imperative to make an early diagnosis as the chances of a successful outcome after treatment diminish in direct ratio to the duration of the symptoms prior to treatment. This probably accounts for the fact that the expansive type responds better to treatment than the others, as such symptoms early attract attention.

Treatment.—Antisyphilitic measures at an early stage of infection may prevent the onset of General Paralysis of the Insane. Treatment with the usual arsenicals and antisyphilitic drugs is of no avail in the fully developed disease. Tryparsamide, a pentavalent arsenical compound, penetrates the parenchyma of the nervous system, and on this account is the most useful, but in rare cases has caused optic atrophy. Penicillin has been given in doses of 8 million units or more in periods of 7 to 14 days. The results are more favourable in meningeal than parenchymatous neurosyphilis. At present a combination of penicillin and fever therapy is practised. The febrile reaction may be produced by (1) malarial inoculation, (2) chemical means or (3) diathermy. Diathermy requires special electrical apparatus and is not without risk. Malarial inoculation is most frequently practised in this country, and may be effected by mosquitoes or by malarial-infected blood (provided by the Ministry of Health). Mosquitoes are contained in a jar protected at the opening by a netting ; this opened end is placed on the patient's skin which must be perfectly clean and free from soap, etc. Defibrinated malarial blood injections may be subcutaneous, intramuscular or intravenous. The needle and syringe are sterilised by boiling and allowed to cool, as heat and antiseptics destroy the parasite. The incubation period varies from one to thirty days or more. The rigors last from a few minutes to two and a half hours. In uncomplicated

cases, after eight bouts of fever, the fever is terminated by administering 10 grs. of sulphate of quinine given twice daily for at least ten days. Treatment by Malaria is accompanied by definite risks which must be fully appreciated. It should be employed only when the patient is not over 55, and the general condition is satisfactory. Contra-indications are the presence of other infections, or diseases of the gall-bladder, liver or spleen, organic disease of the heart or aorta, and obesity. During treatment, daily blood-pressure readings are necessary to exclude hypotension; blood counts and films must be made twice weekly, and the urine examined daily for albumen and bile. The fever should be terminated prematurely if there develops: (i.) sudden loss of strength, (ii.) evidence of cardiac weakness, as shown by an increase in the pulse rate over 150 or the development of irregularity in rhythm, (iii.) a temperature above 106° which does not respond to tepid sponging, (iv.) toxic or infective jaundice, (v.) a marked increase in the number of parasites in the blood film taken during a rigor, (vi.) continuous vomiting that does not respond to treatment, (vii.) anæmia with the red cell count under 2 million, (viii.) pulmonary complications, (ix.) occurrence of seizures. Death may ensue from progressive exhaustion or anæmia, coronary thrombosis, toxic jaundice or uræmia. Provided such treatment is used in the early stages, patients are able to return to work and to continue at it for years. They should subsequently live a regular life, with outdoor exercise and amusements, and endeavour to avoid all causes of anxiety or mental strain.

§ 903. **Arterio-sclerotic Dementia.**—The characteristic mental symptoms are impairment of recent memory, restlessness and mental deterioration. There is a gradual but progressive failure of the intellectual faculties, invariably accompanied by increased fatiguability. Inability to make decisions or to give prolonged attention to any problem soon becomes apparent, followed by failure to recall names or places. These symptoms are greatly aggravated by epileptiform or confusional attacks. Memory defects become obvious, at first for recent events, and there is a tendency for such gaps to be made up by fabrication. Emotional control is impaired; sudden outbursts are common over failure to perform what was formerly a simple task. A suspicious attitude is frequent and paranoid delusional ideas may be prominent. In some cases sexual urges are not controlled, and this may lead to misdemeanours. The condition progresses to dementia.

Dementia is seen as a *primary* condition in (*a*) advanced life (senile dementia) and (*b*) chronic alcoholism. It comes on as a *secondary* condition in (*a*) untreated general paralysis of the insane, and as the concluding stage in other forms of mental disease, notably chronic schizophrenia; and (*b*) after vascular and other gross intracranial lesions, especially in the frontal cortex. Even after a small lesion of the brain the mental capacity for business is hardly ever as good as before its occurrence, and the patient often becomes childish, peevish, forgetful, emotional, and by degrees in severe cases completely demented.

Pre-Senile Dementia occurs at an earlier age, between 40 and 60 years, and runs a more rapid course. Two varieties have been described, by Alzheimer and by Pick, more easily differentiated pathologically than clinically. Extensive degeneration of the grey matter occurs chiefly in the frontal lobes; plaques being found in Alzheimer's disease but not in Pick's disease. Progressive memory impairment, aphasic disturbances and epileptiform attacks are the prominent features.

Epileptic Dementia.—About 10 per cent. of epileptics become so far unmanageable

as to be regarded as insane. The mental aberration may be (1) pre-paroxysmal, (2) post-paroxysmal, (3) associated with petit mal only or as an epileptic equivalent. Such symptoms are invariably those of excitement, confusion, delirium, stupor, or a general mental deterioration.

§ **904. Prognosis** of mental illness in general.—The *Course* and *Prognosis* in several of the various forms of mental disorders have been referred to. In general terms the chief points on which the prospect of recovery depends are (1) the absence of heredity, especially direct heredity; (2) the personality and make-up of the individual; (3) the presence of an adequate cause; (4) the rate of onset of the attack, being more favourable in a rapid than a slow, insidious onset; (5) the duration of the illness prior to treatment; and (6) the clinical form of the illness.

Under the Matrimonial Causes Act, 1937, mental defect and unsoundness of mind are grounds of petition for divorce. It must be shown that the person concerned is incurably of unsound mind and has been continuously under care and treatment under certificate for a period of at least five years immediately preceding the presentation of the petition.

§ **905. The Treatment of Mental Illness** in detail has been referred to under the different forms; the general principles resolve themselves into (1) Physical methods, (2) Psychological methods, (3) Occupational therapy, and (4) Social factors.

PHYSICAL METHODS include the control of excitement, insomnia, the prevention of self-injury, adequate feeding, prolonged narcosis, hydrotherapy, endocrine preparations, and the treatment of any physical defect discoverable. They also include such specific methods as malarial or convulsive therapy and hypoglycæmic therapy.

PSYCHOLOGICAL METHODS are employed by the physician in everyday practice. The results depend in large measure on the degree of rapport between patient and physician, the influence of the latter being a dominant factor. They assume a belief by the patient that the illness can be cured. *Reassurance, persuasion, suggestion* and *analytic methods* are employed.

Reassurance: Often a free discussion with the patient of his symptoms and his problems, with an explanation as to their development and a reassurance as to their significance, will effect considerable improvement.

Persuasion: Here the aim is to convince the patient of the absence of any organic basis for his symptoms and such to be effective is accompanied by emotional force. No attempt is made to treat the cause of the symptoms; consequently a recurrence in a fresh site is not uncommon.

Suggestion is a process of implanting ideas of a corrective nature; by this means mental improvement ensues. The impressions desired to be made on the mind may be implanted when the patient is awake or in a drowsy hypnotic state. Many believe that suggestions are reinforced by *hypnosis,* which may be defined as a condition of partial consciousness resembling sleep, in which the subject's capacity to receive and to act upon suggestions is greatly improved. This increased suggestibility is made use of by the operator for the implanting of new and healthy conceptions and the removal of morbid ideas, the object being to influence the body through the mind. Only by trial can one determine whether a person is able to be hypnotised. Various methods of inducing hypnosis

are available and details of these may be obtained in text-books on the subject. The method should be used only by medical men, and with proper precautions. The consent of the patient and his friends should be obtained ; often a third person should be present during the treatment. In competent hands no bad effects result from its employment even over prolonged periods, but much moral and physical evil follow the abuse or misuse of this powerful agent. Its use for purposes of public exhibition should be forbidden by law. Hypnotism has been employed to restore memory in cases of hysterical amnesia, to reform alcoholics and moral perverts, to cure various neuroses, and to relieve various hysterical manifestations such as anæsthesiæ or paralyses.

Psycho-analysis is a method of investigation of the unconscious mind which has been advocated by Freud and modified subsequently by his pupils. It consists of a minute study of the patient's previous life by special methods—dream analysis, hypnosis, and free association. The patient is requested to state every thought and word that casually occur to him whilst under examination, in the hope of discovering some hidden psychic trauma of early life. Freud emphasises unduly the sexual content of the unconscious mind: he considers that dreams have definite symbolic meaning requiring special interpretation, and that complexes are discovered by their elucidation. Jung has extended the method by "word-associations," using 100 selected stimulus words and observing the character and time of the words of response. Any delay in reaction as shown by a stop-watch indicates that a repressed complex has been affected which when brought to consciousness and fully explained assists in curing the patient. The results of analytic treatment are difficult to assess, but the method is more successful in psychoneurotic than psychotic forms of illness. Psycho-analysis is not employed in patients past fifty years of age or those with organic disease. It requires a certain degree of intelligence and ability to co-operate on the part of the patient and its application is limited owing to the time and expense involved—many cases requiring an hour's sitting daily over a period of months. Although possessing therapeutic value in some cases otherwise intractable, psycho-analysis occasionally upsets patients and does harm. It is therefore best left in the hands of experts of acknowledged experience and repute.

Occupational Therapy has been defined as the treatment under medical care of physical or mental disorders by the application of occupation and recreation with the object of promoting recovery, of creating new habits and of preventing deterioration. The value of work in health is generally conceded, and much time and energy is now spent in directing healthy adolescents into their appropriate sphere of activity. In the treatment of the sick occupational therapy includes more than mere occupation. It should find expression also in the social and recreational outlets of the hospital. Occupation should consist of much more than the mere doing of something "diversional"; being busy is not necessarily therapeutic. When properly applied the method arouses interest

and the successful completion of some form of work naturally helps towards self-confidence. This is particularly seen in those suffering from depressive illnesses. In the schizophrenic much may be done to delay and prevent the development of deterioration. Since the outbreak of War its application in the treatment of physical diseases and injuries has received a great stimulus. There is no doubt that much more might be achieved in this direction.

SOCIAL FACTORS: Because of the great frequency of environmental factors in the etiology of mental illness, it is generally necessary, at an early stage, to decide where the patient is to be treated. No hard and fast rules can be elaborated as to when the patient should enter hospital.

§ 906. Certification.—The **question of removal** to a mental hospital depends on many things, chiefly (i.) the manageability of the patient; (ii.) the means at home for control; and (iii.) the character of the mental disorder and its potentiality for homicide or suicide.

The patient is not legally certified. Any mental patient, however mentally ill, can be taken care of by his or her relations *without certification,* provided it is done without payment or restraint, they being responsible for the patient's safety.

Cases of slight eccentricity and *uncertifiable* mental aberration may be received into the house of a medical man or other householder for payment; but directly a case becomes *certifiable* (in the opinion of the Commissioners) it must be treated in a place approved by the Board of Control. The penalties for breach of this are very heavy. No medical man or other householder may retain in his house more than one certified patient at a time without special permission from the Commissioners.

Additional provisions for the reception of mental patients were made in the Mental Treatment Act of 1930. *Voluntary patients* may be received not only in private mental hospitals and registered mental hospitals, but also in State institutions. It is necessary that the patient should sign a form of request for admission. He is free to leave on giving seventy-two hours' notice of his intention to do so. Otherwise patients can be admitted under certificates or in the category of *Temporary patients.* In the latter case they are admitted to mental hospitals without a Magistrate's Order and on the recommendations of two medical men, one of whom must be specially appointed under the Act, and it must be stated in the recommendations that the patient is in such a condition that he is unable to express himself as being either willing or unwilling to receive treatment. This absence of volition in the case of Temporary patients is an important condition.

The patient is legally certified. **Procedure for Removal of Persons of Unsound Mind under the Lunacy Act.**—The procedure for removal is somewhat intricate, and it is useful to remember that the duly authorised officer of the Local Health Authority is a most convenient person to apply to, bearing in mind that it is no part of his duty to undertake private cases, but that, nevertheless, if he be approached with due regard to the importance of his office, he may save those concerned a great deal of trouble, and supply them with all the necessary forms and particulars as to modes of procedure. He is also in constant relation with the justices.

A person deemed to be of unsound mind and found *wandering at large* not under proper care, can be apprehended by a constable or the duly authorised officer of the area, and taken to the institution. Any person deemed to be of unsound mind can, for his own safety or that of others, be removed from a dwelling-house by a duly authorised officer to the institution. In either case the patient can be detained there for three days upon the certificate of such constable or duly authorised officer, and, further, upon the certificate of the medical officer of the institution, for a total of fourteen days. Meantime the procedure under No. 3 (*a*) below can be instituted. This method is now often utilised for persons in all classes of life who are dangerous and away from their friends.

The Urgency Order (1, below) can be used in urgent cases. This holds good for seven days from its date; if not urgent (2) is the usual method.[1]

A patient can be removed to a mental hospital in England or Wales in the following ways:

(1) Under an Urgency order signed by a relation (or guardian) and one doctor.
(2) Under a Reception order of a Justice obtained by petition of a relative on two doctors' certificates (used also for certification in a case for single care).
(3) Under a Summary Reception order of a Justice.
  (a) On information from a duly authorised officer that a person is deemed to be a person of unsound mind and a proper person to be taken charge of and detained under care and treatment, a Justice calls in one doctor, who certifies unsoundness of mind.
  (b) On information from the police or a duly authorised officer that any person wandering at large is deemed to be of unsound mind, a Justice calls in one doctor who certifies unsoundness of mind.
(4) Under an order after Inquisition, being a written authority from the "Committee" of the person, together with an office copy of the order of the Court of Chancery appointing the "Committee." The "committee" is a legal phrase for the guardian appointed by the Court.
(5) Under a written application by a relative, or responsible person, supported by two medical recommendations.
(6) Under the Criminal Justice Act, 1948:
  (a) A Court may include in a Probation Order a requirement that an offender shall submit to treatment in a Mental Hospital for a period not exceeding 12 months from the date of the Order.
  (b) A Court of Summary Jurisdiction has the power to make an Order for the detention of an offender in a Mental Hospital. This Order has the same effect in law as a Summary Reception Order.

The procedure in Ireland and Scotland is somewhat different, as is also that under the Mental Deficiency Act, *vide* § 907.

Testamentary Capacity.—A knowledge of what constitutes the testamentary capacity of a patient is of great importance to the practitioner, because it is often on his evidence that courts of justice decide such matters. The testamentary capacity of a person of unsound mind depends practically on three questions:

1. Did he at the time understand the nature of a will and its effects, and did he understand the extent of the property of which he was disposing ?
2. Did he provide for his relatives, or, if not, why did he leave them out ?
3. Had he any delusion bearing on testamentary matters ?

If these questions can be satisfactorily answered and proven, the will is valid, however eccentric the patient may have been, or even if he was at that time certified as of unsound mind. The fourth question—undue influence—is a non-medical question.

The patient is a child, showing signs of MENTAL DISORDER.

§ **907.** VII. **Mental Abnormalities in Children** may be classified as:

A. PSYCHONEUROSES and BEHAVIOUR DISORDERS.
B. PSYCHOSES.
C. MENTAL DEFICIENCY.

A. (1) The *nervous child* corresponds in childhood to the anxiety neurosis in the adult. Anxiety is frequently displayed by children. It may occur in the form of acute anxiety attacks with concomitant signs and symptoms to the adult; more often it appears to become absorbed into the constitution. Such children are generally

[1] All the forms necessary are procurable from Shaw, Fetter Lane, London, or from the duly authorised officer. Names of Justices for individual areas can be obtained from the local Clerks to the Justices. A list of these can be found in the Local Government Manual and Directory (at all reference libraries).

the offspring of nervous parents from whom the children absorb their anxiety. In the majority of children with such symptoms, there is nothing intrinsically wrong; the condition is the result of environmental factors. Hysterical manifestations of a minor degree are common; headache, nausea and vomiting; usually they are the result of suggestion. Marked symptoms, such as paralysis, anæsthesia, etc., are much less frequent; when they do occur, the psychological mechanisms are usually superficial. Having satisfied oneself as to the absence of a physical cause, disregard of the hysterical symptom, and refusal to allow any gain from it are often sufficient to cause the symptom to cease. Obsessive compulsive features are present in minor degree in most children, and are rarely to be taken seriously. Simple explanation and reassurance are usually adequate.

(2) *Habit disorders* of varying degree form a large proportion of children's problems of psychiatric interest. In the earliest stages habit training comes from parents, later from the school influence and school companions. Morbid as well as healthy reactions may be impressed. The most frequent habit disorders are enuresis, nail-biting, stammering and sleep-walking. Hubert has recently drawn attention to the pronounced hereditary tendency so frequently found in these conditions. Enuresis often occurs in several members of a family; it may be the result of anxiety, lack of education or negativism. Sleep-walking also commonly occurs in more than one member of a family; when psychological in origin the goal usually suggests the interpretation. Stammering is common in timid, over-anxious children, factors already increased by their disability. Speech training is advocated together with the readjustment of psychological difficulties. Amongst the grosser forms of behaviour abnormalities come lying, unmanageability, temper tantrums, stealing and truancy. Isolated instances probably occur, at some time or other, in the life of all children; but their repeated occurrence makes them pathological and renders further investigation necessary. Lying may be of two varieties: (*a*) defensive to protect from consequences, (*b*) the result of phantasy, and is then a projection of wishes and desires into realisation in words. Temper tantrums are usually the result of methods of handling, and are developed as a means of achieving some end.

These problems in children indicate, as a rule, some difficulty in adaptation, and a frustration of some desire. Beyond obtaining, in a general way, the child's attitude towards the problem, in most cases it is undesirable and unprofitable to submit the child to a more detailed psychiatric investigation; very rarely is this necessary. More can be gained by a study of the environmental factors, the setting in which the child moves, and the attitude adopted towards him, by those with whom he comes in contact. Educational difficulties are responsible for numerous problems. Some are the result of varying degrees of retardation, but on the other hand great intellectual capacity is not synonymous with good mental health. These difficulties are investigated by means of psychological tests (see § 701). The value of such an investigation is seen particularly in studying delinquency. Many of these children show difficulty in school adjustment, and the restlessness and discouragement thus created play no small part in determining their conduct.

The detailed investigation of adult psychiatric problems now employed has revealed the great frequency of neurotic symptoms in childhood. The "problem" adult is frequently the end result of the "problem" child. Consequently greater attention must be paid to these symptoms while it is possible to eradicate them. Discrimination is necessary as to how much the child should be treated, and how much the environment. Only in a few cases is psycho-therapeutic treatment called for. A readjustment of the environment is frequently necessary; to obtain this, the temporary removal of the patient to new surroundings may be necessary. One should endeavour, then, to modify the conditions that either suggest ideas of misconduct, or that may reawaken the ideation which creates the impulse to misconduct.

B. **Psychoses.**—Psychoses in children are rare. Especially is this so prior to the development of secondary sexual characteristics. Thereafter affective disorders, manic-depressive and schizophrenia, are sometimes met with. The clinical picture

in the former condition is the same as that found in adults (§ 895). Where children are affected there is usually a strong family history of depressive conditions. Schizophrenia is characterised by an abnormal emotional reaction. Such patients lose interest and become apathetic; news that previously would have caused sorrow now provides cause for laughter. The most prominent feature is the incongruity between the emotional state and the thought processes. These patients withdraw from reality; phantasy formation becomes prominent and acquires for them an objective reality. Numerous theories have been elaborated to account for the condition. There is no definite evidence of organic changes. The onset is in most cases of an insidious nature, beginning in childhood; the previous personality is that of a reserved, seclusive type with few friends and interests. During childhood peculiarities of behaviour occur which separately seem of no importance, but, viewed collectively later, they show their true significance. Meyer has suggested that the condition is the result of inadequate adaptation of the individual to his environment; that it is the result of faulty habits of reaction whereby the problems of life are inadequately dealt with, culminating later in the substitution of phantasy for activity. Insufficient attention has been paid to these oddities of behaviour, and when advice has been sought the patients have been unable to co-operate in treatment. The aid of the specialist should be sought before the stage of readaptation is past.

Congenital forms of *General Paralysis*.—Not infrequently they occur, after the age of seven, in the offspring of adult general paralytics. The condition is characterised usually by deterioration in a previously alert and active child. Memory changes occur, and marked intellectual impairment develops in a short space of time. The younger the child when the symptoms appear the more chronic the course of the condition. Physical signs in the form of speech abnormalities, Argyll-Robertson pupils, tremors and active tendon reflexes are found. Serological examination gives the same findings as in the adult (§ 902). Treatment by malaria plus arsenical compounds is less effective than in the adult. Unless the condition is diagnosed and treatment instituted in the early stages the prognosis is very poor.

Behaviour difficulties of all degrees of severity may be sequelæ of encephalitis lethargica (§ 698). The milder forms are characterised by nocturnal wakefulness and excitability, disobedience, irritability, stealing and outbursts of temper entirely unprovoked. Frequently the child is aware of the impulse to do wrong but is unable to control it. Young children show a greater degree of mental impairment, whereas older children show a moral change. In the Apache group the symptoms are more severe. The children become aggressive, untruthful, quarrelsome and often subject to outbursts of acute excitement. Many of these require institutional treatment, and the ultimate prognosis is not good.

Under the Mental Treatment Act, 1930, provision is made for the reception, into Mental Hospitals, as voluntary patients, of children under sixteen years of age, whose condition is such that they are likely to benefit from treatment there. Application must be made by the parent or guardian, and it must be accompanied by a medical recommendation by the family physician, or by a physician approved for the purpose by the Minister of Health.

C. **Mental Deficiency.**—Four degrees of mental deficiency have been defined in the Mental Treatment Act. They vary largely according to the degree of social incapacity, viz., idiots, imbeciles, feeblemindedness and moral defect. The first three vary according to mental capacity and are differentiated by means of special psychological tests. As the result of numerous experiments carried out on normal school children, these tests have been grouped according to the period of life at which accomplishment may be reasonably expected, and they have been elaborated into a definite scale (§ 701). For each year of life a combination of tests is employed, the average of which gives a more representative value than any one test alone. If the child cannot do the tasks proper to its age, but can only accomplish those proper to a younger child, its mental age is reckoned to be that of the younger child, in other

words, so much less than its real age. Normally the mental age and the chronological age should correspond; the ratio of the one to the other is termed the *intelligence quotient*. Apart from the actual results achieved by the child, valuable data are obtained from a study of its application in the performance of the various tasks. It may be taken that idiots have a mental age under three, imbeciles under seven, and feebleminded under twelve. If the child has attended a Council School in England a rough indication of its ability may be formed by ascertaining the standard to which it reached. Thus the average age in the infants' school is under five years and in Standard I seven to eight years, with an increment of one year for each succeeding standard, Standard VII being reached by normal children at thirteen or fourteen.

CONGENITAL APHASIA (§ 744) though uncommon is of considerable importance, as the sufferer may be wrongly regarded as mentally defective.

MENTAL DEFICIENCY may be primary—*i.e.*, due to factors causing defective germ plasm; or secondary—due to causes acting upon the fœtus or infant. 75–80 per cent. of mental defectives belong to the primary group. The pathogenesis of certain types is not yet clearly defined, consequently it is impossible to be dogmatic regarding their appropriate groups.

The main clinical varieties of PRIMARY MENTAL DEFECT are: 1. *Simple* or *Genetous* Idiocy—a large group without characteristic features enabling it to be further subdivided. It includes children without any obvious abnormality of the cranium or limbs, only in the face or palate. In some the facial expression may be fairly intelligent, but most of the lower grade present an animal expression, thick lips, pug-nose, large coarse ears, broad, thick, depressed bridge of nose, narrow or hairy forehead, and underhung jaw.

2. The *Mongol* or *Chinese* type of congenital deficiency is so called from the resemblance of the face to that of the Chinese, the palpebral fissures sloping downwards and inwards. With flat face, flat back to the head, and constant protrusions of the tongue, this form of idiocy presents an unmistakable physiognomy. The fingers also are stunted and the little fingers incurved. Congenital heart disease occurs in about 30 per cent. They may be regarded as "unfinished" children, as they are often born of mothers who have suffered from continued ill-health during pregnancy; sometimes they are the youngest of a large family, or born of parents advanced in life. These children are imitative, and therefore educable to a limited extent, but they make no progress beyond a certain point.

3. *Microcephalic idiocy* includes children whose heads have a smaller circumference than the normal, which averages about 19 inches. The head may measure 17, 15, or even 12 inches; the forehead is narrow, and slopes backwards, corresponding with the deficiency of the frontal development of the brain. The small skull is the expression and not the cause of the small brain. The features are frequently normal, eyes large, and nose aquiline. These children rarely make much improvement, for they have but little power of attention, though some of them are imitative. The majority are imbeciles.

4. *Sclerotic amentia* due to an overgrowth of neuroglia occurs in two forms—(*a*) nodular (tuberose), and (*b*) diffuse. (*a*) *Tuberose sclerosis* is characterised by mental defect, epileptiform attacks, and adenoma sebaceum, a skin eruption appearing on the face as a rule between the ages of 4 and 6 years. (*b*) *Diffuse sclerosis* may give rise to an increase in the size of the brain, producing what is frequently described as hypertrophic amentia. The condition may be differentiated from hydrocephalus by the level of maximum enlargement; also in hydrocephalus the enlargement is generally more marked and is accompanied by a bulging of the fontanelles and sutures. Weakness, epileptiform seizures, and varying degrees of mental defect are the conspicuous features.

5. *Oxycephaly* is accompanied by a marked deformity of the skull (§ 13). As the sutures are united prematurely, the cranium is expanded upwards so that the frontal region is greatly increased in height, and the head is short from before backwards. The

bones of the skull are abnormally thin and ocular changes are frequent. There may be synostosis of the fingers and toes. Varying degrees of mental defect are not infrequent, but the changes in the cranium may exist without any mental defect. The condition may occur in more than one member of a family.

The main clinical varieties of SECONDARY MENTAL DEFECT are :

1. *Hydrocephalic*, often due to the occlusion of the foramina of Magendie or Monro, causing distension of the ventricles with fluid and atrophy of the cortex. The bones become widely separated and the head is globular in shape. Most cases are quiet and docile and there is frequently muscular weakness or paralysis. Epileptiform convulsions are frequent, but tend to decrease as the condition becomes stationary (§ 830).

2. *Epileptic.*—Infantile convulsions, indistinguishable in many cases from those of ordinary epilepsy, may result from many causes. Where no cause can be ascertained it is looked upon as idiopathic. A large proportion of such occur in the offspring of epileptic, psychotic or psychopathic individuals. When the fits develop in early life, before the age of 7, intellectual development is arrested and mental defect frequently results (§ 721).

3. *Paralytic.*—The majority of cases in this group result, as a rule, from trauma at birth and only very occasionally from an injury during early life. There is some support for the view that certain cases are of intra-uterine origin, and not dependent, merely, upon the result of intra-cranial hæmorrhage. The resultant lesion is dependent on the site and degree of damage. Accordingly hemiplegia, diplegia, or epileptiform attacks may be concomitant symptoms. Sometimes these cases are associated with spasticity or choreiform movements and symptoms are produced due to a cerebral scar (§ 762).

4. *Inflammatory.*—This follows from encephalitis and meningitis, from scarlet fever or other exanthema, and the mental defect may not supervene till later, as is also seen after epidemic encephalitis (§ 698).

5. *Syphilitic.*—Signs of congenital syphilis are often present ; in some there is evidence of gross brain damage such as paralyses, seizures, deafness, and blindness. The degree of defect consequently varies greatly and treatment offers little hope of improvement (§ 552).

6. *Amaurotic Family Idiocy* occurs chiefly, though not entirely, in Jews, often in more than one member of the same family, sometimes in successive generations. The symptoms appear during the first three to six months of life and lead to idiocy accompanied by a progressive paralysis and blindness. The characteristic cherry red spot is visible, on ophthalmoscopic examination, in the region of the macula (§ 763).

7. *Schilder's Disease* may occur in various members of one family. The symptoms may appear in early life or during childhood in those previously of normal mental development. Mental enfeeblement together with blindness, deafness and a spastic paraplegia are the conspicuous symptoms. The disease is the result of extensive cortical demyelinisation and effective treatment is unknown.

8. *Mental defect from deprivation of the senses.*—The mind is cut off from environmental stimuli owing to sight and hearing being affected from acute infections, trauma or hæmorrhage. The defect may be remedied by means of special training.

9. *Cretinism* may be endemic or sporadic. The head is usually large, flat at the top, spread out at the sides. The hair is coarse and dry and the voice squeaky. Under treatment by thyroid these cases make remarkable progress (Figs. 8*a*, *b* and *c*), but the treatment must be continued during the whole of life. And see § 191.

Etiology.—The influence of heredity is noticeable in these children; they frequently come from a neuropathic stock. Their family history invariably shows varying degrees of mental abnormality in both the immediate and more remote members. The exact influence of syphilis is difficult to assess. A positive Wassermann reaction is rarely met with. It has been suggested that the association of tuberculosis and mental deficiency is indirect, probably dependent on the intermediation of poverty.

Minor ætiological factors are injury, anxiety and worry. There is support for the belief that the early application of forceps is preferable to an indefinitely prolonged labour. Of the factors operating after birth the chief are glandular deficiencies, epilepsy, brain disease, injuries, sense deprivation and infectious diseases.

The *prognosis* of mental deficiency is always grave. The degree of defect, the clinical type, and the environmental factors are the chief points to be considered. The variety of simple aments do better than the special forms. Many backward children are made more defective by the home atmosphere, and much would be achieved if relatives would treat such children from the point of their mental maturity rather than their actual age and physique.

Treatment in all cases consists in utilising to the best advantage what abilities there are. Recovery cannot be expected. Idiots and imbeciles usually require institutional treatment or private care. The results of training are, in some cases, surprisingly good. Much can be accomplished for feeble-minded children in Special Schools; the conditions there permit of a great amount of individual attention. The aim is to emphasise the physical and personal training, and to organise the behaviour of such children into helpful and useful activity. Much attention has been given to the question of sterilisation. The Departmental Committee recommended recently that voluntary sterilisation should be legalised in the case of (*a*) one who is mentally defective or has suffered from mental disorder, (*b*) one who is believed to be likely to transmit mental defect or disorder. In certain European countries and in some of the States in the U.S.A., compulsory sterilisation has been enforced. The proposal, so far as this country is concerned, is to create facilities for performing the operation only on those persons who agree to it. As yet sterilisation is illegal, except as a therapeutic measure.

The Mental Deficiency Act is devised to protect and control defectives, and thus to prevent them from doing harm to themselves and to society. Mild cases of defect can be received in approved homes without certification. Other cases can be placed under single care, or in houses and institutions certified under the Act, including state institutions. Two medical certificates are necessary for all grades, and for an idiot or imbecile the order of the parent or guardian suffices, but for the feeble-minded and moral defective the intervention of a Justice is required in addition. The requisite forms for the Act may be procured from Shaw, Fetter Lane, London.

CHAPTER XXI

EXAMINATION OF PATHOLOGICAL FLUIDS AND CLINICAL BACTERIOLOGY

In this chapter the methods of obtaining various pathological fluids, how to examine them, and their characters, are briefly described, and an epitome of the chief bacteriological data required for clinical work is given.

§ 918. **Sterilisation of Syringes.**—To eliminate the virus of homologous serum jaundice ("syringe jaundice") and spores, all-glass syringes and stainless steel needles (No. 14 Record or No. 20 Luer) are placed in a large test-tube, the mouth of which is plugged with cotton wool. To sterilise, place in a hot air steriliser at 160° C. for not less than 1 hour; alternatively in an autoclave at 120° C. (15–20 lbs. pressure) for 20 minutes. Otherwise boil in water for not less than 5 minutes, or immerse the barrel and plunger separately in 70–75 per cent. alcohol and rinse in sterile water or saline subsequently—but these methods do not necessarily destroy spores or the icterogenic virus.

§ 919. **Methods of Obtaining and Examining Pathological Fluids.**—Fluid from the serous cavities is best obtained with a 20-c.c. all-glass or Record syringe and an exploring needle of larger bore than the ordinary hypodermic needle to permit a thick purulent fluid being drawn through it. Sterile precautions must be adopted. The needle and syringe are sterilised before use. The operator thoroughly cleanses his hands by washing and scrubbing, and uses a sterile towel to dry them. The patient's skin is sterilised with ether or tincture of iodine. To render the puncture as painless as possible, inject intradermally a small quantity of 2 per cent. procaine hydrochlor. B.P. (novocain) with a sterile hypodermic needle and syringe at the site of intended puncture; through the intradermal wheal insert the exploring needle. In the case of non-purulent fluids, a portion collected for pathological investigation should be added to a sterile tube containing a small amount of 3 per cent. sodium citrate solution. This prevents clotting and so facilitates accurate protein content estimation, cytological examination, and demonstration of tubercle bacilli.

The **pleural** cavity is best explored at the seventh or eighth space in the post-scapular line or at a site where localised dullness is maximal. For details see § 119.

Pericardial fluid is obtained by inserting a needle in the fourth or fifth left inter-space at the extreme left limit of cardiac dullness. It should only be attempted when a moderately large effusion is present. See paracentesis pericardii, § 46.

The **peritoneal** cavity is explored for fluid by **paracentesis abdominis** midway between the umbilicus and pubes, or in the right iliac fossa midway between the anterior superior iliac spine and the umbilicus. The puncture must be made over a dull area and with an empty bladder. The patient is propped up in bed with two or three pillows; a many-tailed bandage placed around the abdomen, and tightened to maintain the abdominal pressure as the fluid drains away: otherwise the patient may collapse from the rapid dilatation and congestion of the splanchnic area. After sterilisation of the skin, anæsthetise it and the subjacent abdominal wall with 2 per cent. procaine at the proposed site of puncture. A small incision is made in the skin with the point of a sterile scalpel. Then insert a small trocar and cannula into the abdominal cavity. The trocar is withdrawn and, if fluid escapes, sterile rubber tubing is attached to drain the fluid into a receptacle at the side of the bed. The cannula is fixed in position with gauze and strapping. If the flow of fluid stops, and ascites is still present, alter the direction of the cannula in an attempt to restart it, or turn the patient towards the right if the puncture is in the right iliac fossa. When no more

fluid can be obtained, the cannula is withdrawn, the site of puncture painted with tincture of iodine and covered with a sterile collodion dressing.

Liver Biopsy may reveal conditions such as Kala-azar, Boeck's sarcoidosis, cirrhosis, hæmochromatosis, primary or metastatic growth and reticulo-endothelial disorders. Using a Gillman or Terry's instrument, insert the needle under local anæsthesia through the 7th right intercostal space in the anterior axillary line, the patient holding his breath during the puncture. A core of liver tissue 3 cms. in length is obtained and suitably fixed for histological section. With a hæmorrhagic tendency, estimate the prothrombin level of the blood beforehand and give Vitamin K when necessary. Only experienced operators should undertake the biopsy—fatalities or complications are occasionally met.

Liver Aspiration for the presence of pus or of Leishman-Donovan bodies is undertaken with a needle where indicated, or in the mid or anterior axillary line with a needle not longer than 90 mm. to avoid possible injury to the portal vein. When staining for Leishman-Donovan bodies, spread the contents of the needle on a glass slide. Liver aspiration must not be performed if a hydatid cyst is suspected, for fear of dissemination in the peritoneum.

Spleen Puncture is used in the diagnosis of Kala-azar, and Gaucher's disease: it should only be resorted to after other methods have failed. Leukæmia or a hæmorrhagic tendency is a contra-indication. *Method.* With the patient flat on his back and hands folded beneath the head an assistant holds the spleen firmly against the diaphragm and ribs. The skin over the intended site of puncture is sterilised with ether or liq. iodi mit. B.P. Puncture is carried out with a direct firm thrust using a No. 14 size needle and a dry syringe, the patient holding his breath. Without delay forcible aspiration is made and the needle then immediately withdrawn sharply in one motion. The patient is kept recumbent for 1½ to 2 hours and the pulse rate checked at intervals for signs of hæmorrhage.

Gland Puncture is employed to detect plague bacilli, trypanosomes, and treponema pallidum. The technique is similar to that of puncture in any other region.

Lung Puncture.—When no sputum is available, and an area of infection can be located, lung puncture may provide material for examination and culture.

Lumbar Puncture is employed to (1) collect specimens of the cerebro-spinal fluid for examination, (2) relieve intracranial and intrathecal pressure, (3) drain or wash out the spinal canal, for which purpose it may be combined with cisternal puncture, and (4) as a preliminary to the injection of sera, drugs and antibiotics. It is advisable that all patients should remain in bed after lumbar puncture for twelve to twenty-four hours. In certain conditions, notably cerebral tumour and disseminated sclerosis, this should be made an absolute rule, and the patient should not even be allowed to sit up for several hours afterwards. Lumbar puncture should be performed with the patient lying on his side with his back over the edge of a firm couch or bed. The head is on the same level as the sacrum and is flexed well forwards; the knees are drawn up and the back arched. The best site for the puncture is the interspace between the third and fourth lumbar vertebræ. A line joining the highest points of the iliac crests crosses the spine at this level. After sterilising the skin at the intended site of puncture, anæsthetise it with an intradermal injection of 2 per cent. procaine hydrochlor. and then infiltrate the subjacent tissue. The operator, after sterilising his hands, presses deeply into the interspace between the third and fourth lumbar spines and pushes the point of the lumbar puncture needle through the anæsthetised skin either directly in the middle line, or slightly to one side of it. The needle, with the bevel downwards, is then passed forwards and slightly upwards towards the spinal canal which it should reach at a depth of 4 to 6 cm. without encountering any resistance except at the ligamentum flavum. When it is felt that the needle has entered the spinal canal the stylet should be withdrawn, and after a few seconds the first drops of cerebro-spinal fluid should appear. If no flow results the stylet is replaced, and the needle pushed a little farther on. If the needle strikes bone, it should be withdrawn a short distance and inserted in a slightly different direction. When the puncture is

made solely for diagnostic purposes as little fluid as possible should be removed, and the sample should be free from blood. The fluid normally runs out slowly (about one drop per second): but when under increased pressure, as in hydrocephalus and meningitis, it may spurt out; the intrathecal pressure can be roughly judged by the rate of flow; an accurate reading is obtained with a special manometer. In normal patients in the horizontal position the pressure varies from 60 to 150 mm. of cerebro-spinal fluid. For examination (§ 920) 5 c.c. are enough, but for treatment (below) 10 or 15 c.c. up to 50 c.c., according to indications, may be removed. The following precautions are essential: (1) strict asepsis; (2) the rate of withdrawal should be slow, not more than 4 or 5 drops a second; and (3) the patient should lie down for several hours afterwards. An additional safeguard is the employment of the smallest possible needle, which will inflict the minimum injury on the spinal theca, and so prevent the escape of cerebro-spinal fluid. There is one contra-indication. When there is increased intracranial pressure due to a cerebral tumour, the sudden reduction of this pressure in the cavity of the spine which results from the withdrawal of fluid may cause the descent into the foramen magnum of a part of the cerebellum. Especially is this so when the cerebellum has already been moulded to form a "cerebellar cone" in its efforts partially to slip into the foramen magnum to relieve the intracranial pressure. When this accident occurs, there may be direct compression of the medulla, the symptoms of which range from syncopal attacks to sudden death. A further danger is that the reduction of the pressure may start hæmorrhages into a soft growth, or allow arrested bleeding to begin again.

Cisternal Puncture is useful (1) when the spinal subarachnoid space is blocked by tumour or adhesions; (2) as a means of irrigating the spinal subarachnoid space (in combination with lumbar puncture); and (3) as the best route for injecting anti-sera or other remedies in meningitis. It is also valuable as a means of determining the presence or absence of spinal subarachnoid block—firstly, by the introduction of 1 c.c. of lipiodol into the cisterna magna, followed by X-ray examination of the spine; the level to which the opaque lipiodol has fallen indicates the upper level of the block (§ 757):—secondly, by a careful manometric study of the fluid in the cisterna magna and that in the lumbar cul-de-sac, and a comparison of the physical and chemical characters of the fluid obtained from these two situations. The cerebro-spinal fluid is tapped by a needle introduced between the cerebellum and the medulla. The usual method is: Shave the hair from the external occipital protuberance downwards to the neck. Sterilisation of the skin should be thorough, but, as the operation may have to be repeated, not too drastic. Do not use iodine on very young patients. The most convenient position for the patient is lying on the left side. Make certain that the line of the spine and head is straight; the head should be slightly bent forwards, and supported on a firm pillow. A general anæsthetic is not necessary; local anæsthetisation of the skin is usually employed. The utmost care is necessary to prevent the patient from moving once the operation is begun. *First Stage:* Place the index finger of the left hand on the external occipital protuberance, and pass it down the neck till the tubercle of the atlas vertebra, the next bony point, is found. Introduce the needle half a centimetre above this point. The direction of the needle should be towards, or a little above, the outer end of the left eyebrow. No fixed mark can be given; aim at touching with the point of the needle that part of the occipital bone which forms the posterior margin of the foramen magnum. It is better to go too high, than too low, with the point of the needle. *Second Stage:* As soon as the point of the needle is felt to be against the bone it is slightly withdrawn, its direction altered and the point depressed so that it just clears this edge of bone when it is again advanced. Several advances and withdrawals may be required before the point of the needle is free of the bone and gripped by the occipito-atlantoid membrane. The stylet is then withdrawn, and the needle pressed forwards till a flow of fluid is obtained. The depth at which the bone is met should on every occasion be noted, for the final advance must not exceed an extra 1·5 centimetres. The total depth of the puncture varies so greatly that no representative

figure can be given. Two figures should be remembered: half a centimetre above the tubercle of the atlas, the point of introduction, and 1·5 centimetres, the maximum extra depth required after last touching the occipital bone. The specimen should be collected in two sterile tubes. After the operation keep the patient at rest lying down.

Epidural Treatment (used in obstinate sciatica).—The epidural space is reached by piercing the posterior sacro-coccygeal ligament; the superior mesial protuberance and two lateral tubercles of the sacrum can readily be felt. The injection is made in the middle line between the two tubercles. After a preliminary injection of 20 c.c. of a 1 per cent. solution of procaine, inject sterile normal saline from 60 to 80 c.c. Thereafter the patient should rest for twenty-four hours.

Treatment of Increased Intracranial Pressure by Means of Hypertonic Solutions.—Hypertonic solutions lead to a withdrawal of water from the tissues, a reabsorption of cerebro-spinal fluid, and a pronounced fall of C.S.F. pressure—the fluid reaches the perineuronic spaces *via* the perivascular spaces and is absorbed by the capillaries of the brain. To obtain a reduction of intracranial pressure, (1) rectal administration is on the whole the most useful. 1 oz. magnesium sulphate and 1 oz. olive oil are diluted to ½ pint with water: this should be run into the rectum by means of a catheter and funnel, and effort made to retain it for half an hour or longer. (2) A more rapid reduction is obtained with intravenous injections of sterile solutions of 30–50 c.c. of saline (30 per cent.) or of dextrose (50 per cent.).

Veni-Puncture.—Blood may be required for serological, cultural, chemical and other purposes. Veni-puncture is also necessary for transfusions, intravenous medication and simple bleeding. The most convenient site is one of the superficial veins of the front of the elbow. Apply a tourniquet to the upper arm just sufficiently firm to obstruct the venous return, and ask the patient to clench his fist. The veins stand out clearly; in fat persons the veins are often felt even when not seen. In difficult cases, warm the limb in hot-water or by a hot towel. Cleanse the skin with ether. Introduce the needle into the vein in a direction nearly parallel to the skin surface. Fill the all-glass syringe; loosen the tourniquet and withdraw the needle, cover the puncture hole with collodion on a pad of wool. The blood is transferred to a suitable sterile tube, dry if serum is required, containing an anti-coagulant if whole blood is wanted. Bayer's venules are convenient in practice. In infants the external jugular vein is used. The nurse holds the child on her lap, with the head low and turned to one side. Crying distends the vein further.

Sternal puncture with biopsy of the bone marrow is of great value in the diagnosis of leukæmia (especially aleukæmic leukæmia), Addisonian anæmia, aplastic anæmia, myelosclerosis and multiple myelomatosis. It is useful in the recognition of Gaucher's disease, kala-azar, trypanosomiasis and rarely of carcinomatosis with metastases in bone. Negative results exclude leukæmia, and Addisonian anæmia, when the peripheral blood pictures are inconclusive. *Method.*—A Salah needle with stylet, furnished with an adjustable guard, is used. In an adult premedication is unnecessary, but in children syrup of chloral may be given half an hour beforehand. The site of puncture is the body of the sternum opposite the second or third interspace, a little to one side of the mid-line (in males shaving the chest wall may be indicated). The patient lies on his back with a thin pillow beneath the head. The skin, subcutaneous tissues and periosteum are anæsthetised with 2 per cent. procaine (novocain) solution. The Salah needle, previously sterilised and with the guard set at 1 to 1·5 cms. above the skin level, is then pushed through the outer diplöe of the sternum into the narrow cavity with a boring movement. This is accompanied by a characteristic "crunch" and the needle will stand upright when let go. The stylet is withdrawn, and with a 2-c.c. syringe about 0·5 c.c. of marrow fluid is gently aspirated. Dilution with blood must be avoided. Suction of the marrow is often associated with a twinge of pain if the point of the needle is in the marrow cavity. Films are made on slides and suitably stained. Some of the marrow may also be fixed and sections cut. The puncture wound is covered by a dry or collodion dressing. In young children marrow is best

obtained by puncture of the iliac bone 2–3 cms. below and behind the anterior superior iliac spine.

Sputum may be required for direct microscopical examination for bacteria, for malignant cells, or for cultural investigation. The value of a general bacteriological examination largely depends on the care taken to obtain the specimen as free as possible from contamination. A mixture with saliva or pharyngeal secretion gives unreliable results which are difficult to interpret. The material required is the phlegm coughed up from the chest; when this is scanty the best sample is obtained on rising in the morning. First the mouth and throat should be washed out by rinsing and gargling with plain water. The sputum should be expectorated directly into a sterile bottle which is sent to the laboratory with the least possible delay.

Fæces.—Only a small quantity is required for bacteriological purposes, but for full chemical analysis a larger specimen, *e.g.*, an ounce, should be sent. For bacteriological examination any mucus present should be included in the specimen. For the recognition of dysentery amœbæ the stool must be fresh and not allowed to cool; transmittance in a thermos flask may be necessary. For occult blood, see § 303.

Urine.—For cultural examination a catheter specimen should be obtained from females. In the male, a specimen for bacteriological examination may be collected under reasonably aseptic conditions as follows: the urinary meatus is washed with soap and water and dilute antiseptic (*e.g.*, perchloride of mercury 1 in 1000). The patient is then instructed to pass an ounce or so of urine, which is discarded; the remainder is collected into a sterilised bottle. If direct examination alone is required, the centrifuged deposit from an early morning specimen is the most satisfactory. For tubercle bacilli, when passed in small numbers and at irregular intervals, the standing deposit from a "twenty-four hours' specimen" gives the best chance of their demonstration.

Pus.—Pus may be collected in a sterile tube, or with the aid of a sterile swab. As large a specimen as possible should be collected, up to about 1 c.c. When pus is being evacuated from an abscess it is best to collect the specimen at the earliest possible moment, as later it is often mixed with blood, and sometimes contaminated.

How to Examine Pathological Fluids.—The source of the fluid is as a rule known, but certain cysts—hydatid, pancreatic and ovarian—give characteristic features. The possibility of an abdominal fluid being urine, as in hydronephrosis, should not be forgotten. For *cytological* and *bacteriological* examination about 10 c.c. of the fluid should be stood in an upright tube overnight, or spun in a centrifuge; the supernatant fluid is then poured off for chemical investigation, while with the deposit cultures are sown, films are made, both wet and dry, and if necessary animal inoculations carried out. Wet films should be examined with the $\frac{2}{3}$rd and $\frac{1}{6}$th objectives, to detect cells, crystals and hydatid hooklets. Stained films will show the type of cells, and the nature of any micro-organisms. It is difficult to diagnose isolated malignant cells, but in the case of malignant disease of the pleura or peritoneum, large multinucleate cells showing well-marked mitotic figures (the so-called Foulis' cells) are occasionally seen in the associated pleural and ascitic effusion.

Chemical examination of the clear fluid should include estimation of the specific gravity, reaction and the quantities of albumen and sugar present. For *sugar* the albumen should be removed by boiling and filtering; the same tests are employed as in urine analysis. For *albumen* see § 379.

Characters of Pathological Fluids (Table LX). In the pleural, pericardial and peritoneal cavities, inflammatory effusions (exudates) are to be distinguished from dropsical effusions (transudates). The presence of a large jelly-like clot formed rapidly on standing, a specific gravity above 1016, a protein content over 2 per cent., and a relatively high cellular content indicate an exudate. In a transudate, clot formation is slight or

Table LX.—Physical and Chemical Characters of Pathological Fluids.

Constituents, etc.	*Serous exudation as in Ascites or Pleurisy.*	*Transudation.*	*Hydatid Cyst.*	*Pancreatic Cyst.*	*Ovarian Cyst.*	*Hydronephrosis.*	*Cerebro-spinal Fluid (Normal).*	*Distended Gall-Bladder.*
Colour.	Greenish yellow.	Clear yellow or greenish yellow.	Clear and colourless or opalescent.	Colourless. Often mixed with blood.	Variable—clear yellow to black.	Clear and watery.	Clear.	Clear —or stained with bile.
Specific gravity.	Usually over 1016.	Rarely over 1012.	1006 to 1010.	Variable often—1008 to 1010.	From 1002 to 1005 (low sp. gr. points to cyst of broad ligament).	1008 to 1020.	1006 to 1007.	Low.
Spontaneous coagulability.	Very marked. Clots in 24 hours or earlier.	Slight. May clot after long standing.	Absent.	Absent.	Absent. Consistence variable—watery to colloid.	Absent.	Absent.	Absent.
Chemical composition.	Alkaline. Much albumen, 2 per cent. or more (serum alb. and globulin). Small quantities { Sugar. Uric acid.	Alkaline. Albumen 1 per cent. or less (serum alb. and globulin).	Alkaline. No albumen or negligible quantity. Much sodium chloride. Succinic acid.	Digests albumen in alkaline medium. Serum albumen present.	Alkaline. Albumen (serum alb. and globulin, and *metalbumen*).[1]	Traces of albumen, urea, and uric acid.	Trace of albumen and albumose. Small amount of a reducing agent. Protein 0·02%. Sugar 0·05–0·07%. Chlorides 0·73%.	Mucin present; may contain bile.
Microscopic Examination.	Very little débris. { Red blood cells. Leucocytes. Endothelial cells.	Even less débris than in serous exudation —similar constituents.	Characteristic hooklets, scolices, cyst membrane, hæmatoidin crystals.	Often cholesterin crystals.	Cylindrical ciliated epithelial cells, squamous cells, colloid concretions, red blood-cells, leucocytes, fat globules, cholesterin, fatty acid crystals, and hæmatoidin.	Sometimes renal epithelial cells, uric acid crystals.	A few lymphocytes, 1 or 2 per cu. mm.	May have cholesterin crystals.
Remarks.	May be variable quantity of pus present. Occasionally it is red with blood as in cancer.[2]	May be sanguineous, and, rarely, chylous.	Diagnosis made by microscopic examination.	The ability to digest albumen is a positive test; but in very old cysts the power is lost.	Metalbumen and ciliated cells the diagnostic points.	Only diagnostic points are presence of renal cells, and these may be absent; or urea.	In acute meningitis the fluid is cloudy, with more albumen and no sugar. Micro-organisms found aid diagnosis.	

[1] Metalbumen is thus tested for: Acidify fluid with acetic acid, boil and filter to remove other forms of albumen. Add to the filtrate three times its bulk of alcohol. Stand for twenty-four hours. Filter. Squeeze out precipitate and suspend in water. Filter. Filtrate has following reactions: (i) On boiling it becomes turbid, but no precipitate falls; (ii) on adding acetic acid no precipitate forms; (iii) on adding acetic acid and strong sulphuric acid a violet colour is produced; (iv) on adding acetic acid and pot. ferrocyanide the fluid is thick and yellow.

[2] Ascitic fluid usually shows the characters above described; "hæmorrhagic ascites," where blood is found in the fluid, occurs chiefly in cancerous peritonitis, and has been considered a fatal sign. Middleton reported cases of chronic peritonitis associated with hæmorrhagic ascites in alcoholics, who recovered.

TABLE LXI.—CEREBRO-SPINAL FLUID.

Condition.	*Appearance of Fluid.*	*Pressure of Fluid in Recumbent Posture.*	*Protein per cent.*	*Cells per cu. mm.*	*Type of Cells.*	*Organisms.*	*Wassermann.*	*Colloidal Gold Curve.*	*Sugar per cent.*	*Chlorides.*
Normal.	Clear, colourless; no clot on standing.	60–150 mms. water.	0·02–0·05.	0–5.	Lymphocytes.	None.	Negative.	0000000000 or 0011100000.	·05–·07.	0·72–0·75
Tuberculous meningitis.	Clear, colourless; spidery clot on standing.	Increased.	Up to 0·30.	10–400.	75 per cent. lymphocytes.	Tubercle bacilli in clot.	Negative.	Normal.	Diminished.	Fall to below 0·66.
Cerebro-spinal meningitis.	Hazy or turbid; dense clot on standing.	Increased.	Up to 0·30.	10–2000 or more.	Polymorphs.	Intra - cellular gram negative diplococci.	Negative.	Normal.	Diminished or absent.	·60–·70.
Pneumococcal, streptococcal and staphylococcal meningitis.	Hazy, turbid or purulent; dense clot on standing.	Increased.	Up to 0·30.	10–1000 or more.	Polymorphs.	Gram positive cocci.	Negative.	Normal.	Diminished or absent.	·60–·70.
General paralysis of insane.	Normal.	Increased.	0·05–0·10.	20–400.	Lymphocytes.	None.	Positive in over 99 per cent.	Curve such as 5555443200.	Normal.	Normal
Tabes dorsalis.	Normal.	Normal.	0·03–0·08.	10–80.	Lymphocytes.	None.	Positive in 70 per cent.	Curve such as 1233210000.	Normal.	Normal.
Meningo - vascular syphilis.	Normal.	Normal.	0·03–0·08.	10–80.	Lymphocytes.	None.	Positive in 50 per cent.	Curve such as 0014320000.	Normal.	Normal
Encephalitis lethargica.	Normal.	Usually increased	0·02–0·05.	0–10.	Lymphocytes.	None.	Negative.	Normal or luetic.	Normal or slightly raised.	Normal.
Disseminated sclerosis.	Normal.	Normal.	Normal.	Normal or slightly increased.	Lymphocytes.	None.	Negative.	Normal or 1121000000.	Normal.	Normal.
Acute anterior poliomyelitis.	Normal.	Normal or increased.	Raised to 0·10 from end of first to end of sixth week.	10–1000 from pre-paralytic stage to end of second week.	Polymorphs, then Lymphocytes.	None.	Negative.	Curve such as 0123210000	Normal.	Normal.
Cerebral tumour.	Normal.	Increased.	0·02–0·10.	Normal.	Lymphocytes.	None.	Negative unless due to syphilis.	Normal.	Normal.	Normal.
Spinal blockage (Loculation or Froin's syndrome): and some forms of polyneuritis.	Clear, straw to amber; dense clot.	Diminished or normal below block	0·30–4·00.	Normal, or slight increase.	Lymphocytes.	None.	Negative unless due to syphilis.	Normal.	Normal.	Normal.

absent, the specific gravity usually below 1012, the protein content under 1 per cent., and the cytological content small. The transudate of cardiac œdema has a higher protein percentage than that of renal œdema. Blood in distinct amount in these cavities suggests neoplasm, but occurs with tuberculous disease of the pleura and with peritonitis associated with cirrhosis of the liver. A few blood cells, sufficient to give the fluid a rosy tinge, may occur with simple acute inflammation. The character of the cells in a pleural fluid may aid diagnosis of the cause of the effusion ; thus, an excess of lymphocytes points to tuberculous pleurisy, the predominance of polymorph cells to pyogenic or other causes.

Guinea-pig inoculation of a sterile pleural effusion may establish its tuberculous origin when tubercle bacilli cannot otherwise be demonstrated in it. The clot ground up in normal saline in a sterile mortar, or the centrifuged deposit form the best material for inoculation. A positive result is obtained only in about 70 per cent. of cases, whose subsequent history proves to be tuberculous ; in some cases several animals should therefore be used. Cultivation of the deposit or clot on a medium specially selective for tubercle bacilli, such as that of Lowenstein-Jensen, is successful in a high percentage of cases and frequently in a shorter time than a positive animal inoculation result.

The special characters of the various fluids and cysts are given in tabular form ; but a few words should be said here about the cerebro-spinal fluid.

§ 920. **Cerebro-Spinal Fluid** should be a clear, colourless, watery fluid, alkaline in reaction and with no clot formation. A trace of albumen (0·025 grm. per cent.) is normally present, and also a trace of sugar (partial reduction of Fehling's solution). In acute meningitis the fluid may exhibit varying degrees of *cloudiness*, from slight turbidity to almost pure pus, and in pyogenic infections the causal organism may be demonstrated in stained films. The presence of *blood* may result from a head injury, cerebral hæmorrhage, or trauma of the venous plexus on the anterior surface of the vertebral canal at the time of the lumbar puncture. Recent hæmorrhage (as in the latter) can be distinguished from old hæmorrhage by centrifuging some of the fluid ; if the supernatant layer be clear, with no sign of hæmolysis, the blood present is probably the result of trauma at the time of the lumbar puncture ; old hæmorrhage is almost always associated with some degree of hæmolysis, and the supernatant fluid is tinged yellow. Further, if the blood-stained cerebro-spinal fluid be collected in successive small amounts in three or four test-tubes, in recent hæmorrhage caused by trauma at the time of the puncture, that in the first one or two tubes will be more deeply blood-stained than in the third or fourth. In old hæmorrhage the blood admixture in all tubes will be about equal.

BACTERIOLOGICAL examination may be made on direct films, or films of the centrifuged deposit ; cultures should also be put up. Pneumococci, meningococci, streptococci, staphylococci, B. Pfeiffer and gonococci may be demonstrated in this way. Tubercle bacilli may often be found in the "spider-web" clot, which forms in the fluid on standing, by staining with the Ziehl-Neelsen method. Guinea-pig inoculation or culture on Lowenstein-Jensen medium may confirm the diagnosis in an obscure case.

The normal CYTOLOGICAL content is two to four mononuclear cells (lymphocytes or endothelial cells) per cu. mm. The total cell count is best made with the Fuchs-Rosenthal counting chamber. It is well to stain the fluid before, as a differential count can then be made at the same time as the total number per cu. mm. is estimated. To ½ c.c. of the cerebro-spinal fluid in a clean dry test-tube add 0·05 c.c. of a 0·2 per cent. solution of toluidin blue or methyl violet. Shake the tube and allow the mixture

to stand for ten minutes. This gives a dilution of ten parts in eleven of the cerebro-spinal fluid, which can be allowed for in the subsequent calculation. The nuclei of the lymphocytes and polymorphonuclear cells take up the stain and can be distinguished by their characteristic shape; the red cells are uninfluenced.

A *differential count* can also be performed on a Leishman stained film of a centrifuged deposit of fluid. A slight *increase in lymphocytes* (5–10 per cu. mm.) in the cerebro-spinal fluid occurs in any form of syphilitic nervous disease after the earlier stages and especially in the more chronic forms, such as tabes or chronic meningeal syphilis. It may also occur in disseminated sclerosis, cerebral tumour and abscess, poliomyelitis, encephalitis lethargica and some forms of polyneuritis. A greater increase in lymphocytes (10–50 per cu. mm.) is common in syphilitic nervous disease of all forms during the onset or the progress of the disease. It also occurs in tuberculous meningitis and in the other conditions mentioned above. A slight increase of both lymphocytes and polymorphonuclear cells (5–10 per cu. mm.) is characteristic of brain abscess or of inflammation of the cranial sinuses with involvement of the dura mater. A larger increase comprising both these types of cell occurs in tuberculous meningitis. Marked polymorphonuclear increase is the rule in all forms of cerebro-spinal meningitis, whether due to pyogenic cocci, the meningococcus, or to any of a variety of bacillary forms, *e.g.*, B. Pfeiffer. In these conditions the causative organism can usually be demonstrated in the fluid.

Chemical Tests.—For these, the clear fluid obtained after centrifuging should be used. *Protein.*—Mestrezat's method is preferable. In this the degree of opalescence produced by the precipitation of the protein in the fluid by trichloracetic acid is compared with that of a series of standards ranging from 0·01 to 0·1 G. per cent. 2 c.c. of the fluid are put into a tube of similar weight and diameter to those used for the standards. 0·3 c.c. of 30 per cent. trichloracetic acid is added; the tube is set aside for twenty to thirty minutes, then shaken up and compared with the standard scale. The normal protein content is 0·02–0·03 G. per cent.

Globulin.—The Nonne-Apelt reaction consists in pouring 1 c.c. of the cerebro-spinal fluid on to the surface of 1 c.c. of a neutral saturated solution of ammonium sulphate. A normal fluid gives a faint opalescent ring at the junction of the two surfaces. A definite opacity indicates an increase of globulin.

Chlorides.—These are estimated against a standard silver nitrate solution, using potassium chromate as an indicator. The chlorides are precipitated as insoluble silver chloride and the indicator, potassium chromate, forms red silver chromate as soon as all the chloride is used up. The silver nitrate solution is made by dissolving 5·814 G. of pure silver nitrate in a litre of distilled water (or 2·5 G. $AgNO_3$ in 430 c.c. of distilled water). This solution keeps indefinitely in a brown bottle. For the test, exactly 2 c.c. of cerebro-spinal fluid is measured by a delivery pipette into 15 or 20 c.c. of distilled water, and 2 drops of 10 per cent. potassium chromate added. The silver nitrate solution is run in from a graduated burette with constant stirring. A permanent change of colour from lemon to orange yellow indicates the end point. Each cubic centimetre of silver solution then used indicates 1 G. of chloride per 1000 c.c. of cerebro-spinal fluid (or 100 mgm. per 100 c.c.). The normal chloride content should fall between 725 and 750 mgm. per 100 c.c. of fluid.

Sugar.—A routine qualitative test is sufficient. One c.c. of the cerebro-spinal fluid is boiled with 0·25 c.c. of Fehling's solution. Normal fluids give a heavy reddish-yellow precipitate which on standing sinks to the bottom of the test-tube, leaving the supernatant fluid pale blue.

Lange's Colloidal Gold Reaction.—Normal cerebro-spinal fluid causes little or no alteration in a colloidal gold solution, but precipitation of the gold may occur with cerebro-spinal fluid from cases of general paralysis of the insane, tabes and some forms of meningitis (Table LXI). Precipitation in these conditions occurs in different dilutions, though these to some extent overlap. Ten dilutions of cerebro-spinal fluid are used in the test, ranging from 1 in 10 up to 1 in 5120. Numbers denote various appearances of fluid depending on degree of gold precipitation.

0 denotes no change (rose-red colour); 1, very slight change to deeper red, scarcely lilac; 2, lilac to purple; 3, deep blue; 4, light blue with purplish precipitate; and 5, complete decolorisation of the top fluid with a heavy bluish precipitate.

A report 0011100000 would be the reading of a normal cerebro-spinal fluid, and indicates that there is no gold precipitation in most of the dilutions, but a very slight one in the dilutions 1 in 40, 1 in 80 and 1 in 160. In the paretic response—typical of general paralysis of the insane—precipitation occurs in the eight strongest dilutions, diminishing gradually, *e.g.*, 5555443200. In the luetic response, seen in tabes and cerebro-spinal syphilis, precipitation occurs in the higher middle dilutions, *e.g.*, 1233210000. A positive curve of either response may be given by some cases of disseminated sclerosis; the Wassermann Reaction here is negative.

§ 921. Bacteriological Examination.—The subjects of serum-therapy and immunity have been dealt with in §§ 519 to 521.

In practice it will be found that in the majority of cases the microscopic examination of direct specimens of pathological material is all that is possible; the cultivation of every micro-organism demands too complicated an amount of apparatus. *Direct films* give invaluable information and present no great difficulty. The apparatus required is of the simplest: a microscope with $\frac{2}{3}$-, $\frac{1}{6}$-, and $\frac{1}{12}$-inch objectives, some slides and cover-glasses, a platinum loop, a Bunsen flame or spirit lamp, a few test-tubes, and some glass jars and bottles. The stains should include methylene blue, methyl violet (6*b*), carbol fuchsin, neutral red, and Leishman's stain, together with absolute alcohol, Gram's iodine solution (1 per cent. iodine in a 2 per cent. aqueous solution of potassium iodide), sulphuric acid (25 per cent.), spirit (70 per cent.), and distilled water. With these it is possible to examine films of pus, sputum and urine, also swabs from throat and nose. They provide valuable means of diagnosing B. tuberculosis, B. anthrax, Streptothrix actinomyces and Vincent's angina, and are useful for B. diphtheriæ, though in this case it is necessary to make cultures as well. The type of pyogenic organism in pus can also be seen, and suitable therapy decided on. For collecting specimens for cultural purposes the following are required: Sterile swabs, test-tubes, culture media, citrate solution, syringes and needles, platinum loop, and capillary pipettes for serum from suspected chancres.

Films for microscopic examination may be made as follows: Take a clean dry slide, place on it a small piece of the material to be examined, and with the platinum loop smear it evenly over a surface the size of a farthing. If the material is very thick it may be mixed with a drop of water to help it to spread. Allow the film to dry in the air, and then heat it over the flame to a degree just sufficient to coagulate the proteins of the material and cause it to stick to the slide; this temperature makes the slide feel uncomfortably hot to the back of the hand. The slide is then left to cool, when it may be stained in one of the following ways: (1) *Methylene Blue* is a simple stain suitable for pus and for throat swabs. Pour a few drops of an aqueous solution of methylene blue on the slide and leave for $\frac{1}{2}$ to 1 minute; wash the slide well in water, and examine with the $\frac{1}{12}$-oil immersion objective. (2) *Gram*, Jensen's modification, is a differential stain for bacteria. Cover the film with 0·5 per cent. methyl violet (6*b*) in water, and leave on for 2 minutes; wash the violet off with Gram's iodine solution and leave the iodine solution on for 2 minutes. Pour off the iodine solution and wash the film in absolute alcohol till no more stain can be washed out; this stage must not exceed 3 minutes. Then wash in distilled water and flood the slide with 1 in 1,000 neutral red in distilled water, and leave this on for $\frac{1}{2}$ to 2 minutes as required; wash in water, blot, and dry. Examine with the $\frac{1}{12}$ objective. Gram-positive organisms are stained purple or black; Gram-negative organisms are stained pink.

(3) *Ziehl-Neelsen Stain for Bacillus Tuberculosis.*—Flood the slide with filtered strong carbol fuchsin and heat gently until steam rises. Allow the preparation to stain for five minutes, heat being applied at intervals to keep the stain hot. Do not allow the stain to evaporate and dry on the slide. Wash with water, and

immerse the slide in 25 per cent. sulphuric acid for a few minutes, and then in 70 per cent. spirit for a similar period. Repeat this process of decolorisation until after washing with water the film is colourless or of a faint pink tinge only. Counterstain with methylene blue or dilute malachite green for 10 to 20 seconds: then wash, blot, dry and examine with the oil immersion objective. The tubercle bacilli stain bright red, whilst cells and other organisms are stained blue or pale green.

(4) *Neisser's Stain for the Diphtheria Bacillus.*—Two solutions are required: (*a*) Neisser's Methylene blue—Methylene blue 1 gram, dissolved in absolute alcohol 20 c.c. and added to glacial acetic acid 50 c.c. in distilled water 950 c.c. (*b*) Bismarck-brown —Bismarck-brown 1 gram, in distilled water 500 c.c. The Neisser's methylene blue is poured on the film and allowed to act for one minute. Wash with water and stain with Bismarck-brown for fifteen seconds. Wash again in water, blot and dry. The diphtheria bacilli are stained brown with a dark granule at one or both poles.

The following organisms are those most likely to be found in direct films:

GRAM-POSITIVE COCCI.

(1) *Staphylococci* in clumps and bunches; (2) *Streptococci* in chains; (3) *Pneumococci* in pairs and short chains. They are lanceolate in shape and may show a capsule. Select a rust-coloured piece of sputum, if obtainable. Confirm by culture. Immediate typing, by observing capsular swelling, can be carried out with Lederle diagnostic antipneumococcic sera, but chemotherapy renders this rarely necessary.

GRAM-POSITIVE BACILLI.

(1) *B. Anthracis* causes Malignant Pustule. Smears should be made from the serum from the ring of vesicles which surrounds the black eschar. The bacillus is very large, usually in pairs or chains. Confirm by culture. (2) *B. diphtheriæ.* Stain also with Methylene blue and Neisser. Confirm by culture: pathogenicity is proved by mouse inoculation. (3) *B. tetani*, the characteristic drum-stick form, the result of a terminal unstained spore, should be sought. Confirm by culture and/or animal inoculation. (4) *Streptothrix actinomyces.* Direct films of material. Search for "sulphur granules," mix as much pus as possible with water, shake and allow to stand for five minutes, pour off the supernatant fluid; repeat until the fluid is clear. The granules sink and so are readily separated from the pus. Collect a granule; crush it on a slide; and stain it with Gram. Gram-positive filaments, with occasionally a Gram-negative terminal club, are seen. (5) *B. tuberculosis.* Though actually Gram-positive, it is best stained by Ziehl-Neelsen's method.

GRAM-NEGATIVE COCCI.

(1) *Gonococcus*, found in pairs, facing each other, kidney-bean shaped. Typically found inside a pus cell. Reject the superficial pus, and make films from the depths of the urethra or cervix. Vaginal or prepucial pus is of little value for diagnosis. (2) *Meningococcus.* In films of cerebro-spinal fluid it resembles the Gonococcus. Purulent cerebro-spinal fluids, in which no organisms are demonstrable in a Gram-stained film, should be cultured; if stored in the warm the cocci will grow, and may be found in direct films twelve to twenty-four hours later. The success of chemotherapy has eliminated, except in special circumstances, the necessity of typing the organisms and the use of specific antiserum. (3) *Micrococcus catarrhalis* is found in sputum; it shows no typical arrangement.

GRAM-NEGATIVE BACILLI.

(1) *B. mallei.* If a Gram-negative bacillus is found in chronic lymphangitis glanders should be considered. Cultures and inoculation will be needed. (2) *B. pestis;* fluid from unbroken buboes or pus may show the characteristic "cocco-bacillus" with its bipolar staining. Cultures and animal inoculation will be required in confirmation. It may also be found in blood and sputum. (3) *B. influenzæ*, a very minute Gram-negative bacillus, best stained with dilute carbol fuchsin. (4) *B. Friedlander*, a stout thick Gram-negative rod with a large capsule. The organism often occurs in diplo-form, or in short chains or groups surrounded by a continuous

capsule. (5) *B. Pertussis*, a small ovoid bacillus which exhibits bi-polar staining and somewhat resembles the influenza bacillus. (6) The organisms of the *Coli-Typhoid-Dysentery* and food poisoning groups are almost identical in appearance. They are easily seen, but only distinguished by careful cultural, fermentative, and serological tests. (7) The organisms of Vincent's Angina, a *spirochæte and a fusiform* bacillus, are usually Gram-negative; the bacillus is occasionally Gram-positive. They stain with methylene blue or Leishman's stain. (8) *B. melitensis.* Originally described as a coccus, this minute organism causes Malta Fever; infection is by goat's milk. A near relation, *B. abortus* (Bang), causes a similar, but usually less severe, condition; infection is through the cow (§ 501). Both organisms may be present in the blood, but cultures or serological tests (see the Widal) are as a rule required.

The **Sputum** is examined bacteriologically for tubercle bacilli, and also for other organisms which may cause a respiratory tract infection, *e.g.*, pneumococcus, influenza bacillus, B. Friedlander, M. catarrhalis, and streptococci. Actinomyces may be demonstrated in actinomycosis of the lung. In whooping-cough, B. pertussis may be isolated from a pharyngeal swab, or by allowing the patient to cough on to a plate of Bordet-Gengou medium, which is subsequently incubated.

A **False Membrane** occurring on the throat or palate must be examined for the **diphtheria bacillus.** Taking care not to touch the lips, cheek, or mouth, touch the suspected patch with a sterilised swab, or remove a piece of the membrane with a platinum loop or forceps. Transfer some of this to a sterile test-tube, or at once inoculate a blood serum slope or tellurite medium. Spread some on a microscope slide, fix and stain with methylene blue, Gram's or Neisser's stain. Frequently so many micro-organisms are present that it is impossible to make out satisfactorily the diphtheria bacillus. Then cultures must be made on Löeffler's coagulated serum or tellurite medium. The inoculated culture tube should at once be sent to a laboratory, where it is incubated and examined. After twelve hours there is ample growth for this purpose.

Pleural Effusion.—See examination of pathological fluids.

Pus obtained from any part of the body should be stained by Gram's method and examined microscopically. Further films should be stained for tubercle bacilli (*q.v.*) and swabs should be taken for cultivation.

It is useless to examine films of the **stools** microscopically for any micro-organism except the tubercle bacillus. Cultural methods must be used. Microscopical examinations may show the amœbæ of dysentery or their cysts, also the ova of various worms and flukes. Undigested food particles, muscle-fibres, fats, fatty acids, soaps and starch granules may be seen (§ 303).

The ENTAMŒBA HISTOLYTICA and its cysts must be distinguished from the non-pathogenic ENTAMŒBA COLI. The cytoplasm of the vegetative form of *E. coli* is not differentiated into endo- and ectoplasm, amœboid movement is sluggish and no ingested red cells are seen in the cytoplasm. The cysts usually contain eight nuclei.

The **Amœba of Dysentery** (*E. histolytica*) is spherical, sometimes of a pale greenish colour refracting the light strongly. It is 30–40 μ in diameter, has a granular endoplasm and a clear ectoplasm, and is actively motile. Ingested red blood-cells may be seen in the cytoplasm when fresh. The fresh stool should be examined for amœbæ while the discharges are still warm and alkaline. The glass slides, cover-glass, and the

microscopic stage should be warmed (by standing the microscope in the incubator before use). A drop of mucus from the stools, or a scraping from the wall of a tropical abscess, or from the ulcerated mucous membrane obtained via a sigmoidoscope, is diluted with warm saline solution and placed on the slide. The amœba will then be seen actively moving. Often no active amœbæ are present in a stool, yet the patient may be passing numbers of amœbic cysts; these are round refractive bodies containing four nuclei, and stain faintly with iodine (§ 304).

The **urine** may contain the following organisms, demonstrable bacteriologically in infection of the urinary tract—B. coli, B. proteus, staphylococci (aureus and albus types), streptococci (usually fæcalis type), gonococci, typhoid or paratyphoid (carrier state), and tubercle bacilli.

Blood.—*Direct examination* of blood films may be necessary for the discovery of such blood parasites as malaria, trypanosoma, anthrax, filaria, the spirochæte of relapsing fever, kala-azar, etc. *Cultural examination* is required in septicæmia, malignant endocarditis, typhoid fever, etc. For convenience, 10 c.c. of blood, taken from a vein with a dry sterile syringe, is added to 0·5 c.c. of sterile 5 per cent. sodium citrate solution in a sterile test-tube. After mixing gently, 5 c.c., 2 c.c., 1 c.c., 5 drops and 1 drop of this citrated blood are inoculated into separate tubes of glucose-broth. The tubes are incubated at 37° C. and examined for growth at the end of 24 hours, 48 hours and 7 days. Especially valuable in infective endocarditis is blood culture with pour plates. To ½-1 c.c. of blood in a sterile Petri dish is added 6-8 c.c. of nutrient agar, cooled to 45° C. after melting: these are mixed by gentle agitation and after allowing to set, are incubated. When typhoid or paratyphoid is suspected, an additional culture should be made of 3 c.c. of blood in a tube of bile broth or sterilised ox bile; subculture on to MacConkey's or litmus lactose agar medium is made after 24 hours' incubation. If undulant fever be a possibility a parallel set of glucose-broth cultures should be incubated in an atmosphere of 10 per cent. carbon dioxide. It may, in some cases, be necessary to incubate cultures anærobically. Relative anærobic conditions are obtained by boiling and cooling the tubes of glucose-broth prior to adding the blood and then covering the surface with a layer of sterile liquid paraffin.

§ **922. Widal's Serum Reaction** illustrates the phenomenon of immunity (§ 519). An infecting organism acts in the human body as an antigen, a substance which stimulates the formation of antibodies. These antibodies are of various types; among them are agglutinins which cause the infecting organisms to mass together in clumps, *i.e.*, to agglutinate. The test, originally described for typhoid fever, is now applied to many other bacterial infections, notably the paratyphoid fevers, the dysenteries, Malta fever, food poisonings and infections with B. abortus. A somewhat similar technique is used for typing the pneumococcus and meningococcus. In typhoid the reaction may occur any time after the first week, rarely by the third day, and persists for several years (§ 493). Inoculated persons may give a positive result for many years. In doubtful cases the strength of the reaction is estimated, and this is repeated after a week. With an active infection the strength of the reaction will increase, while an old case, a carrier, or an inoculated subject will show no change.

Dreyer's modification (a macroscopic agglutination) is the best means of performing the test. Serum obtained by veno-puncture from the patient is diluted 1 in 10,

using a Dreyer's dropping pipette. Into a small metal rack, provided with three rows of five small holes, are placed the special agglutination tubes with pointed ends. With the Dreyer standard pipette ten drops of the diluted serum (1–10) are placed in the first tubes of each row, five in the second, two in the third and one in the fourth. Normal saline is added as follows—five drops to the second tube in each row, and eight, nine and ten drops to the third, fourth and fifth tubes in each respectively. Fifteen drops of dead B. Typhoid emulsion, B. Paratyphoid A and B emulsions are added to each of the tubes of the first, second and third rows respectively, and the contents mixed by inversion. The last tube in each row contains no serum and acts as a control. The resulting dilutions of serum in the other tubes are 1–25, 1–50, 1–125, and 1–250. The rack is placed in a water bath at 55° C. for two hours, and then examined. Complete agglutination is indicated when the bacilli form a white flocculent precipitate at the apex of a tube; partial agglutination, by fine flocculi in the tube just visible to the naked eye but without sedimentation. The result is expressed according to the highest dilution (titre) of the serum in which agglutination results. In a positive case it may be necessary to carry the dilution of the serum to a much higher figure than that in the first test before no or only partial agglutination is obtained.

(The Standard Agglutinable Emulsions are obtained from the Department of Pathology, University of Oxford.)

§ 923. **Treponema Pallidum (Spirochæta Pallida).**—*Precautions:* The operator is advised to wear rubber gloves in all cases. Serum should be obtained *from below the surface* and, so far as possible, contamination with the surface organisms which may include spirochætes other than *Spirochæta pallida* avoided. The presence of blood in the specimen renders the examination for spirochætes more difficult. The specimen should be obtained from the margin of the lesion or sore, especially if it is ulcerated. If a local antiseptic has been applied to the lesion, it may be impossible to discover the spirochætes until a wet dressing of lint or plain gauze soaked in boiled water (or N. saline) has been applied to the sore for three days prior to collection of the specimen of serum.

Procedure.—(*a*) Cleanse the sore and its immediate surroundings with a swab of non-medicated gauze or lint moistened with warm water or with N. saline. (*b*) Apply a pad of non-medicated lint wrung out in very hot water to the surface of the sore and keep in contact for about a minute. (*c*) Now apply a pad of plain lint moistened with absolute alcohol or methylated spirit, and keep in contact for about half a minute. (This often causes some smarting, but is less painful to the patient than scraping with an instrument.) (*d*) On removing the spirit pad the surface of the sore looks dry and glazed. Now squeeze the base of the sore, when clear serum will exude from the surface: collect a few drops of this into the capillary tube provided for the purpose. (*e*) Hold upwards the end of the capillary tube furthest removed from the serum, and seal, allowing the end just to touch the flame. The serum will then travel up the tube towards the sealed end, and the other end can be sealed without heating the serum in the tube. Send the specimen to the laboratory as quickly as possible. A film of this serum is made. The spirochæte pallida may be examined by the method of "dark ground illumination" or by staining methods. A description of the organism is given under Syphilis (§ 552, and Fig. 133).

§ 924. The **Wassermann Reaction** can be carried out on any body fluid—most frequently with blood, less often with cerebro-spinal fluid. The test depends upon the fixation or adsorption of complement by a factor present in syphilitic serum, as follows: Syphilitic serum + antigen + complement, results in the adsorption of complement. *Syphilitic serum* contains an immune thermostable substance, upon which depends the intensity of the positive Wassermann reaction. *Antigen* consists of a mixture of alcoholic heart extract and a solution of cholesterol. It was formerly thought necessary to employ a specific antigen, in the form of an extract of syphilitic liver, but later researches have shown that the test is equally reliable with a non-specific antigen. *Complement* is present in varying amounts in all fresh serum. In order to supply it

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§ 924. The **Wassermann Reaction** can be carried out on any body fluid—most frequently with blood, less often with cerebro-spinal fluid. The test depends upon the fixation or adsorption of complement by a factor present in syphilitic serum, as follows: Syphilitic serum + antigen + complement, results in the adsorption of complement. *Syphilitic serum* contains an immune thermostable substance, upon which depends the intensity of the positive Wassermann reaction. *Antigen* consists of a mixture of alcoholic heart extract and a solution of cholesterol. It was formerly thought necessary to employ a specific antigen, in the form of an extract of syphilitic liver, but later researches have shown that the test is equally reliable with a non-specific antigen. *Complement* is present in varying amounts in all fresh serum. In order to supply it

in known quantity, the complement in the test serum is destroyed by heating, and fresh complement is added in the form of guinea-pig's serum. The adsorption of complement, which occurs when these three substances interact at 37° C., is believed to be a colloidal phenomenon, possibly resulting in a fine precipitate, not discernible by the eye. To make the reaction visible, the method of using sensitised sheep cells has been devised. When the red blood corpuscles of a sheep are injected into a rabbit, an anti-substance is formed in the blood of the rabbit which is capable of hæmolysing the sheep cells. This substance is called hæmolytic amboceptor. When sheep cells, sensitised in this way, are added to a serum in which complement has been destroyed by heating, no hæmolysis takes place, and the fluid becomes, on shaking, an opaque, pinkish colour. If, on the other hand, fresh complement is added, the cells become hæmolysed, resulting in a clear red fluid.

Now, as stated above, syphilitic serum mixed with antigen and with complement, causes the complement to be adsorbed, so that none is left to enable the amboceptor to hæmolyse the sheep cells. In the case of normal serum, however, there is no adsorption of complement, with the result that the fresh complement added is free to produce hæmolysis. An opaque, pinkish fluid, therefore, indicates that the specific syphilitic immune substance was present, and that the test is positive, while a clear red fluid indicates that it was absent and that the test is negative. By altering the nature of the antigen, tests for other antibodies (*e.g.*, gonococcal, hydatid, tuberculous) can be carried out.

Interpretation of the Wassermann Reaction.—(1) Blood. It is often helpful for the practitioner to consult the pathologist, for there are borderline cases where the exact position is more easily conveyed by word of mouth than by written reports.

As a general rule the following terms are used: Strong Positive, double plus: this may be taken as proof of syphilitic infection. Positive, one plus: a valuable contributory sign when the diagnosis is in doubt; if found repeatedly it may be accepted as proof of syphilitic infection. Weak, or a Doubtful Positive, or a Doubtful result, plus over minus. The test should be repeated, often after a provocative dose of N.A.B., and a thorough clinical examination of the patient made. A diagnosis of syphilis should never be made on such a result alone. Negative, Complete Negative, Minus. A patient with active secondary or tertiary syphilis seldom gives a negative Wassermann. Early cases frequently give this result. In Tabes and G.P.I. a Positive is usual but not constant. (For *Fallacies,* see page 715.)

(2) Cerebro-spinal fluid. A positive result is proof of central nervous syphilis; a negative does not exclude it. In G.P.I. a positive is the rule. In Tabes, especially during remissions, negatives are often obtained. In meningo-vascular syphilis a high proportion of positives is obtained.

Flocculation Tests.—Sachs-Gorgi, Sigma, Kahn, etc. These tests differ from the Wassermann in that only syphilitic antigen and patients' serum are used. There is direct union of antigen and antibody without the need for complement and with a visible result. Suitable quantities of serum and heart-cholesterol extract are mixed and kept at a fixed temperature; after a certain time flocculation is observed in the positive tubes. The significance and interpretation may be taken as identical with those of the Wassermann, when dealing with blood; with C.S.F. the tests are less delicate.

Paul-Bunnell Reaction.—This is a diagnostic test for glandular fever dependent on the discovery that the blood in this affection contains heterophil antibodies in the form of an agglutinin for sheep's red cells. Normal serum may possess agglutinin to a titre of 1 in 16: in glandular fever the titre is usually 1 in 64 or higher by the end of the first week, and remains high during the active phase of the disease. Sometimes it is only transient, occasionally delayed, and rarely absent. This latter suggests that glandular fever is caused by different viruses some of which do not give the typical serological reactions. The patient's serum is inactivated by heating for fifteen minutes at 55° C. and is then put up in serial dilutions, to each of which is added a known volume of a 2 per cent. suspension of freshly washed sheep's cells.

The test is read after incubation in a water bath or incubator for one hour at 37° C. followed by overnight in a refrigerator at 2–4° C. A raised titre occurs in serum sickness, but other than this the reaction is specific for glandular fever.

§ 925. **Basal Metabolism.**—The determination of the basal metabolic rate is of value in diagnosis, prognosis and in estimating progress at various stages of treatment. In cases of exophthalmic goitre it establishes with certainty the degree of toxicity due to the thyroid condition. In cases of hypo- or hyper-thyroidism it is of value as a guide to treatment. It may be defined as a measure of the capacity of the individual to consume oxygen under certain definite conditions which by precise adjustment permit of comparison with a standard normal.

There are many methods of determining the basal metabolic rate. The more elaborate include gas analysis and need to be conducted in hospital and in close proximity to a well-equipped laboratory. These are more accurate, as all factors are taken into consideration and only highly-trained technicians are employed. For general clinical purposes, a method carried out by any physician and at any bedside is obviously of greater practical value. Such a method is offered by several types of apparatus (*e.g.*, the portable Benedict apparatus). The precautions necessary are :—(*a*) That the patient should be fasting and should have been resting in bed for the preceding twelve or fourteen hours ; (*b*) That he should practise for a day or two the use of the nose-piece and mouth-piece. These are at first uncomfortable ; but after a little practice he will grow thoroughly accustomed both to the method of breathing and to the strangeness of the mechanical devices in the mouth and nose. This precaution is of the first importance—for mental or physical unrest at the time of the test will materially impair its accuracy.

The apparatus consists of a cylindrical spirometer with the necessary attachments to the mouth-piece and for administering the supply of oxygen. It also contains soda lime, through which both the inspired oxygen and the expired air pass—thus removing all the CO_2. The readings on the scale represent accurately the amount of oxygen consumed. These readings are interpreted by means of a table which accompanies the apparatus and which makes allowance for variations of temperature, barometric pressure, height, weight, age and sex. One is then able to ascertain the percentage of oxygen consumed per minute above or below the normal. This is expressed as the B.M.R. (basal metabolic rate), plus or minus, as the case may be.

§ 926. The **Zondek-Aschheim Pregnancy Test** has proved reliable for the diagnosis of pregnancy as early as the fifth week, when clinical signs are still inconclusive. The test depends upon the fact that the urine of pregnant women contains an anterior pituitary lobe hormone which induces the formation of hæmorrhagic Graafian follicles and of corpora lutea in the ovaries of sexually immature female mice. The specimen of urine used should be an early morning one and collected not earlier than one week after the first missed menstrual period. In hydatidiform mole and chorion epithelioma the amount of hormone excreted is greatly increased compared with that in a normal pregnancy. The inoculation of urine diluted 1 in 100 or more will still produce the characteristic ovarian changes and this is of valuable diagnostic aid.

Five immature female mice, twenty-one days old on the date of commencing the test, are injected subcutaneously over three days with six doses of urine, each of 0·3 c.c. The animals are killed on the fifth day, and the ovaries examined macroscopically and if necessary microscopically. Characteristic changes in any one of the animals indicates a positive result.

The **Friedman Pregnancy Test** is a modification of the Zondek-Aschheim test and is carried out on a juvenile female rabbit, or one that has been segregated for three months. Ten c.c. of the urine to be tested (an early morning specimen) are injected intravenously into a marginal ear vein and the animal killed after 48 hours, laparotomy performed and the ovaries examined. A positive result is indicated by the presence of corpora hæmorrhagica in the ovaries. The test becomes positive on the seventh to the tenth day after the first missed menstrual period and is extremely accurate, although it does not, in using a single animal, allow for individual variation in response.

The Xenopus Pregnancy Test (Toad ovulation test) depends on the production of ovulation in female Xenopus lævis—an African toad—following the injection of the anterior-putuitary-like hormone in the urine of pregnant women. Three or four toads are inoculated with 2 c.c. of urine into the lymph sac under the dorsal skin, and a positive result is indicated by the shedding of eggs in 12 to 24 hours. The reliability of the test does not differ from that of the Zondek-Aschheim or the Friedman reaction, and the animals need not be killed to obtain the result. A related test is by injection of 10 c.c. of a patient's untreated urine into the dorsal lymph sac of the adult male toad *Bufo arenarum* Hensel indigenous to South America. In 2–4 hours masses of spermatozoa in the toad's urine indicate a positive result.

§ **927. Blood Sedimentation Rate.**—The sedimentation rate of the red cells was first studied by Fahræus, but there have been many subsequent modifications in technique. *Method.*—The Westergren method is the one most commonly used. 1·6 c.c. of blood is mixed with 0·4 c.c. of 3·8 per cent. sodium citrate solution, and some of the mixture drawn up into a standard Westergren tube; the latter is 2·5 mm. in diameter and closely resembles a 1 c.c. pipette, but is calibrated in mms. of length. The zero mark is exactly 200 mm. from the point, and the blood is drawn up to this mark. The tube is then set upright in a special stand and the red corpuscles begin to settle down, leaving a clear supernatant plasma. At the end of one hour, the result is read as the distance sedimented in mms. by the top of the red cell column. In men the normal range is 3–5 mm. and in women and children 4–7 mm. An increased sedimentation rate is present in pregnant women after the third or fourth month, in localised acute inflammations, in rheumatoid arthritis, in active tuberculosis and in active rheumatic disease of children (rheumatic fever, carditis, etc.). In tuberculosis, the degree of sedimentation is of some value in prognosis as it increases with the activity of the disease; in rheumatic disease of childhood, an increased rate points to a latent activity even in the absence of physical signs, and indicates continued rest to limit cardiac damage. The sedimentation rate is apparently dependent on the ratio of albumen, globulin and fibrinogen in the plasma. The test is of more value in prognosis than diagnosis, and in all cases should be interpreted in conjunction with other clinical and laboratory investigations.

THE VITAMINS

§ **928.** Vitamins are present in small amounts, measurable in standardised units, in plant and animal tissues, and are essential for the transformation of energy and the regulation of metabolism. They may be divided into three groups :—

(I) Those which have been shown to be needed by man—vitamin A, vitamin B_1, four components of the vitamin B_2 complex (nicotinic amide, riboflavin, folic acid and the most recently discovered B_{12}); vitamins C, D and K, with their different forms and modifications.

(II) Those whose significance for human nutrition is probable but not certain—two factors of the B_2 complex (biotin or vitamin H, and choline) and vitamins E, F, and P.

(III) Vitamins needed for experimental animals—four factors of the B_2 complex (B_6, pyridoxin, pantothenic acid, inositol, and *p*-amino benzoic acid).

GROUP I

(1) **Vitamin A,** (" anti-xerophthalmic ") includes vitamin A, or axerophthol, the principal provitamin, β-carotene, and several other forms of vitamin A including vitamin A_2 and active carotenoids. The first recognisable symptoms of vitamin A deficiency are cessation of growth, and night blindness; the light adaptation test is used as a measure of the vitamin A requirement. Xerophthalmia, increased susceptibility to infection, and pathological changes in epithelial cells, leading to a keratinised papular skin eruption, are associated with vitamin A deficiency.

Vitamin A is prepared in crystalline form, from natural sources and synthetically. Its chief sources are animal fats and oils, especially halibut and cod-liver oils, liver, eggs and dairy produce. It can also be manufactured in the body from its precursor carotene, widely distributed in yellow root vegetables, green leaves, coloured fruits and seeds. Vitamin A_2 is present in freshwater fish.

The daily adult requirement of vitamin A is 3,000 I.U. This is represented by 1–2 teaspoonfuls of cod-liver oil, 1–2 quarts of milk, or 2–3 ozs. of liver. Lactating women whose diet is suspected of being deficient in vitamin A need a daily addition of about 6,000 I.U.

(2) **Vitamin B Group** includes the heat labile vitamin B_1 and the heat stable vitamin B_2 complex.

Vitamin B_1 (aneurin or thiamin—" anti beri-beri " or " anti-polyneuritic "). Deficiency of vitamin B_1 is associated with human beri-beri, especially when the diet consists chiefly of milled rice. Vitamin B_1 is directly concerned with intermediary carbohydrate metabolism, especially in relation to the oxidation of pyruvic acid in the brain. Excess of carbohydrate in the diet, pregnancy and lactation, increase the requirement for vitamin B_1. Deficiency appears to be the underlying factor in various forms of nutritional polyneuritis; it is also said to be associated with gastro-intestinal disturbances (anorexia, glossitis, achlorhydria) and with circulatory disorders (dyspnœa and palpitation on exertion, tachycardia, and œdema). Its chief sources are yeast and cereals, particularly the germ of whole wheat. The use of highly milled cereals, from which the germ has been removed, will markedly reduce the vitamin B_1 content of the diet. It is present to a smaller extent in some green vegetables, potatoes and in milk. The average daily requirement is about 300 I.U.; white bread contains about 30 units per 100 gms.; the national loaf 80–85 units; dried yeast 1,000–2,000 units; egg-yolk 100 units.

Vitamin B_2 Complex

(*a*) **Nicotinic amide** (niacin amide or P.P. factor) was at first identified with the pellagra preventive vitamin; pellagra is now considered to be a multiple deficiency disease, and shortage of nicotinic amide only one causal factor. Apart from pellagra it has been used empirically in cases of stomatitis, gastro-intestinal disturbance and psychoses resulting from

deficient nutrition or toxæmia: also in conjunction with sulphonamide therapy to reduce symptoms of intolerance. Its chief sources are liver, eggs, salmon, whole cereal and yeast. Daily requirement about 10–12 mgm. Therapeutic dose 50 mgm., t.i.d.

(*b*) **Riboflavin** (formerly called lactoflavin). Its deficiency syndrome includes seborrhœic dermatitis of the face, fissures at the angles of the mouth, glossitis, and ocular lesions (conjunctivitis, corneal opacities, keratitis). Chief sources are yeast, milk, white of egg, fish roe, liver, kidney, and leafy vegetables. Daily requirement, 1–2 mgm.; therapeutic dose 5 mgm. daily.

(*c*) **Folic Acid** (lactobacillus casei factor; pteroylglutamic acid) obtained in nearly pure form from spinach, has now been synthesised. It produces a reticulocyte response in pernicious anæmia and regeneration of red-blood cells in macrocytic anæmias, but is apparently ineffective in controlling subacute combined degeneration. It has been found effective in the treatment of sprue. Therapeutic dose 5–100 mgm. daily by mouth or 15 mgm. parenterally.

(*d*) **Vitamin B_{12}** obtained from purified liver fractions, has shown great hæmopoietic activity in pernicious anæmia in extremely small doses. It is not yet certain whether this material is actually the anti-pernicious anæmia liver principle. Suggested curative dose 7·5 μg.

(3) **Vitamin C** (ascorbic acid; cevitamic acid; "anti-scorbutic"). Deficiency is followed by scurvy in man and animals. One of the first signs of sub-clinical scurvy can be detected by the skin capillary fragility test. It is believed that patients with various infections, including rheumatism, require large amounts of vitamin C if normal levels are to be maintained in the blood and excreted in the urine, where its amount can be determined by titration with 2, 6-dichlorphenolindophenol. There is evidence that it is necessary for wound healing: it has been used in treating allergic conditions, in Addison's disease, hyperthyroidism, and as a detoxicating agent in various poisonings by metals and chemicals and intolerance to certain drugs. Its chief sources are fruits and vegetables, especially paprika, followed closely by lemons, oranges and tomatoes. Vitamin C is now known to be identical with hexuronic or ascorbic acid, derived from the adrenal cortex. Since Vitamin C is easily destroyed by cooking, especially in the presence of alkalies, an antiscorbutic diet should contain a large proportion of raw fruits and vegetables: canned foods do however contain vitamin C. Daily requirement 30 mgm.

(4) **Vitamin D** ("anti-rachitic"). The specific function of vitamin D is the control of the calcium-phosphorus metabolism, its action being to increase the absorption of calcium and phosphorus or to diminish their intestinal excretion. Rickets and osteomalacia and defective calcification of the teeth are the chief disorders associated with its deficiency. It is also related to the acid-base value of the diet, to the parathyroid hormone, and to the serum phosphatase. It has recently been used in the treatment

of lupus vulgaris. Various forms of vitamin D (D_2, D_3, D_4, D_5, and D_6 are now described, but the only two of practical significance are :—

Vitamin D_2 (calciferol) which is synthetic and is formed from ergosterol, found also in certain irradiated plants and fungi. Daily requirement for children 500–1,500 I.U., for nursing and expectant mothers 1,500–2,000 I.U. Curative dose 1,500–3,000 I.U.

Vitamin D_3 is the naturally occurring vitamin which can be found in foodstuffs, or derived from various precursors, provitamins D, belonging to the sterol compounds, the chemical constitution of some of which is still obscure. Its elaboration takes place on the surface of the body under the influence of ultra-violet light. Vitamin D_3 is less toxic than D_2 and probably has greater anti-rachitic power for children.

The chief food sources of vitamin D are milk, butter, eggs and green vegetables, but many foodstuffs otherwise devoid of rickets-preventing activity, can be rendered anti-rachitic by exposure to ultra-violet rays. For therapeutic purposes the richest sources are halibut liver oil, cod-liver oil, and various preparations of irradiated ergosterol, especially calciferol which, weight for weight, is 300,000 times more potent than cod-liver oil. Overdosage of vitamin D in the form of concentrates or of cod-liver oil may lead to hypervitaminosis, characterised by anorexia, loss of weight, diarrhœa, and pathological calcification of the arteries, heart and internal organs.

(5) **Vitamin K**—" anti-hæmorrhagic "—includes vitamins K_1 and K_2 and other methyl naphthaquinone derivatives. Deficiency causes lack of prothombin in the blood. Vitamin K is of clinical value in the prevention and treatment of hæmorrhagic disease of the new-born, in hæmorrhage associated with obstructive jaundice and liver damage, and in the hypoprothrombinæmia associated with sprue and steatorrhœa. The chief sources of vitamin K_1 are green vegetables, especially spinach and alfalfa, and hog-liver fat. Fruits and other vegetables are poor sources. The synthetic analogue 2-methyl-1, 4-naphthaquinone is chiefly used in medicine. Prophylactic doses may be given to the mother (1 mgm.) or the infant (10 μg.). Curative dose 1–4 mgm. daily. Vitamin K_2 is synthesised by most bacteria, especially those of the intestinal tract.

GROUP II

(1) **Vitamin H** or biotin (" skin factor ") protects experimental animals from skin lesions associated with a diet of raw egg-white which contains an " antivitamin." Its association with human deficiency is still uncertain.

(2) **Choline** deficiency in animals is associated with fatty liver, and hæmorrhages in the kidney. Its use in human beings has been proposed in cases of a negative nitrogen balance where a high protein diet or methionine would be applicable.

(3) **Vitamin E,** " anti-sterility " (α-, β- and γ-tocopherol). The relation of vitamin E to human fertility and to muscular dystrophy is still disputed.

It is stated to have been beneficial in cases of habitual and threatened abortion, and in the anæmia of pregnancy. In animals its deficiency leads to sterility—in the female as a result of intra-uterine death and resorption of the fœtus, in the male through degeneration of the germ cells of the testes. Its richest source is wheat germ ; milk and animal tissues contain a small amount. Cod-liver oil is a poor source. The recommended therapeutic dose is 6 mgm. per day of tocopherol.

(4) **Vitamin F** (in linoleic and other essential unsaturated fatty acids). Deficiency in rats causes lesions of the tail and kidneys. Its use in human beings has been suggested for eczematous conditions.

(5) **Vitamin P** (citrin, " permeability vitamin ") is related to vitamin C and follows its distribution in fresh fruits and vegetables. It is considered to regulate capillary permeability generally, and to modify the pathological picture of scurvy. It has been used clinically in doses of 50–150 mgm. in hæmorrhagic conditions of non-specific character such as bleeding in the kidney and stomach.

GROUP III

(1) **Pyridoxin** (vitamin B_6), though its necessity for human beings has not been proved, has been given with some success in cases of pellagra which have not responded to vitamins B_1, B_2, and nicotinic amide ; and to cases of pseudo-hypertrophic muscular dystrophy and myasthenia and of pernicious anæmia in relapse. It is present chiefly in yeast and rice polishings, and to some extent in molasses, fish and liver.

(2) **Pantothenic acid** (" chicken pellagra " factor). Deficiency causes dermatitis in birds and rats and growth failure and other lesions in rats. Its significance in human beings is obscure.

(3) **Inositol** (Bios I). Deficiency in animals is said to cause alopecia. In man it is thought to be concerned with the synthetic action of intestinal bacteria on essential nutrients.

(4) **Para-aminobenzoic acid** (PABA) extracted from yeast, is sometimes called " sulphonamide antivitamin," because it can inhibit the antibacterial effect of the sulphonamide drugs. It has been suggested that PABA may have an anti-infective action in animals.

FORMULÆ OF USEFUL PRESCRIPTIONS

(referred to as F. in the text)

The proportions given are those for one adult dose unless otherwise stated. Imperial Weights and Measures are used in the Formulæ. The metric equivalents given in brackets represent grammes for solids and millilitres for liquids.

(1) Balneum Alkalinum.

Add two large handfuls (8 ozs. or 226 gms.) of common washing soda to 30 gallons (136 litres) of water at 95° F. The patient remains twenty minutes in first bath, and the time is gradually increased up to forty-five minutes. Put to bed in blankets.

Valuable for chronic rheumatism—daily for six weeks. At first the pains are increased. Also useful for chronic eczema.

(2) Balneum Cyllin, vel Picis.

Cyllin ♏ 30 120 (1·8-7·1) *or* Liquor Carbonis Deterg. ♏ 60–240 (3·6–14·2) in 20 to 30 gallons (90 to 136 litres) of water, well stirred.

Useful for pruritus, prurigo, chronic eczema, and all itching affections.

(15) Garg. Phenolis c̄ Cocaina. D.D.A.

Phenol	♏ 60 (4)
Cocainæ Hydrochlor. .	gr. 8 (0·5)
Glycerini Boracis .	fl. oz. ½ (14·2)
Aquam Rosæ . .	ad fl. oz. 12 (341)

For acute pharyngitis and laryngitis.

(16) Gargarisma Acidi Tannici.

Glycerini Acidi Tannici	♏ 60 (3·6)
Aquam	ad fl. oz. 1 (28·4)

For relaxed throat and to check bleeding after tonsillectomy.

(17) Gargarisma Boracis Compositum.

Pulveris Aluminis .	
Pulveris Boracis . .	āā gr. 7½ (0·5)
Tincturæ Myrrhæ .	♏ 5 (0·3)
Mellis	gr. 10 (0·6)
Aquam	ad fl. oz. 1 (28·4)

(18) Gargarisma Chlorini.

Potassii Chloratis . . .	gr. 120 (8)
Acidi Hydrochlorici Fortioris.	♏ 60 (3·6)

Cork and set aside for five or ten minutes, then add—

Glycerini . . .	fl. oz. ½ (14·2)
Aquam	ad oz. 12 (341)

To be freshly prepared. A very prompt and efficacious remedy for scarlatinal and diphtheritic sore throat, and follicular tonsillitis. For children it should be applied with a brush every two hours.

(19) Gargarisma Potassii Chloratis.

Potassii Chloratis .	
Aluminis . . .	āā gr. 90 (6)
Aquam . . .	ad fl. oz. 10 (284)

(30) Linctus Communis.

Oxymellis Scillæ . .	♏ 120 (7·1)
Syrupi Tolutani . .	♏ 120 (7·1)
Syr. Pruni Virg. . .	♏ 120 (7·1)
Aquam Destillatam .	ad fl. oz. 1 (28·4)

Dose.—A teaspoonful for bronchitic cough.

LOTIONES.

(36) Lotio Calaminæ.

Calaminæ	gr. 20 (1·3)
Zinci Oxidi	gr. 20 (1·3)
Glycerin	♏ 30 (1·8)
Liq. Calcis	♏ 180 (10·6)
Aquam Destillatam.	ad fl. oz. 1 (28·4)

For erythema and acute eczema.

The 1932 British Pharmacopœia recommends that instead of the usual symbols employed in prescriptions (ʒi. to represent 60 grains, and also to represent 1 fluid drachm; and ℥i. to represent sometimes 480 grains, sometimes 437·5 grains, and also to represent 1 fluid ounce), that the prescribers should use instead, when the Imperial system is employed, gr., fl. oz., and ♏.; and that in order to avoid the possibility of confusion between gramme and grain, the symbol G. should be used in prescriptions as the contraction for gramme; also, that the quantities should be written in Arabic numbers.

(42) Lotio Plumbi cum Zinco.

Liq. Plumbi Subacet. Dil. . . .	♏ 60 (3·6)
Zinci Oxidi . . .	gr. 20 (1·3)
Glycerini . . .	♏ 30 (1·8)
Aquam . . .	ad fl. oz. 1 (28·4)

Invaluable for acute eczema.

(51) Mistura Salina Laxans.

Sodii Bicarbonatis .	gr. 15 (1)
Sodii Chloridi . .	gr. 5 (0·3)
Sodii Sulphatis . .	gr. 30 (2)
Magnesii Sulphatis .	gr. 60 (4)
Aquam Menthæ Piperitæ . . .	ad fl. oz. 1 (28·4)

A morning purgative draught for plethora, obesity, gout and chronic rheumatism.

(53) Mistura Diaphoretica.

Spiritûs Ætheris Nitrosi	♏ 30 (1·8)
Liq. Ammonii Acetat. .	♏ 120 (7·1)
Aquam Camphoræ .	ad fl. oz. 1 (28·4)

Diaphoretic and febrifuge.

(54) Mistura Digitalis Co.

Tincturæ Digitalis .	♏ 5 (0·3)
Ammonii Carbonatis .	gr. 3 (0·2)
Potassii Nitratis . .	gr. 5 (0·3)
Tincturæ Nucis Vomicæ	♏ 5 (0·3)
Aquam Chloroformi .	ad fl. oz. 1 (28·4)

For cardiac disease.

(55) Mistura Diuretica.

Potassii Acetatis . .	gr. 15 (1)
Spiritûs Ætheris Nitrosi .	♏ 15 (0·9)
Spiritûs Juniperi .	♏ 30 (1·8)
Decoctum Scoparii .	ad fl. oz. 1 (28·4)

(56) Mistura Ether Ammon.

Sp. Ammoniæ Aromat.	♏ 20 (1·2)
Spiritûs Etheris . .	♏ 20 (1·2)
Spiritûs Chloroformi .	♏ 20 (1·2)
Aquam	ad fl. oz. 1 (28·4)

For cardiac failure. More efficacious if accompanied by hypodermic injection of Liq. Strychn. ♏ ii.

(57) Mistura Expectorans.

Ammonii Carbonatis .	gr. 5 (0·3)
Tincturæ Scillæ . .	♏ 15 (0·9)
Spiritûs Etheris . .	♏ 15 (0·9)
Tincturæ Strophanthi .	♏ 3 (0·2)
Infusum Senegæ . .	ad fl. oz. 1 (28·4)

For acute bronchitis in the second stage.

(64) Mistura Olei Ricini.

Olei Ricini	fl. oz. ½ (14)
Mucilag. Acaciæ . .	fl. oz. ½ (14)
Syrup. Zingiberis . .	fl. oz. ¼ (7)
Aquæ Menthæ Pip. . .	fl. oz. ¾ (21)

♏ 60 every hour for diarrhœa and unhealthy stools in children.

(66) Mistura Stomachica.

Magnesii Carbonatis .	gr. 10 (0·6)
Sodii Bicarbonatis .	gr. 15 (1)
Phenol pur. . . .	♏ 1 (0·06)
Tincturæ Rhei Compositæ . . .	♏ 15 (0·9)
Infusum Calumbæ .	ad fl. oz. 1 (28·4)

Tea-drinker's dyspepsia and pyrosis.

(67) Mistura Strychninæ.

Liq. Strych. Hydrochlor. . . .	♏ 3 (0·2)
Acidi Nitro-Hydrochlorici Diluti . .	♏ 5 (0·3)
Tincturæ Capsici . .	♏ 1 (0·06)
Spt. Chlorof. . .	♏ 10 (0·6)
Aquam	ad fl. oz. 1 (28·4)

The tonic for old age.

(68) Mistura pro Tussi.

Tincturæ Opii Camph.	♏ 10 (0·6)
Tinct. Ipecacuanhæ .	♏ 5 (0·3)
Oxymellis Scillæ .	♏ 60 (3·6)
Aquam Anisi . .	ad fl. oz. 1 (28·4)

For chronic bronchitis.

(75) Pasta [1] Lassar.

Zinci Oxidi	gr. 120 (8)
Pulv. Amyli . . .	gr. 120 (8)
Paraff. Moll. . . .	oz. 1 (32)

Sometimes equal parts of Paraff. Moll. and Lanolin Hyd. are used.

Mix well. A valuable protective paste for subacute eczema. For chronic conditions, Salicylic or Phenol (20 to 60 grs. (1·3–4)) may be added.

[1] **Pastes are stiff ointments which act as protectives and absorb exudation.**

(84) Pilula Digitalis Composita.

Pulveris Digitalis . .	
Pulveris Scillæ . .	
Pilulæ Hydrargyri . .	āā gr. 1 (0·06)

Valuable in cardiac dropsy, and as a diuretic in ascites. It is apt to cause salivation unless the bowels are acting regularly.

(88) PILULA COLCHICINÆ.

Colchicinæ . . .	gr. 1/60 (0·001)
Ext. Nuc. Vom. . .	gr. 1/6 (0·01)
Ext. Hyoscyami . .	gr. 1/2 (0·03)
Ext. Gentianæ. . .	gr. 1 (0·06)

Twice or thrice a day for acute gout.

(90) PILULA PODOPHYLLI COMPOSITA.

Resinæ Podophylli .	
Pulveris Ipecacuanhæ .	
Hydrargyri Subchloridi.	āā gr. 1 (0·06)
Extracti Hyoscyami .	gr. 2 (0·12)

A useful liver pill in hepatic congestion.

(104) UNGUENTUM PETROLEI COMP.

Hydrargyri Ammoniati .	gr. 10 (0·6)
Liquoris Carbonis Detergentis	♏ 30 (1·8)
Paraffini Mollis . . .	oz. 1 (32)

A mild tar and mercury ointment, useful in many chronic skin diseases.

(105) UNGUENTUM SALICYLICI ET CARBOLICI.

Ac. Salicylici . . .	
Ac. Carbolici . . .	āā gr. 30 (2)
Vaselin	ad oz. 1 (32)

Stimulating ointment for chronic skin affections with excess of dry scales.

(106) UNGUENTUM SULPHURIS CO.

Sulph. Sublimat. . .	gr. 30 (2)
Acid. Carbol. . . .	♏ 8 (0·5)
Saponis Mollis. . . .	gr. 30 (2)
Adipis Benz. . . .	ad oz. 1 (32)

For acne. Should be rubbed in night and morning. In obstinate cases Sapo Mollis gr. 120–180 (8 to 12), and more sulphur may be added.

(110) VAPORES (Inhalations).

Directions.—A teaspoonful to be added to a pint of boiling water, to be inhaled for five minutes every night and morning from a narrow-necked jug or suitable inhaler. In this way use Tr. Benzoini Co. as an expectorant and local sedative in bronchitis and laryngitis: Tr. Iodi as a stimulant in chronic catarrh. Ext. Lupuli will allay irritability of mucous membrane. Ol. Eucalypti, Terebene, Creosote, Ol. Pini Sylvestris, may all be employed in the same way (strength ♏ 40 to Oi.), and certainly produce alterative effects in chronic catarrh if persevered with for several weeks.

If intended for Eustachian medication the following directions should be observed: About six times in the five minutes well fill the mouth with steam, close the nostrils with the thumb and forefinger, shut the mouth and expire forcibly, so as to drive the vapour towards the ears.

NAUHEIM BATHS.

This treatment is commenced with weak saline baths at a temperature of 92° to 95° F., consisting of 1 pound of common salt, and 1½ ounces of calcium chloride to every 10 gallons of water. These should be given every other day for a week, the patient remaining in the bath six minutes. The strength is then gradually increased to 3 pounds of salt and 4½ ounces of calcium chloride for every 10 gallons of water, and the patient remains in the bath for twenty minutes, with the temperature lowered to 85° F. if he can bear it. In a fortnight or more effervescing baths are employed. In every 10 gallons of water dissolve 2 ounces of sod. bicarb., and add 3 ounces of hydrochloric acid just before the patient enters. Gradually increase the strength to 8 ounces of sod. bicarb. and 12 ounces of acid. hydrochlor.

It is simpler to employ "Sandow's Tablets" and powders, which contain the ingredients for the baths specially prepared in a convenient form for ready use.

Treatment extends over five weeks. The effervescing baths are ordered according to the discretion of the physician, and in severe cases it is sometimes unsafe to employ them at all.

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The principal reference is in black type